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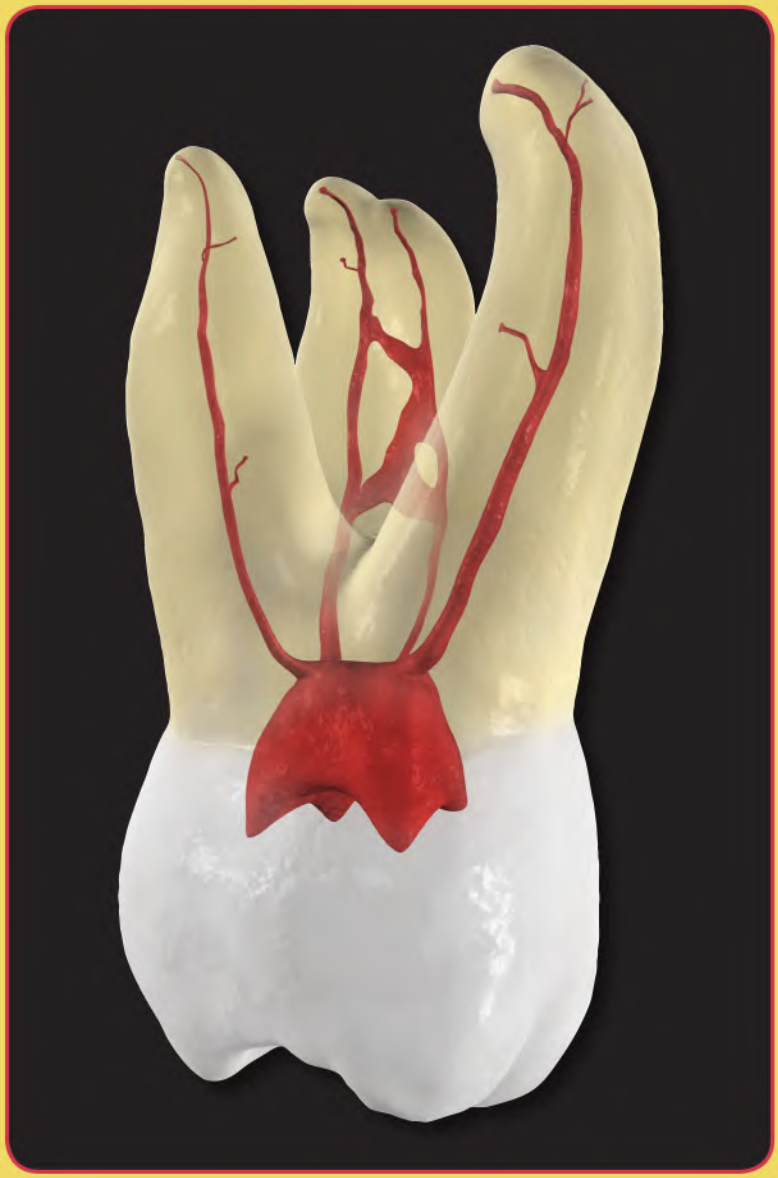
INGLE'S ENDODONTICS 7



ILAN ROTSTEIN / JOHN I. INGLE

Ingle's

ENDODONTICS 7

About the Editors

Ilan Rotstein

Dr. Ilan Rotstein is Professor and Associate Dean, and Chair of Endodontics, Orthodontics and General Practice Residency at the Herman Ostrow School of Dentistry, University of Southern California, Los Angeles, California.

He has served in leadership roles for various dental organizations, including chair of the Research Committee of the International Federation of Endodontic Associations, member on committees of the American Association of Endodontists and the European Society of Endodontology, and as scientific reviewer for international endodontic and dental journals. He has also served as president of the Southern California Academy of Endodontists, president of the International Association for Dental Research–Israel Division, president of the Israel Endodontic Society, and chair of the Israel National Board of Diplomates in Endodontics.

Dr. Rotstein has published over 150 scientific articles and research abstracts in the dental literature as well as chapters in international endodontic textbooks including *Ingle's Endodontics*, *Pathways of the Pulp*, *Endodontics: Principles and Practice*, *Seltzer and Bender's Dental Pulp*, *Endodontia: Biologia e Técnica*, and *Harty's Endodontics in Clinical Practice*. He has lectured extensively in more than twenty-five countries throughout five continents.

John I. Ingle

Dr. John I. Ingle, is best known as author of *ENDODONTICS*, a leading text in the field, first published in 1965. He is also the author of *PDQ ENDODONTICS*, a compilation of information from the larger text.

Dr. Ingle obtained his dental degree from Northwestern University in 1942. He received his graduate degree in periodontics and endodontics from the University of Michigan in 1948. Among his many contributions to endodontics, in 1957 Dr. Ingle standardized the size and shape of endodontic instruments, an international standard that remains today. In 1965, he was granted the Alumni Merit Award from Northwestern University. In 1987, he received the Ralph E. Sommer Research Award from the American Association of Endodontists, and in 1999 the Edgar D. Coolidge Leadership Award. He has also received the International Louis I. Grossman Award from the French Society of Endodontics.

Dr. Ingle is a Fellow of the American and International Colleges of Dentists and is listed in *Who's Who in America*. He has published over 80 scientific articles in the dental literature and has lectured extensively throughout the world.

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ILAN ROTSTEIN, DDS
Professor and Associate Dean
Chair, Division of Endodontics, Orthodontics and
General Practice Residency
Herman Ostrow School of Dentistry of USC
University of Southern California
Los Angeles, California

JOHN I. INGLE, DDS
Loma Linda University
School of Dentistry
Loma Linda, California

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PMPH USA, Ltd.

5711 Six Forks Road, Suite 210
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Fax: 919-502-7673
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Jaypee Brothers Medical Publishers
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4838, 24 Ansari Road, Darya Ganj
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In Memoriam: John I. Ingle, DDS, MSD, 1919–2017

FIGURE 1

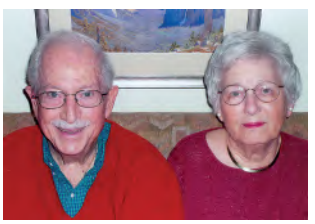


FIGURE 2

FIGURE 3

A beloved endodontic leader, Dr. John I. Ingle, passed away on September 25, 2017 in San Diego at the age of 98. He was predeceased by his wife, Joyce Ledgerwood Ingle, who passed away on March 8, 2014. They leave three children, five grandchildren, and two great grandchildren. John and Joyce Ingle knew each other all their lives: their families were close friends when they were born seven months apart in June 1918 and January 1919, and they attended school together from elementary school through college. Joyce became an accomplished jazz pianist and even learned Braille in order to translate books for blind children. They married on July 11, 1940 (Figure 1), and were married for nearly 74 years (Figure 2).

Dr. John Ingle was born in 1919 in Colville, Washington. His career in dentistry and endodontics spanned more than six decades, beginning with dental school at Northwestern University in Chicago, which he completed in 1942.

After serving the country in the military for four years as a dental officer (Figure 3), Dr. Ingle began a long and distinguished career in dental education. He accepted an offer to teach periodontics at the University of Washington in Seattle. However, Dr. Ingle had an interest in both periodontics and the new emerging field of root canal therapy—endodontics. Because of that interest, he was sent to the University of Michigan, where he earned a graduate degree in both periodontics and endodontics in 1948, returning to Seattle to teach both disciplines. He subsequently earned certification from both the American Board of Periodontics (ABP) and the American Board of Endodontics (ABE).

Establishing the first endodontic specialty program on the West Coast in 1959, Dr. Ingle was involved in the education of many endodontists who followed in his path of endodontic education. He took great pride in inspiring others to become engaged in teaching endodontics. After 16 years of teaching at the University of Washington, Dr. Ingle moved to Los Angeles, California to assume the position of Dean of the School of Dentistry at the University of Southern California, which allowed him to become involved in the expanding endodontic community in Southern California.

Dr. Ingle's interest in endodontics covered all aspects of the discipline, including the instruments used for root canal treatment, the outcomes of treatment, the dissemination of information, and the participation in activities of the specialty. Frustrated with the lack of standardization of root canal instruments, Dr. Ingle tried to interest dental companies in developing a worldwide standard for the size and shape of endodontic files and reamers. In the United States, nonmetric measurements were used while in the rest of the world the metric system was universal. In addition, each manufacturer had its own standards for sizes and shapes of instruments. In an attempt to generate an interest in the problem, Dr. Ingle published a paper pointing out the need for standardization of instruments, and following intensive research he developed the standardization system in 1957—the 0.2 taper and metric measurements—that is still in use today.

The precision in design and manufacturing of endodontic instruments that Dr. Ingle's innovation created so early in the history of endodontics is taken for granted today. It illustrates, however, how he would identify a problem and work hard to find a solution. His constant search for solutions to treatment problems made him open to accepting many innovations in techniques and instruments, and he ensured that they were described in his textbooks. While root canal therapy probably originated in the mid-1800s, the treatment of teeth with root canal infections was set back when in the early 1900s the focal infection theory put a roadblock in the path of such treatments. It took the efforts of pioneers such as Dr. Ingle to change the attitudes of many in the dental and medical professions. Even in the 1950s, endodontics was not widely accepted, and questions were raised about both the success and the safety of such treatments.

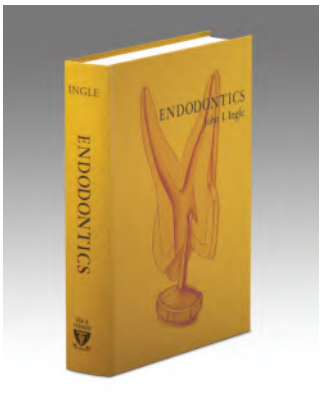


FIGURE 4

Along with pioneers such as Dr. Larz Strindberg in Sweden, Dr. Ingle conducted research in endodontic outcome that enabled us to describe important clinical and radiographic factors associated with the success and failure of root canal treatment. It soon became known as the "Washington Study." Its importance was that it demonstrated a high rate of success in root canal treatment, as well as identifying the main causes of failures: poorly performed treatment procedures. The conclusion was that root canal therapy could be successful if done properly. Thus, the classic "Washington Study" has taken its place in the history of important contributions to endodontics.

One of Dr. Ingle's passions in life was to disseminate information on all aspects of endodontics. That led to the publication of a milestone in endodontic textbooks—*Ingle's Endodontics*—the first edition of which was published in 1965 (Figure 4). It was followed by six additional editions spanning over half a century. *Ingle's Endodontics* gained notice as soon as it was published. The bright yellow-colored book [a color suggested by his wife, Joyce] was an innovative textbook for its day, and its nine by eleven-inch size was unique for medical texts at that time. In addition to the superb illustrations, it contained full-size plates of access preparations. It soon became a textbook that inspired many dentists to pursue endodontics as a specialty and has been a model for other textbooks to follow.

In recognition for his many contributions to our specialty, Dr. Ingle received the American Association of Endodontists's (AAE) Ralph F. Sommer Award in 1987 and the Edgar D. Coolidge Award, the AAE's highest honor, in 1999. The Sommer Award had a special meaning for Dr. Ingle in that Dr. Sommer had been one of his teachers at the University of Michigan. In 2015, the AAE General Assembly, at the Annual Session, honored Dr. Ingle with a special recognition for his contributions to endodontics. He was also honored by many other international organizations.

Dr. Ingle's interests spanned many areas. After completing six years as Dean of the School of Dentistry at the University of Southern California, he was honored by being appointed Senior Staff Officer to the Institute of Medicine in the National Academy of Sciences in 1972.

The words "icon," "legend," and "giant" are so overused that John Ingle would not have wished to be described in such terms. He would likely have been pleased to be remembered as a mentor, a teacher, and a friend to numerous colleagues in the United States and abroad, and one who has had a lasting impact on the lives and careers of dental professionals and endodontists worldwide.

—Leif Bakland
—Ilan Rotstein

Dedication

To my wife Lucille and our daughters Meirav, Netta and Tamar, and in loving memory of Sara and Samuel (Milu) Rotstein.

With gratitude to those special people who influenced my professional path:

Miriam Rotstein
Miriam Yacobovich
Eduardo Llamosas
Harold C. Slavkin
John I. Ingle

Contents

Contributors

Paul V. Abbott, BDSc, MDS [7]
Winthrop Professor of Clinical Dentistry
School of Dentistry/Oral Health Center of Western Australia
University of Western Australia
Crawley, WA, Australia

Khalid Al-Hezaimi, BDS, MSc [36]
Research Chair for Growth Factors and Bone Regeneration, 3D Imaging and Biomechanical Laboratory
College of Applied Medical Sciences
King Saud University
Riyadh, Saudi Arabia

Jens O. Andreasen, DDS, Odont Dr [15]
Department of Oral and Maxillofacial Surgery
University Hospital (Rigshospitalet)
Copenhagen, Denmark

Adham A. Azim, BDS [19]
Director, Advanced Program in Endodontics
School of Dental Medicine
University at Buffalo
Buffalo, New York

Nadim Z. Baba, DMD, MSD [35]
Professor and Director
Hugh Love Center for Research and Education in Technology
Loma Linda University School of Dentistry
Loma Linda, California

Leif K. Bakland, DDS [13, 15]
Professor Emeritus of Endodontics
Loma Linda University School of Dentistry
Loma Linda, California

J. Craig Baumgartner, DDS, MS, PhD [30]
Professor Emeritus of Endodontology
Oregon Health & Science University
Portland, Oregon

Yaara Y. Berdan, DDS [38]
Assistant Professor of Clinical Dentistry
Herman Ostrow School of Dentistry of USC
University of Southern California
Los Angeles, California
Private Practice
Tarzana, California

Louis H. Berman, DDS [14]
Clinical Associate Professor of Endodontics
University of Maryland School of Dentistry
Baltimore, Maryland
Private Practice
Annapolis, Maryland

Enrique Bimstein, DDS [38]
Professor Emeritus of Pediatric Dentistry
University of Kentucky College of Dentistry
Lexington, Kentucky

George Bogen, DDS [27]
Senior Lecturer
School of Dentistry
Faculty of Health and Behavioural Sciences
The University of Queensland
Brisbane, Australia

Tatiana M. Botero, DDS, MS [5]
Clinical Associate Professor
Department of Cariology, Restorative Sciences, and Endodontics
School of Dentistry
University of Michigan
Ann Arbor, Michigan

Arnaldo Castellucci, MD, DDS [22]
Assistant Professor of Endodontics
Master in Endodontics
University of Cagliari
Cagliari, Italy
Assistant Professor, Micro Surgical Endodontics
Department of Oral Surgery
University Federico II
Naples, Italy
Private Practice in Endodontics
Florence, Italy

Nicolas P. Chandler, BDS, MSc, PhD [27]
Associate Professor of Endodontics
University of Otago
Dunedin, New Zealand

Kyung-Soo Choi, MS, DDS, PhD [26]
Private Practice
Chicago, Illinois

William H. Christie, DMD, MS [1]
Professor of Dentistry, Retired
Department of Restorative Dentistry
College of Dentistry
Faculty of Health Science
University of Manitoba
Winnipeg, Manitoba, Canada

Blaine M. Cleghorn, DMD, MS [1]
Professor and Assistant Dean, Clinics
Faculty of Dentistry
Dalhousie University
Halifax, Nova Scotia, Canada

Elisabetta Cotti, DDS, MS [9E]
Professor and Chair
Department of Conservative Dentistry and Endodontics
University of Cagliari
Cagliari, Italy

Stephen B. Davis, DDS [5]
Former Director, Endodontic Residency Program
Tibor Rubin VA Medical Center
Long Beach, California

Anibal R. Diogenes, DDS, MS, PhD [29]
Associate Professor of Endodontics
University of Texas Health Science Center at San Antonio
San Antonio, Texas

Hatice Dogan-Buzoglu, DDS, PhD [9D]
Professor, Department of Endodontics
Faculty of Dentistry
Hacettepe University
Ankara, Turkey

Paul D. Eleazer, DDS, MS [32]
Professor and Chair, Retired
Department of Endodontics
University of Alabama
Birmingham School of Dentistry
Birmingham, Alabama

Joel B. Epstein, DMD, MSD [31]
Professor
Cedars-Sinai Health Systems
Los Angeles, California
Consulting Staff
City of Hope National Medical Center
Duarte, California

Allan G. Farman, BDS, PhD, EdS, MBA, DSc [9B, 9C]
Professor Emeritus of Radiology and Imaging Science
University of Louisville
Louisville, Kentucky
Independent Consultant on Maxillofacial Imaging Science
Chicago, Illinois

Ashraf F. Fouad, BDS, DDS, MS [4]
Freedland Distinguished Professor and Chair
Department of Endodontics
School of Dentistry
University of North Carolina at Chapel Hill
Chapel Hill, North Carolina

Gerald N. Glickman, DDS, MS, MBA, JD [24]
Professor and Chair
Department of Endodontics
Director of Graduate Endodontics
Texas A&M University
Baylor College of Dentistry
Dallas, Texas

Fernando Goldberg, DDS, PhD [34]
Professor Emeritus of Endodontics
Dental School of the University of El Salvador
Asociación Odontológica Argentina
Buenos Aires, Argentina

Manuel R. Gomez, DDS [9A]
Clinical Associate Professor
Department of Endodontics
University of Iowa
College of Dentistry and Dental Clinics
Iowa City, Iowa

Charles J. Goodacre, DDS, MSD [35]
Distinguished Professor
Department of Restorative Dentistry
School of Dentistry
Loma Linda University
Loma Linda, California

Kishor Gulabivala, BDS, MSc, FDS, RCS, PhD [33]
Professor of Endodontology
Consultant in Restorative Dentistry
Head of Department of Restorative Dentistry and Endodontology
UCL Eastman Dental Institute
London, United Kingdom

James L. Gutmann, DDS, PhD [39]
Professor Emeritus, Restorative Sciences/ Endodontics
Texas A&M University
Baylor College of Dentistry
Dallas, Texas

Markus Haapasalo, DDS, Dr Odont (PhD) [21]
Professor and Chair, Division of Endodontics
Head, Department of Oral Biological and Medical Sciences
University of British Columbia
Vancouver, British Columbia, Canada

Sivakami R. Haug, DDS, Dr Odont [2]
Associate Professor and Head
Section for Endodontics
Department of Clinical Dentistry
Faculty of Medicine and Dentistry
University of Bergen
Bergen, Norway

Geoffrey S. Heithersay, AO, BDS, MDS, DDSc [15]
Clinical Professor
School of Dentistry
The University of Adelaide
Adelaide, Australia

Karin J. Heyeraas, CMD, Dr Odont [2]
Professor Emeritus
Faculty of Medicine and Dentistry
Department of Biomedicine
University of Bergen
Bergen, Norway

Graham R. Holland, DDS, PhD [5]
Professor of Dentistry
Department of Cariology, Restorative Sciences, and Endodontics
School of Dentistry
Professor of Cell and Developmental Biology
School of Medicine
University of Michigan
Ann Arbor, Michigan

Lars G. Hollender, DDS, PhD [9B]
Professor Emeritus of Oral Radiology
University of Washington
School of Dentistry
Seattle, Washington

George T.-J. Huang, DDS, MSD, DSc [4]
Director for Stem Cells and Regenerative Therapies
Professor of Bioscience Research
University of Tennessee Health Science Center
Memphis, Tennessee

Michael Hülsmann, DDS [20]
Professor of Conservative and Preventive Dentistry and Periodontology
University of Göttingen
Göttingen, Germany

John I. Ingle, DDS [Editor]
Loma Linda University School of Dentistry
Loma Linda, California

Bernadette Jaeger, DDS [17]
Associate Professor, Department of Anesthesiology
School of Medicine
Section of Oral Medicine and Orofacial Pain
School of Dentistry
University of California, Los Angeles
Los Angeles, California

Bradford R. Johnson, DDS, MHPE [31]
Department Head and Director of Postdoctoral Endodontics
University of Illinois at Chicago
College of Dentistry
Chicago, Illinois

Asma A. Khan, BDS, PhD [7]
Associate Professor of Endodontics
UNC School of Dentistry
The University of North Carolina at Chapel Hill
Chapel Hill, North Carolina

Syngcuk Kim, DDS, PhD, MD (Hon) [2]
Louis I. Grossman Professor of Endodontics
Associate Dean for Global Affairs and Continuing Education
University of Pennsylvania
School of Dental Medicine
Philadelphia, Pennsylvania

Martin D. Levin, DMD [9B, 9C]
Chair, Dean's Council
University of Pennsylvania School of Dental Medicine
Philadelphia, Pennsylvania
Private Practice in Endodontics
Chevy Chase, Maryland

Thomas A. Levy, DDS, MS [37]
Former Associate Professor of Clinical Dentistry
Herman Ostrow School of Dentistry of USC
University of Southern California
Los Angeles, California

James S. Lin, DDS, MSc [21]
Clinical Associate Professor and Director
Undergraduate Endodontic Program
Department of Oral Biology and Medical Sciences
University of British Columbia
Vancouver, British Columbia, Canada

Eduardo Llamosas, DDS [26]
Founding Professor of Endo-Perio Program
Coordinator of the Dental Specialties
Facultad de Estudios Superiores Iztacala
Universitad Nacional Autónoma de México
Mexico City, México

Pierre Machtou, DDS, MS, PhD [23]
Professor Emeritus
Co-Director of the Specialty Program in Endodontics
Paris, France
Visiting Professor, University of Geneva
Geneva, Switzerland

Stanley F. Malamed, DDS [18]
Professor Emeritus of Dentistry
Herman Ostrow School of Dentistry of USC
University of Southern California
Los Angeles, California

Matthew Malek, DDS [11]
Advanced Education Program Director
Clinical Assistant Professor
Ignatius N. and Sally Quarararo Department of Endodontics
New York University
College of Dentistry
New York, New York

Jose-Maria Malfaz, DDS, MD, PhD [10]
Lecturer
Private Practice Limited to Endodontics, Endodontic Microurgery, and Dental Traumatology
Valladolid, Spain

Vivian Manjarrés, DDS [39]
Dental Medicine Faculty
Assistant Professor, Endodontics
Miami Lakes Periodontal Associates
Fort Lauderdale, Florida

C. John Munce, DDS [19]
Assistant Professor
Department of Endodontics
Loma Linda University School of Dentistry
Loma Linda, California

Yuan-Ling Ng, BDS, MSc, MRD RCS, PhD [33]
Director of Masters Programmes in Endodontology
Senior Clinical Lecturer in Endodontology
UCL Eastman Dental Institute
London, United Kingdom

Stephen P. Niemczyk, DMD [25]
Director, Endodontic Microsurgery
Albert Einstein Medical Center, Dental Division
Philadelphia, Pennsylvania
Director, Endodontic Microsurgery
Harvard University School of Dentistry
Boston, Massachusetts
Private Practice, Limited to Endodontics
Drexel Hill, Pennsylvania

Katsushi Okazaki, DDS, PhD [11]
Clinical Assistant Professor
Ignatius N. and Sally Quarararo Department of Endodontics
New York University College of Dentistry
New York, New York

Terrell F. Pannkuk, DDS, MScD [19]
Private Practice
Santa Barbara Endodontics
Santa Barbara, California

Ellen Park, DDS, MSc, Dip Endo [21]
Clinical Instructor in Endodontics
Graduate Endodontic Division
University of British Columbia
Vancouver General Hospital
Vancouver, British Columbia, Canada

Rajiv G. Patel, BDS, DDS [19]
Private Practice in Endodontics
Endodontic Excellence
Flower Mound, Texas

Wei Qian, DMD, MSc, PhD [21]
Private Practice
Vancouver, British Columbia, Canada

Ramya Ramamurthy, DDS, MS [9B]
Private Practice
Greenroot Endodontics and Microsurgery
San Jose, California

S. Craig Rhodes, DMD [32]
Associate Professor and Pre-Doctoral Director
Department of Endodontics
School of Dentistry
University of Alabama, Birmingham
Birmingham, Alabama

Domenico Ricucci, MD, DDS [3, 6]
Private Practice
Cetraro, Italy

Isabela N. Rôças, DDS, MSc, PhD [3, 6]
Professor of Endodontics
Head, Molecular Microbiology Laboratory
Estácio de Sá University
Rio de Janeiro, Brazil

Rafael A. Roges, DDS [10]
Director, Advanced Endodontics Program
The Wayne G. and Margaret L. Bemis Endowed Professor in Endodontics
Herman Ostrow School of Dentistry of USC
University of Southern California
Los Angeles, California

Paul A. Rosenberg, DDS [11, 30]
Professor of Endodontics
Ignatius N. and Sally Quartararo Department of Endodontics
New York University College of Dentistry
New York, New York

Ilan Rotstein, DDS [26, 36, 37, 40]
Professor and Associate Dean
Chair, Endodontics, Orthodontics and General Practice Residency
Herman Ostrow School of Dentistry of USC
University of Southern California
Los Angeles, California

Clifford J. Ruddle, DDS [23]
Assistant Professor of Graduate Endodontics
Loma Linda University
Loma Linda, California
Assistant Professor of Graduate Endodontics
University of California, Los Angeles
Los Angeles, California

Nikita B. Ruparel, MS, DDS, PhD [7]
Assistant Professor
Department of Endodontics
University of Texas Health Science Center at San Antonio
San Antonio, Texas

Mohammad Sabeti, DDS, MA [24]
Health Science Clinical Associate Professor
Director, Advanced Speciality Education Program in Endodontics
UCSF School of Dentistry
San Francisco, California

Edgar Schäfer, Prof Dr [20]
Head of Interdisciplinary Ambulance
School of Dentistry
University of Münster
Münster, Germany

Parish P. Sedghizadeh, DDS, MS [10]
Associate Professor of Clinical Dentistry
Section Chair of Diagnostic Sciences
Director, USC Center for Biofilms
Herman Ostrow School of Dentistry
University of Southern California
Los Angeles, California

Shahrokh Shabahang, DDS, MS, PhD [28]
Associate Professor of Endodontics
Loma Linda University School of Dentistry
Loma Linda, California

Ya Shen, DDS, PhD [21]
Assistant Professor
Department of Oral Biological and Medical Sciences
Division of Endodontics
University of British Columbia
Vancouver, British Columbia, Canada

Ziv Simon, DMD, MSc [19]
Instructor
Herman Ostrow School of Dentistry
University of Southern California
Los Angeles, California
Private Practice Limited to Periodontics
Beverly Hills, California

José F. Siqueira Jr., DDS, MSc, PhD [3, 6]
Chair and Director, Postgraduate Program in Endodontics
Faculty of Dentistry
Estácio de Sá University
Rio de Janeiro, Brazil

Anthony J. Smith, PhD [29]
Professor Emeritus
School of Dentistry
University of Birmingham
Birmingham, United Kingdom

Jaydeep Shashikumar Talim, BDS, MSc [37]
Assistant Clinical Professor
Department of Endodontics
Herman Ostrow School of Dentistry of USC
University of Southern California
Los Angeles, California

Aviad Tamse, DMD [14]
Professor Emeritus
Department of Endodontology
Tel Aviv School of Dental Medicine
Tel Aviv, Israel

Roderick W. Tataryn, DDS, MS [16]
Lecturer, Endodontics
Loma Linda University
Loma Linda, California
Private Practice, Tataryn Endodontics
Spokane, Washington

Mahmoud Torabinejad, DMD, MSD, PhD [24]
Director of the Torabinejad Institute of Surgical Education and Research Venues
Irvine, California

Martin Trope, BDS, DMD [12]
Clinical Professor of Endodontics
University of Pennsylvania
Philadelphia, Pennsylvania

Richard E. Walton, DMD, MS [9A]
Professor Emeritus of Endodontics
University of Iowa College of Dentistry
Iowa City, Iowa

Zhejun Wang, DDS, PhD [21]
Division of Endodontics
Department of Oral Biological and Medical Sciences
University of British Columbia
Vancouver, British Columbia, Canada

Borja Zabalegui, MD, DDS, PhD [36]
Professor of Endodontics
University of the Basque Country/EHU
Leioa, Bizkaia, Spain
Private Practice Limited to Endodontics
Bilbao, Spain

Foreword

Fifty-three years ago, the year I graduated from the University of Southern California School of Dentistry with John Ingle as our dean he published, unknown to me at the time, his first edition of *ENDODONTICS* — now celebrating its 50th Anniversary Edition. This is remarkable on several different levels.

First, John hired me in 1968 as an Assistant Professor of Biochemistry and Nutrition in the USC School of Dentistry to start my 46-year tenure on the full-time faculty of the University of Southern California.

Second, I have been able to join with others over the years to celebrate John's birthdays. We celebrated John's 98th birthday in January 2017 while he continued to savor the pleasures of authorship, editing, friendship, and staying connected to his passion for endodontics.

Finally, this is a very special occasion to also celebrate my colleague and dear friend Ilan Rotstein who has joined with John to produce the 7th edition of *Ingle's ENDODONTICS*. Together, John and Ilan have organized an excellent and authoritative edition that includes 40 chapters contributed by internationally recognized experts in the field. Collectively, these contributions reflect the most comprehensive textbook in modern endodontics worldwide. Further, they emphasize the science of endodontics, the practice of endodontics, and the interdisciplinary nature of endodontics.

I celebrate their accomplishment with this 7th edition of *Ingle's ENDODONTICS* and encourage the reader to learn and enjoy this fascinating field. Cheers!

—Harold C. Slavkin, DDS
Professor and Dean Emeritus
6th Director of the NIDCR, National Institutes of Health

Preface to the 7th Edition

Endodontic treatment is an important therapeutic modality that allows retention of the natural dentition in the oral cavity and its return to health and function.

The specialty of endodontics has gone through many groundbreaking changes over the past several years. Recent advances in concepts, technology, materials, and equipment have changed the way endodontics is being practiced today. These advances have made it possible to efficiently perform successful endodontic treatment with improved precision and predictability and with greater patient acceptance and comfort.

The seventh edition of *Ingle's ENDODONTICS* is the most recent revision of the text that has been known as one of the most authoritative in the field for half a century. This 50th anniversary edition includes 40 chapters written by internationally renowned authors from six continents, contributing new, cutting-edge knowledge and updates on topics that have formed the core of the specialty. The evidence-based information provided in this edition continues to influence what is thought, what is taught, and what is practiced in modern endodontics worldwide. It is the standard against which all other endodontic texts will be measured.

The three main themes of this edition are The Science of Endodontics, The Practice of Endodontics, and Interdisciplinary Endodontics. Endodontic treatment is an art requiring clinical skills that build upon a robust foundation of scientific evidence. In tandem, the endodontic professional must play a significant role as part of a team of specialists in order to attain the correct diagnosis, proper treatment decision-making, and a predictable outcome.

I wish to thank the authors for their most valued contribution, especially under a very intensive and ambitious timeline. Their efforts and dedication have made this edition the most comprehensive textbook in endodontics today.

I owe a special debt of gratitude to Dr. John Ingle, a pioneer, leader, mentor, and educator for entrusting me with one of his most precious treasures: *Ingle's ENDODONTICS*. I was surprised and humbled when Dr. Ingle asked me to be the Editor-In-Chief of this textbook. Thank you, John, for the mentorship, trust and support. For me, it made this project an extraordinarily enriching learning experience and a labor of love.

A hearty thank you to Dr. Leif K. Bakland and Dr. J. Craig Baumgartner for their advice, guidance, and unconditional help.

I hope you, the reader, enjoy this book and that it will become a well-used reference in your practice.

—Ilan Rotstein

Preface to the 6th Edition "Pull and be Damned Road" Revisited

The original sign for Pull and be Damned Road has been pilfered so many times the authorities have had to place a new sign over 20 feet above the ground. (Courtesy of Dr. James Stephens.)

Over 40 years ago, the preface to the first edition of *Endodontics* featured "**Pull & Be Damned Road.**" Nothing from the first edition left such a lasting impression as that saying. Even today, "old timers" come up to me at meetings to reminisce about this preface. They may not remember the details of endodontic cavity preparation or the chapter on pain first expressed in that edition. Those features have become an integral part of any endodontic practice. But they do remember "Pull and Be Damned Road." And for good reason.

Forty years ago it was more prevalent to extract teeth than to save them by root canal therapy. A plea was made in this preface to trust endodontic treatment and to reverse this trend toward "oral amputation." Gradually this became a fact, as endodontics spurred ahead and full dentures declined.

And this brings to mind an incident I long have savored. I was a speaker at the Hinman Dental Meeting in Atlanta. Joining me as a headliner was Dr. Will Menninger, head of the famous Menninger Psychiatric Clinic, then based in Topeka, Kansas. Dr. Menninger and his brother Karl were unquestionably the world's most famous psychiatrists. Dr. Will had been a brigadier general in World War II, head of all the army's psychiatrists. In 1948 he was the first psychiatrist on the cover of *TIME* magazine. It was an honor for me to be on the same program with him, and I eagerly attended his first lecture that preceded mine.

When I began my lecture, there was Dr. Menninger seated in the front row. I considered it professional courtesy on his part, but I did notice him taking notes.

The Hinman had a format wherein each lecturer would repeat his same lecture the next day. I didn't attend Dr. Menninger's second lecture but he attended mine. There he was again, seated in the front row. I was flattered beyond measure. At the end of the lecture I asked him why his sudden interest in endodontics. His reply was startling.

"Dr. Ingle," he said, "I was so impressed with your lecture, but I was also terribly embarrassed. On behalf of my profession I must apologize to you for my past behavior. When I think of how many patients I have recommended that they have their teeth extracted, I am appalled at the destruction I have caused. I had no idea. Now I find these teeth could have been saved and their abscesses healed. You have no idea how thankful I am to you, for directing me from my past behavior." Spoken like a psychiatrist!

We've come a long way since those days, a time of wholesale extractions. But we face a new challenge today; not wholesale extractions but selective extractions; for the sole purpose of placing an implant. Once again, dentists are urging patients to have teeth extracted, ignoring the fact that a healthy root is far preferable to a mechanical implant; less costly, less painful, less time consuming, and above all, more biological.

Now I'm not saying implants are unhealthy or less successful. What I am expressing is my concern that many salvable teeth are being sacrificed on the altar of insatiability. Back to pull and be damned. I'm not against implants! As a matter of fact, implant therapy is now being taught in a number of endodontic post doctoral programs. The thesis being, however, that teeth

that cannot or should not be saved by endodontics may well be extracted and replaced by an implant rather than a bridge. And who better to place that implant than a well-trained endodontist who has just made that judgment?

The 6th edition of *Endodontics* is replete with new innovations and knowledge. Now, more than ever, it remains the "Bible of Endodontics" a name long applied by others to the previous editions.

I feel most comfortable as I "pass the torch" to the new editors, Leif Bakland and Craig Baumgartner. And I have a feeling the profession will come to its senses; veering off "Pull and Be Damned Road" and onto the "Road Best Traveled."

John I. Ingle
December 2007

Chapter 1

Anatomy and Morphology of Teeth and Their Root Canal Systems

Blaine M. Cleghorn, William H. Christie

Successful root canal therapy requires a thorough knowledge of tooth anatomy and root canal morphology,[1–3] which may be quite variable within the norm. Anomalies or deviations in the usual shape and root number in the adult human dentition outside the norm may also occur. When an anomalous form of tooth or root anatomy occurs fairly frequently in a given population or ethnic group, it should be considered a variant form and recognized.

Root canal treatment may fail if the root canal system is not fully identified and treated. Lack of knowledge of the variability of human root morphology, misadventure, and weakening of root structure may result in a search for a nonexistent canal. Therefore, a thorough knowledge of both normal and abnormal morphologic variations is required.

For each tooth in the permanent dentition, there is a wide range of variations reported in the literature. It specifically focuses on the frequency of occurrence of number and shape of root canals with each root, number of roots,[2,4–17] and incidence of molar root fusion.[18–24] A range of variations in the length of roots, overall size, and curvatures within root morphology are also treatment considerations but should be addressed outside this chapter.

A number of factors contribute to the variations in morphology, which are discussed in a few studies (Table 1-1). Root canal morphology of teeth is often extremely complex and highly variable,[2,4,6,13,15–17] as illustrated in the three-dimensional (3-D) models of the maxillary first molar in Figure 1-1.[6] Variations may also result from ethnic factors, age, and gender of the population studied.

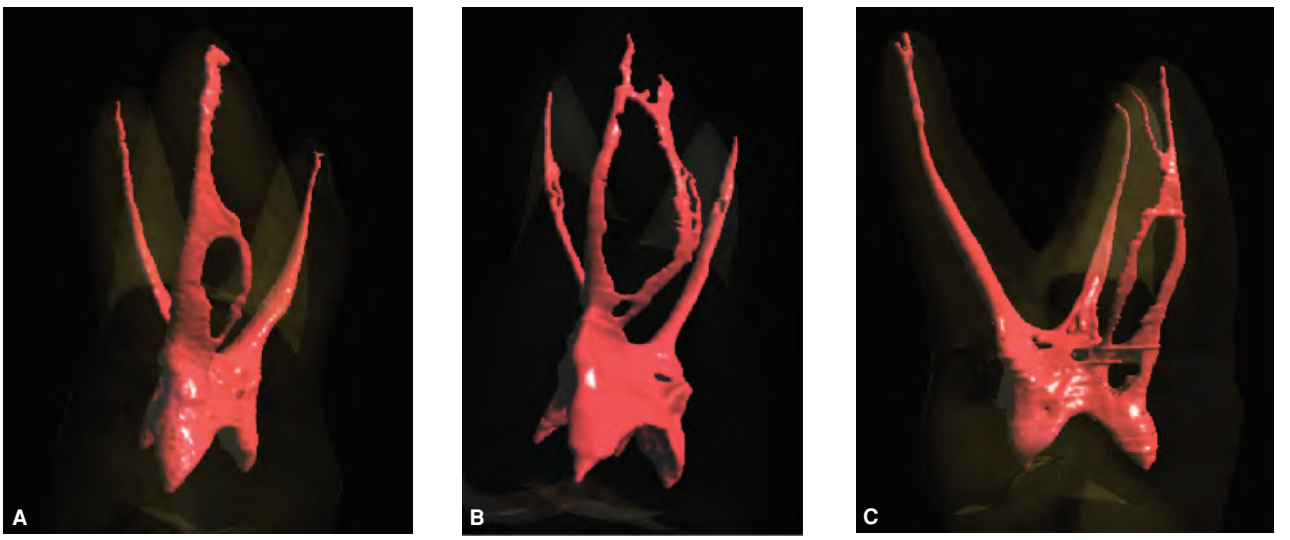


FIGURE 1-1 Root canal systems of a maxillary first molar. ***A & B.*** Mesiobuccal views (MB root canal system is centered). ***C.*** Mesial view (MB root canal system is far right). (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Maxillary First Molar- 3-D Models 1-3.)

TABLE 1-1 Factors Contributing to Variations in Reported Root and Root Canal Morphology

Factors	Further Information and References
Definition of a canal:	Negotiated and obturated to within 4 mm of the apex[25] Separate orifice found on floor of pulp chamber[26] Two instruments placed into two MB canals of a maxillary first molar to a minimum of 16 mm from a cusp of an intact tooth[27] One that can be instrumented to a depth of 3–4 mm[12] A treatable canal[28]
Age:	8,29–31
Ethnicity:	32–61
Gender:	11,21,28
Source of teeth (specialty endodontic practice vs. general practice):	62
Study design (*in vivo* versus *in vitro*):	27,63,64
In vitro methods	
Clinical examination:	65–67
Radiographic examination:	29,31,56,68–72
Radiography and instrumentation:	73–75
Clearing:	Clinical and Radiographic examination[76] Chinese Ink[60,77] Chinese Ink and Hyperbaric oxygen[78] India Ink and radiography[54,55,79] India Ink and Stereomicroscopy[80] India Ink[11,81–111] Hematoxylin dye[35,70,112–115] Hematoxylin dye and stereomicroscope[116] Oil-based dye[117] Stereomicroscope[118,119] Methylene Blue dye[120] Waterproof Black Ink[121]
Sectioning:	52,122–127 & Radiography[128–130] Stereomicroscopy[131] Radiography and instrumentation[58,132] Instrumentation[133]
Injection of stained plastic resin (plastic casts):	19,134–136
Metal castings:	8
Visual examination:	137,138
Scanning electron microscopy:	139,140
Grinding:	5
Radiopaque elastomer and radiography:	136
CBCT:	141,142
CBCT and Histology Comparison:	143
Spiral CT:	144,145
Helical CT:	146
Micro CT:	147–56
Dental loupes and modified access opening:	157
Surgical operating microscope (SOM) and ultrasonics:	158
In vivo methods	
Clinical and radiographic examination:	159–162,127
Review of FMS and/or Panoramic radiographs:	22,40,163,164,36
Review of post-RCT teeth:	30,57,165–172
CBCT:	47,61,173–194
Spiral CT:	195,196
SOM:	25
Dental loupes, headlamp and modified access opening:	28
Dental loupes and SOM:	197
Comparison of Radiology, CBCT, SOM and Start X ultrasonics:	198

Data generated from a series of teeth that were presented for endodontic treatment in a specialty endodontic practice may not represent the overall frequency of incidence in a general population, as more complex cases are more likely to be referred to a specialist,[62] thus skewing the sample reported in some clinical studies. Differences in reported results may also be due to the study design (clinical radiographs versus laboratory, gross morphology or microscopic)[63] or even unintentional bias in the selection of teeth to study and publication bias.

The "gold standard" for assessing canal morphology in laboratory studies is the clearing method, where extracted teeth are rendered transparent and a dye is injected into the canal systems. A 3-D shape of the entire length of the canals may be viewed under magnification and assessed according to the criteria predetermined by independent observers.

Results from early clinical studies generally reported fewer canals than *in vitro* bench studies. In recent years, however, data from clinical studies are becoming more comparable to laboratory studies. The use of dental loupes, headlights, surgical operating microscopes (SOMs), and ultrasonic methods has greatly improved the clinical detection of canals (Table 1-1).

The increased use of computed tomography (cone beam computed tomography [CBCT], spiral CT, helical CT, and micro-CT) has improved the accuracy of identification of canals and dramatically changed the reported results. Although there are some limitations with CBCT, both practically and economically, it has been shown to provide comparable results to those laboratory studies that used clearing methods.[199–207]

Another source of variations in the reported number of canals in clinical studies may be due to the authors' definition of what constitutes a canal. In some studies, a separate canal is defined as a separate orifice found on the floor of the pulp chamber,[26] two instruments placed simultaneously into two mesiobuccal canals of a maxillary first molar, to a minimum depth of 16 mm from the cusp of an intact tooth,[27] of which one can be instrumented to a depth of 3 to 4 mm,[12] or an analysis of treatable canals in retrospective clinical studies.[28] Other studies failed to provide a clear definition of a separate canal and may only look at radiographic outcomes.

CLINICAL ENLIGHTENMENT

From the Three-Canal Molar Tooth to the Four or More Canal Molars

In 1969, Weine et al.[122] provided the first clinical classification of more than one canal system in a single root and used the mesiobuccal root of the maxillary first molar as the sample specimen. Pineda and Kuttler[31] and Vertucci[14] further developed a system for canal anatomy classification for any tooth having a broad buccolingual diameter and is more applicable for use in laboratory studies (Figure 1-2). All canal types reported in this chapter are based on the

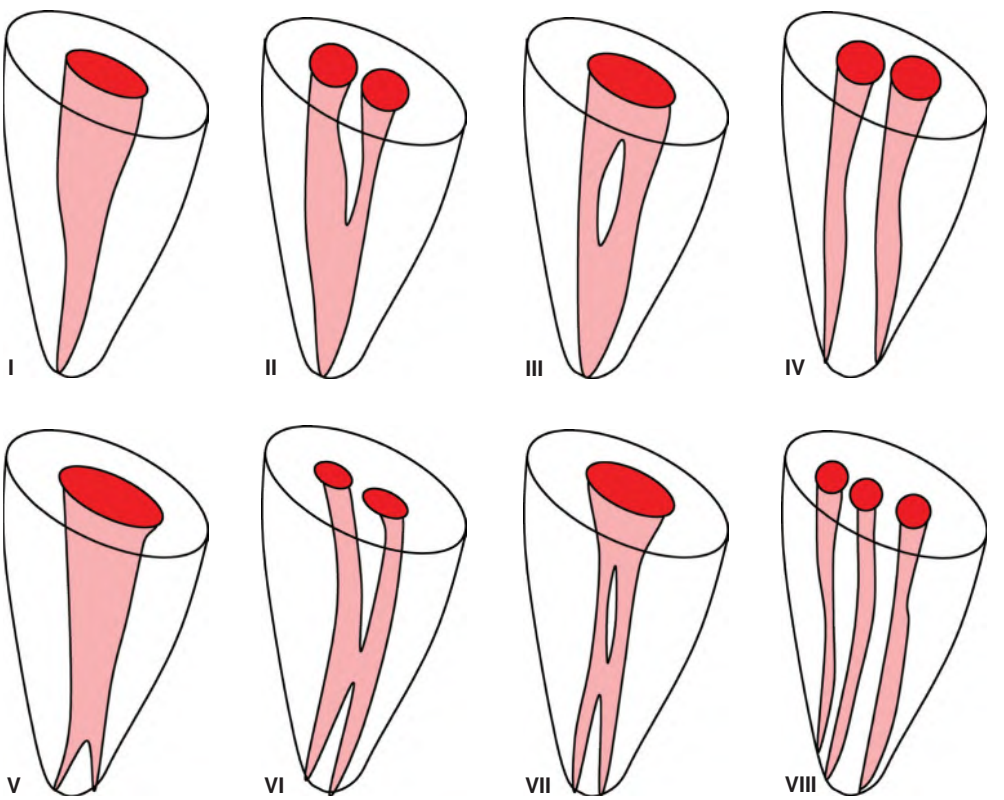


FIGURE 1-2 Vertucci's classification of root canal systems (Types I–VIII).

Vertucci's classification. Additional canal types not included in Vertucci's original classification system have been reported by Sert and Bayirli (Figure 1-3)[11] and Gulabivala et al.[91] Sert and Bayirli[11] reported an additional 14 new canal types that had not yet been classified.

Vertucci[14,208] found the proximity of the canal orifices to each other as indicative of whether they joined or remained as separate canals. If the separation of the orifices was >3 mm, the canals tended to remain separate through their entire length. In contrast, canals were usually joined together if the

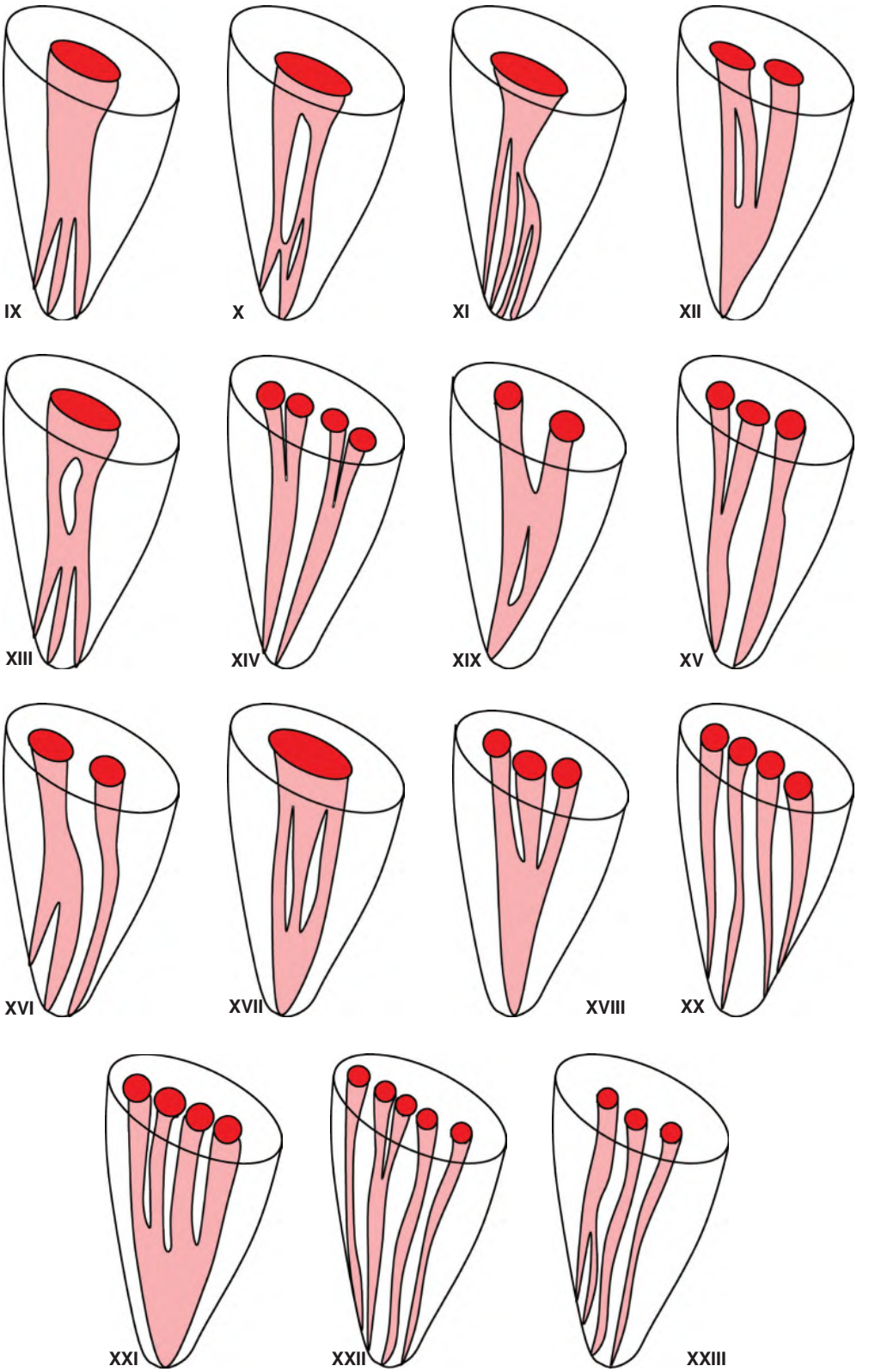


FIGURE 1-3 Sert and Bayirli's additional canal types to Vertucci's classification of root canal systems (Types IX–XXIII).

orifices were <3 mm apart. Canals were found to join more coronally as the distance between the orifices decreased.

As stated before, successful endodontic therapy is dependent on the cleaning, shaping, and obturation of the entire root canal system in three dimensions.[1–3] The presence of lateral canals and the ability to cleanse and seal these canal branches can also have an impact on prognosis.[209] Lateral canals include both accessory and furcation canals. These canals are smaller than the main canal but branch from (or into if one considers blood vascularity) the main canal to the periodontium.[2,208] Pineda and Kuttler[31,77,209] found that 30.6% of teeth had such ramifications. The majority of the ramifications or accessory canals were found in the apical third of the root. Zolty[209] and others have indicated that endodontic failure can be due to infection in these accessory canals. Clinical studies have demonstrated that careful recleansing and sealing of the root canal system can result in healing of associated periapical disease.[209,210]

This chapter provides evidence-based information on the root and root canal morphology, both the number of roots and number of canals within each of the tooth in the permanent dentition from the central incisor to the second molar in both maxillary and mandibular arches. The permanent third molars have not been included in this review due to their high degree of variability of crown and root morphology.

External root anatomy, canal numbers and types, and root canal system anatomy are described through a literature review of anatomical studies and case reports. The percentages reported in the tables may exceed 100% due to rounding of numbers to tenths or due to incorrect tabulations from papers cited. The following is a description of the normal and many variations in human tooth anatomy and root morphology as gleaned from the available literature to date.

MAXILLARY CENTRAL INCISOR

External Root Morphology

The cross-sectional root anatomy of the maxillary central incisor is triangular to ovoid in shape and tapers toward the lingual[1,3,4,6,13,15,17] as illustrated in Figure 1-4. Root concavities are normally not present in maxillary central incisors.[211] The root trunk is generally straight and tapers to a blunt apex. The overall average length of the maxillary central incisor is 23.5 mm with an average crown length of 10.5 mm and an average root length of 13 mm.[4]

Root Number and Form

The majority of anatomic studies found that the maxillary central incisor is a straight, single-rooted tooth (Table 1-2), unless rare developmental anomalies are present.

Canal System

The maxillary central incisor is usually a single root canal system (Table 1-3). Over 99% of the teeth in the studies cited in Table 1-3 had a single root canal and a single apical foramen. Mid-root and apical lateral canals and apical delta canals are common (Figure 1-5). These lateral canals with small arterioles and venules serve as collateral circulation. When the pulp becomes diseased, Schilder[210] referred them as "portals of exit" into the surrounding periodontal ligament space.

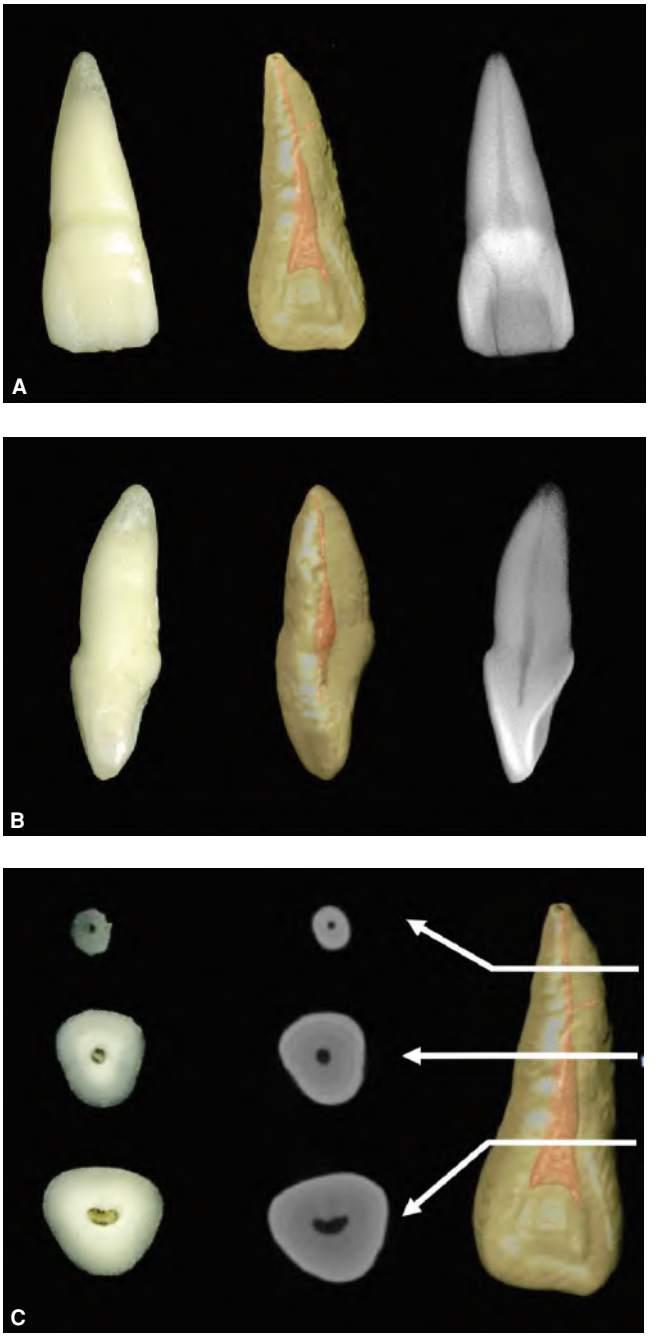


FIGURE 1-4 Maxillary right central incisor. ***A.*** Labial view. ***B.*** Mesial view. ***C.*** Root cross sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Maxillary Central Incisor- Rotations and Slices.)

TABLE 1-2 Root Number in the Maxillary Central Incisor

Maxillary Central Incisors		No. of teeth in studies	1 Root	
			%	No. of teeth
Total number of root studies[5,7,11,14,31,78,99]	7	892	100	892

TABLE 1-3 Main and Apical Canal Number of the Maxillary Central Incisor

Maxillary Central Incisors		No. of teeth in studies	1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of teeth	%	No. of teeth	%	No. of teeth	%	No. of teeth
Total number of canal studies[5,7,11,14,31,78,99,194]	8	2435	99.2	2416	0.8	19				
Total number of canal apices studies	5	760					99.7	758	0.3	2

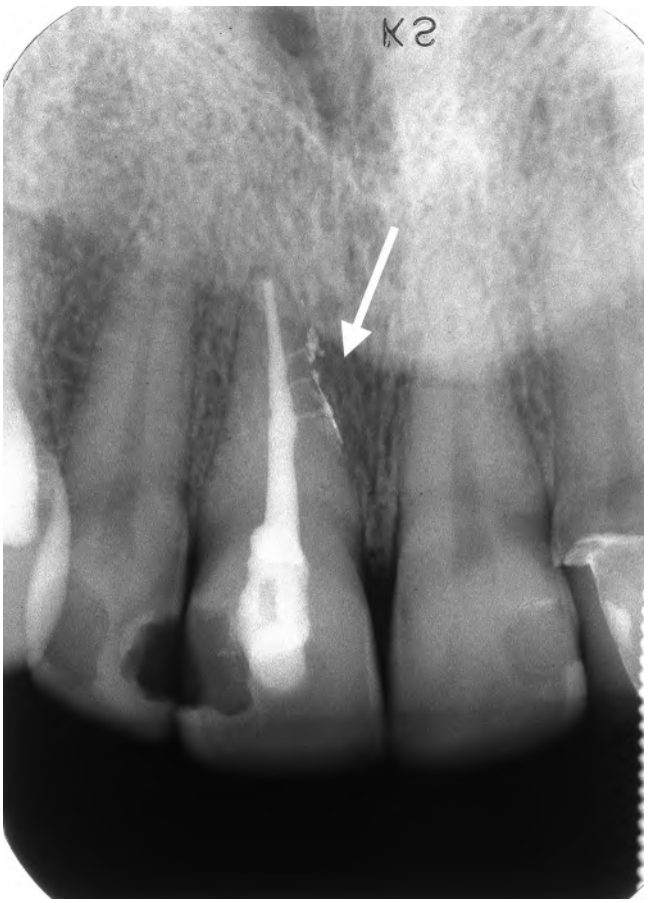


FIGURE 1-5 Maxillary right central incisor with normal anatomy. Presence of lateral canals is demonstrated by sealer extrusion (arrow). (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

Relatively few studies have investigated the apical root anatomy of teeth. Altman et al.[212] investigated 20 extracted maxillary central incisors. On histological examination, accessory or lateral canals were found in three quarters of the specimens, exhibiting one to four accessory canals. One specimen was found to have 20 separate foramina, but this anomalous canal anatomy occurred on a central incisor from a 9-year-old individual. They concluded that radiographic examination was not accurate in diagnosing accessory canals and that resorptions, appositions, pulp stones, and accessory canals were commonly found in the apical 2.5 mm of the maxillary central incisors.

The classic stereomicroscopic study of root apices by Green,[213] in 1956, found that the average diameter of the major foramen in the 50 teeth studied was 0.4 mm, while the accessory foramina were 0.2 mm or less in diameter. The average distance of the major apical foramen from the anatomical root apex was found to be 0.3 mm. Approximately 12% of the maxillary central incisors exhibited accessory foramina in their sample.

Mizutani et al.[214] investigated the anatomic location of the apical foramen in 30 maxillary central incisors. The root apex and apical foramen were displaced distolabially in the majority of the specimens. Coincidence of the apical foramen and root apex was found in only one out of six specimens (~17%).

Kasahara et al.[215] assessed the apical anatomy of 510 extracted maxillary central incisors. Apical ramifications were found in about 12% of cases. Lateral canals were present in 49%. Only 38.6% of the teeth had a simple main canal without lateral canals or apical ramifications. In this study, 90% of the foramina were located within 1.0 mm of the anatomical apex.

Variations and Anomalies

Seventy-five case reports of abnormalities of the maxillary central incisor were identified in a review of the literature (Table 1-4). The four most common variations or anomalies identified in decreasing order were talon cusps, two roots and two canals, one root and two canals and fusion. Ethnic variations have been attributed for some coronal traits. Shovel-shaped incisor crowns with heavy lingual ridge formation and even labial buttressing to varying degrees are a common feature in Asian (including North Americans), Natives formerly described as "Mongoloid" populations, but are relatively rare in Caucasian populations.[13,53,216–220]

A study by Pecora and da Cruz Filho[65] assessed the incidence of radicular grooves in the maxillary incisors of 642 patients. Loss of periodontal attachment associated with these grooves can result in a deep, narrow, vertical periodontal pocket. The incidence of radicular grooves was found to be 0.9% in the maxillary central incisors and 3.0% in maxillary lateral incisors. The prognosis, even with combined periodontal-endodontic treatment, in such cases can be poor.[221] Reports of other types of root anomalies associated with the maxillary central incisor are relatively uncommon.

The prevalence of fusion and gemination was assessed by Hamasha and Al-Khateeb[222] from within a large sample size of 9373 teeth in a Jordanian population. The prevalence of fusion was found to be 2.6% while gemination of the tooth bud was found in 0.94% of the samples for a total incidence of 3.6% of so-called "double teeth." The maxillary central incisor had the highest incidence of fusion and gemination of all permanent teeth.

TABLE 1-4 Variations and Anomalies Associated with the Maxillary Central Incisor

Anatomic Variation	No. of Cases	No. of Refs
Dens evaginatus (Talon cusp)[37,230–232,245–256]	17	16
2 roots and 2 canals[226,236,238–241,257–264]	14	14
1 root and 2 canals[233,234,265–270]	9	9
Fusion[224,225,227,271–276]	9	9
Gemination[224,275,277,278]	4	4
Dens invaginatus Type III[279–281]	3	3
Shovel-shaped incisors[282–284]	3	3
Dens invaginatus[235,237,285]	3	3
Multiple anomalies[243,244]	2	2
Talon cusp and germination[229,228]	2	2
1 root and 3 canals[286]	1	1
1 root and 4 canals[287]	1	1
2 canals and dilacerated root[288]	1	1
2 roots and 3 canals[242,289]	2	2
Dens evaginatus and fusion with a supernumerary tooth[290]	1	1
Dilacerated root[291]	1	1
Talon cusp on a geminated tooth[292]	1	1
Cervical enamel projection[293]	1	1
Total number of case report references and cases	75	74

al-Nazhan[223] reported a case of a central incisor with enamel hypoplasia and two canals in a single root.

Examples of fusion with a supernumerary tooth,[222,224–226] fusion with a maxillary lateral incisor,[227] gemination,[222,224,228,229] talon cusps,[228–232] two canals,[233,234] dens invaginatus,[235] and maxillary central incisors with two roots[236–241] are occasionally documented in case reports. A single case of gemination and fusion with a supernumerary tooth resulting in a tooth with three canals was reported by Hosomi et al.[242]

An unusual combination of anomalies was reported by Lorena et al.[243] Anomalies in the maxillary arch included shovel-shaped incisors, dens invaginatus, peg-shaped supernumerary teeth, and Carabelli cusps on the maxillary first molars in the absence of any developmental anomaly. McNamara et al.[244] also reported a combination of anomalies that included maxillary central and lateral incisors that had talon cusps, short roots and dens invaginatus, premolars with short roots, and Carabelli cusps on the maxillary first and second molars. A rare case of a labial talon cusp on a maxillary central incisor was reported by de Sousa et al.[231]

MAXILLARY LATERAL INCISOR

External Root Morphology

The cross-sectional root anatomy of the maxillary lateral incisor is described as being circular, oval, or ovoid in shape and tapers toward the lingual as illustrated in Figure 1-6.[1,3,4,6,13,15,17,214] Root concavities are normally not present on the root of the maxillary lateral incisors.[211] The root trunk

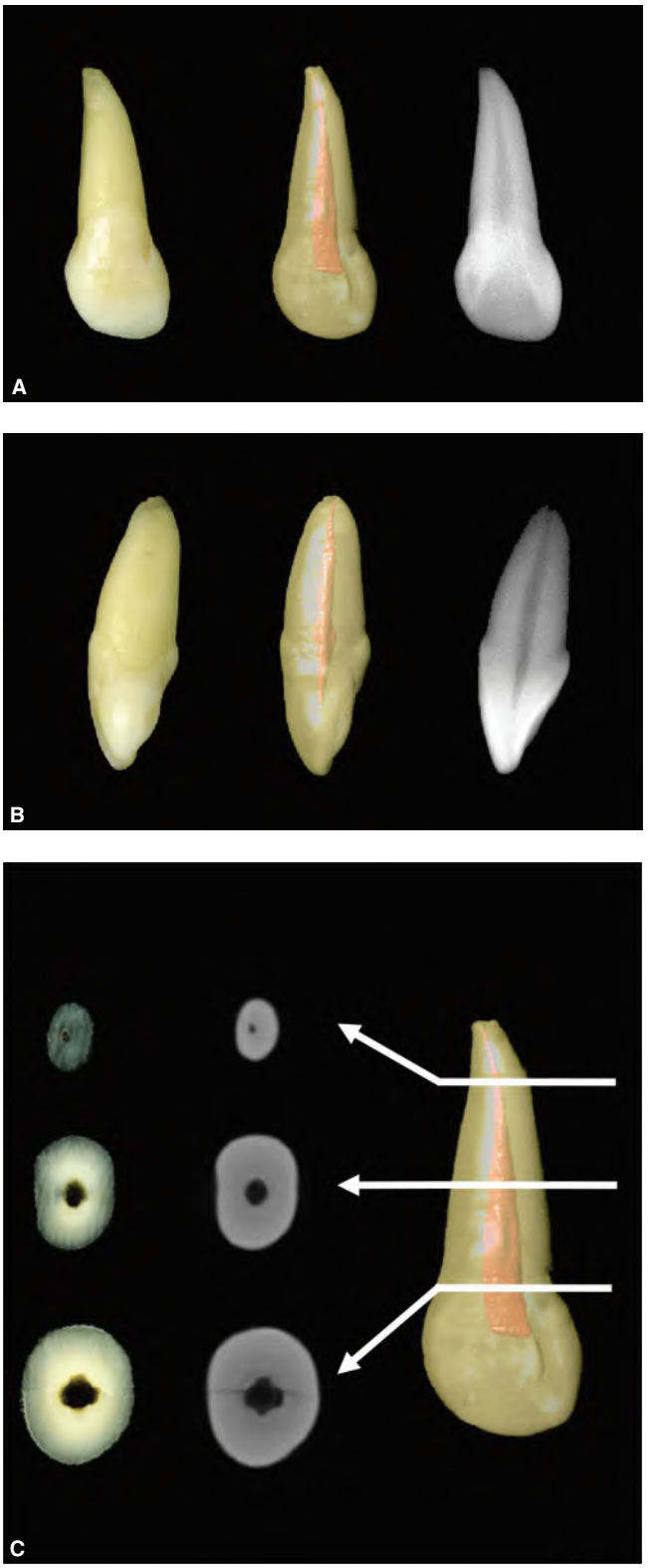


FIGURE 1-6 Maxillary right lateral incisor. ***A.*** Labial view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Maxillary Lateral Incisor- Rotations and Slices.)

is generally smaller than a central incisor and has a finer root tip, often terminating in a curve to the distal or lingual, or both (Figure 1-7). The overall average length of the maxillary

lateral incisor is 22 mm with an average crown length of 9 mm and an average root length of 13 mm.[4]

Root Number and Form

Anatomical studies indicate that the maxillary lateral incisors are single-rooted virtually 100% of the time[5,7,11,14,31] as shown in Table 1-5. However, numerous case reports demonstrate significant variability in anatomy. Most reported cases of two-rooted maxillary lateral incisors are a result of fusion or gemination and are usually associated with a macrodont crown. There are a few reported cases of two roots associated with normal crown dimensions.[294–297] Anomalous two-rooted maxillary lateral incisors are usually associated with a developmental radicular lingual groove. Even in single-rooted maxillary incisor teeth, a deep gingivo-palatal groove may often result in localized periodontal disease.

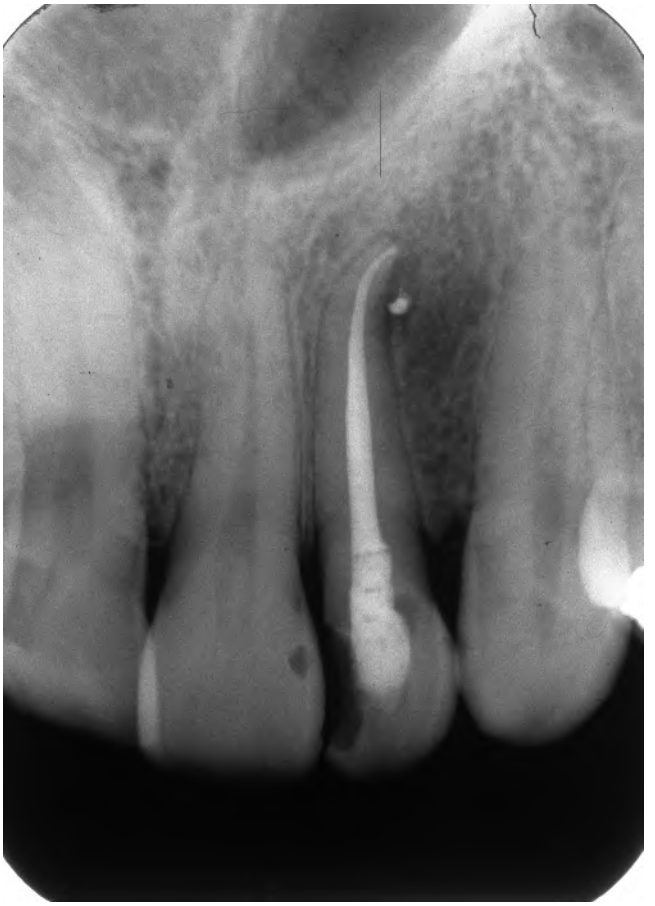


FIGURE 1-7 Maxillary left lateral incisor with moderate "J-shape" apical curvature. (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

TABLE 1-5 Root Number in the Maxillary Lateral Incisor

Maxillary Lateral Incisors Reference		No. of teeth in studies	1 Root	
			%	No. of Teeth
Total number of root studies[5,7,11,14,31,78,298]	7	827	100	827

Canal System

The maxillary lateral incisor usually presents with a single canal as demonstrated by the anatomical studies shown in Table 1-6. Mizutani et al.[214] investigated the anatomic location of the apical foramen in 30 maxillary lateral incisors. The root apex and apical foramen were displaced distolingually in a majority of the specimens. Displacements in all directions were also found in the study. Coincidence of the apical foramen and the anatomic root apex was found in only 2 (6.7%) of the specimens. Therefore, exploration of the apical foramen and constriction, with a fine pre-curved #10 size file tip, delicate tactile sense, and the electronic apex locator, is essential to locate the foramen. The use of CBCT, although not essential, may also help determine the degree and direction of curvatures of the canal system.

An early stereomicroscopic study of root apices by Green[213] found that the average diameter of the major foramen in the 50 maxillary lateral incisors studied was 0.4 mm while the accessory foramina were 0.2 mm or less in diameter. The average distance of the major apical foramen from the anatomical root apex was found to be 0.3 mm. Approximately 10% of the maxillary lateral incisors exhibited accessory foramina.

Variations and Anomalies

One hundred and thirty cases presented in 107 references illustrate the high degree and range of variability in the morphology of the maxillary lateral incisor (Table 1-7). The most common variation reported was dens invaginatus (46 cases), followed by dens evaginatus (talon cusp), palato-gingival grooves, and two roots and two canals in decreasing order of frequency.

Maxillary lateral incisors often present with anomalous anatomy, resulting in diagnostic and treatment challenges. Complications resulting from radicular grooves have been well documented by Simon et al.[221] The incidence of radicular

TABLE 1-6 Main and Apical Canal Number of the Maxillary Lateral Incisor

Maxillary Lateral Incisors References		No. of teeth in studies	1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[5,7,11,14,31,78,194,298]	8	2331	97.4	2271	2.6	91				
Total number of canal apices studies	6	795					98.6	784	1.4	11

grooves is 3.0% in maxillary lateral incisors (Figure 1-8) in a study by Pecora and da Cruz Filho,[65] and there are several case reports in the literature confirming the high probability of this finding.[140,296,299–311] Peikoff and Trott[309] reported the histology of an endodontic-periodontic failure in a maxillary lateral incisor with an accessory root and a radicular groove (Figure 1-9).

An SEM investigation of 14 extracted maxillary lateral incisors with radicular grooves concluded that direct communication between the groove and the pulp was evident in these specimens and that accessory canals were the primary mechanism of communication between the periodontium and the pulp.[140] As with maxillary central incisors, the degree of shovel-shaped feature of the crown is varied and is often based on ethnic background. Again, this feature is common in Asian populations and rare in Caucasian populations.[13,53,216,217,219,220] Reports of this and other coronal anomalies associated with the maxillary lateral incisor are common.

TABLE 1-7 Variations and Anomalies Associated with the Maxillary Lateral Incisor

Anatomic Variation	No. of Cases	No. of References
Dens invaginatus (unclassified)[231,285,310,312–325]	22	17
Talon cusp(s)[37,246,326–329]	16	6
Dens invaginatus Type 3[312,330–342]	14	14
Palatogingival groove [40,299–308]	12	11
2 roots and 2 canals[294,295,297,343–349]	10	10
Dens invaginatus Type 2[350–358]	10	9
1 root and 2 canals[234,265,343,347,359,360,361]	7	7
Gemination[362–366]	6	5
Fusion with a supernumerary tooth[367–371]	6	6
Palatogingival groove and accessory root[296,309–311]	4	4
Dens invaginatus Type 1[356,372]	4	2
Peg laterals (bilateral)[373]	4	1
Accessory root[310,374]	2	2
1 root and 4 canals[375]	1	1
1 root and 2 canals and Dens invaginatus Type 1[266]	1	1
2 roots and 3 canals[376]	1	1
2 roots and complex radicular lingual groove[377]	1	1
2 canals and palatogingival groove[378]	1	1
Dens invaginatus Type 2 and dens evaginatus[379]	1	1
Dens invaginatus Type 2 and transposition with a maxillary canine[65]	1	1
Bilateral hypodontia of maxillary lateral incisors[380]	1	1
Dilaceration[381]	1	1
Macrodont[273]	1	1
Dens invaginatus and dens evaginatus (talon cusp) on the same tooth[382]	1	1
Fusion with a maxillary central incisor[227]	1	1
Double tooth (used when unable to differentiate between germination and fusion)[383]	1	1
Total number of case report references and cases	130	107

Dens invaginatus or "*dens in dente*" can be present in various forms.[65,231,266,285,310,312–325,330–342,350–358,379,382] Oehlers[384] classified dens invaginatus into three types based on the severity of the defect. Type 1 dens invaginatus is an invagination confined to the crown. Type 2 extends past the cementoenamel junction but does not involve periapical tissues. The most severe form and most complex to treat is a Type 3 defect. The invagination extends past the cementoenamel junction and may result in a second apical foramen. Combination of non-surgical and surgical therapy is often used to successfully treat Type 2 and Type 3 cases.[310,312,316,318,320,322–324,331–333,335,336,338,341,342,354,355,358,366] Non-surgical management of the dens invaginatus, including the more severe Type 3 defects, have also been reported.[310,313–315,317,321,325,334,337,339,340,376,385] Occasionally, the vitality of the pulp in the main canal has been shown to be maintained while treating (surgically, non-surgically, or both) the accessory root and canal system, when there has been no communication between the two.[319,323,337–339] (For more details, see Chapter 26, "Endodontic retreatment and management of mishaps.")

Mupparapu et al.[382] reported a rare finding of both dens invaginatus and dens evaginatus associated with one maxillary lateral incisor. It could be noted that most case reports

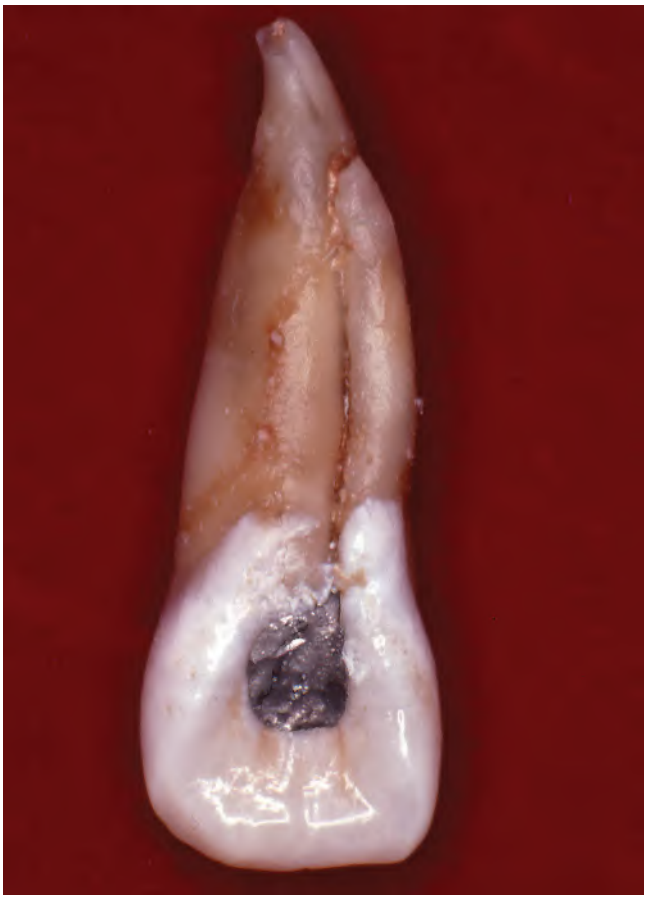


FIGURE 1-8 Radicular developmental groove (lingual view) in a maxillary left lateral incisor caused periodontal failure and was extracted. (Courtesy of Drs. William H. Christie and Colin Dawes, Winnipeg, MB, Canada.)

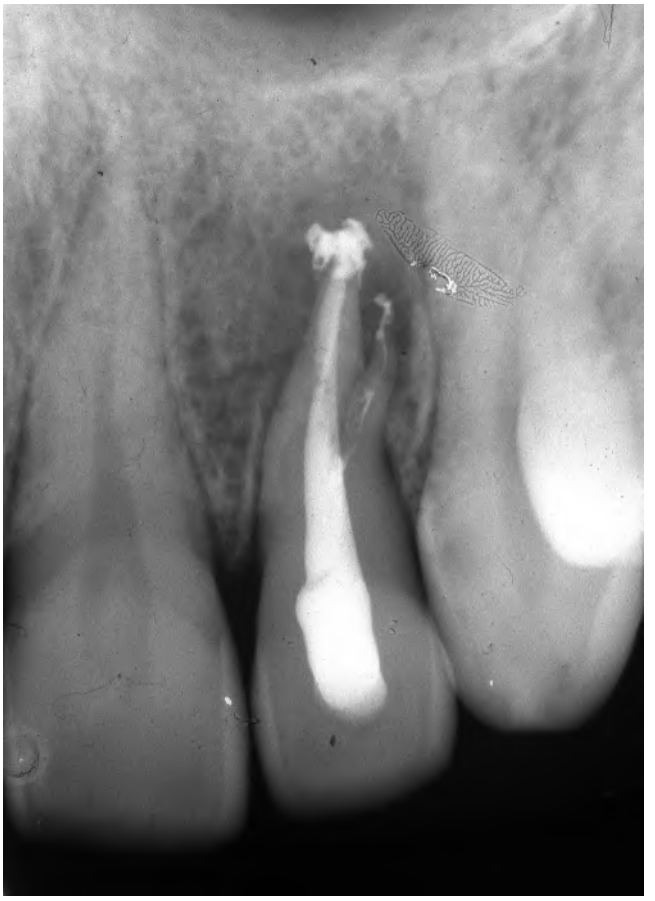


FIGURE 1-9 Maxillary left lateral incisor with accessory root. Note, sealer extruding through accessory root canal. (Courtesy of Dr. Marshall D. Peikoff, Winnipeg, MB, Canada.)

of anomalous root formation in maxillary lateral teeth document single (unilateral) teeth. Kannan et al.[356] documented two unilateral and one bilateral incidence of dens invaginatus in their case report. The presence of bilateral Peg-lateral teeth with conical crowns and small root and root canal system is a common finding.

MAXILLARY CANINE

External Root Morphology

The root of the maxillary canine is oval in shape and tapers toward the lingual as illustrated in Figure 1-10C.[1–4,6,13,15,17] The root is wider labiolingually and is the longest root in the dentition.[4] Prominent developmental depressions that appear as a double lamina dura on periapical radiographs can be present on both its mesial and distal surfaces, especially in the middle third of the root as shown in Figure 1-10A and B.[4,6] The root tip may be blunt or it may end in a fine, often curved tip (Figure 1-10A–C). The overall average length of the maxillary canine is 27 mm with an average crown length of 10 mm and an average root length of 17 mm.[4]

Root Number and Form

Most studies have found that the maxillary canine has a single root 100% of the time[5,7,11,14,31,386] as shown by the

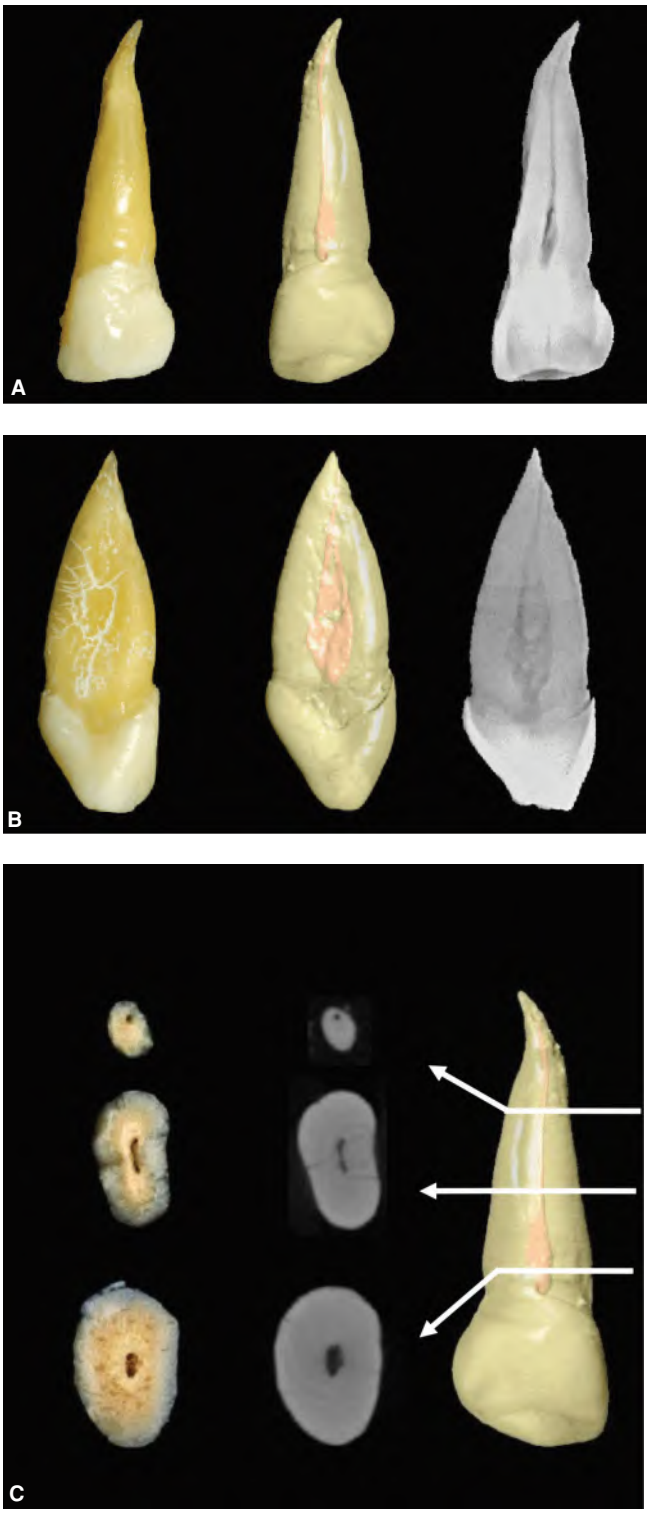


FIGURE 1-10 Maxillary left canine. ***A.*** Labial view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Maxillary Canines-Rotations and Slices.)

anatomic studies in Table 1-8. However, there are case reports of two-rooted maxillary canines.[387,388]

CANAL SYSTEM

The maxillary canine usually has a single canal. A small percentage of maxillary canines have two canals in a figure "8" form (4.7%) as shown in Table 1-9.[5,7,11,14,31,78,298] Of those having two canals, the majority (98.0%) join in the apical third and exit through a single foramen. Accessory (lateral) canals are not uncommon and become evident radiographically as midroot radiolucencies or sealer-puff (Figure 1-11) after completion of root canal treatment. The majority of lateral canals occur in the apical third of the tooth, but midroot lateral canals can also occur (Figure 1-11). Mizutani et al.[214] investigated the anatomic location of the apical foramen in a sample of 30 maxillary canines. The root apex and apical foramen were displaced distolabially in the majority of the specimens. Coincidence of the apical foramen and root apex was found in only 5 (16.7%) of the specimens. The use of the electronic apex locator is more reliable than the radiograph in determining the apical foramen or constriction in most canine teeth.

Green[213] found that the average diameter of the major foramen in 50 maxillary canines studied was 0.5 mm while the accessory foramina were 0.2 mm or less. The average distance of the major apical foramen from the anatomical root apex was found to be 0.3 mm. Approximately 12% of the maxillary canines exhibited accessory foramina.

Variations and Anomalies

The maxillary canine usually presents very few anatomical variations. An extensive literature review found 17 case reports (Table 1-10). When variations do occur, the root is more frequently affected than the crown. However, there are occasional reports of both coronal and radicular anomalies. The root can be dilacerated[16] or can have extreme variations in length.[2,4,6,13,16] One individual with an extremely long root (41 mm) was reported by Booth.[389] His patient was a 5′2″ height, 31-year-old woman of Dutch ancestry.

Dens evaginatus, in a form of a lingual tubercle or talon cusp, was the most common variation or anomaly found associated with the maxillary canine. There are also reports of labial tubercles.[400] Dankner et al.,[40] in an extensive literature review and radiographic examination of 15,000 anterior teeth, found the overall incidence of dens evaginatus to be ~1%. Their survey found no occurrences of dens evaginatus in the maxillary canine. Between 1970 and 1995, there were only four case reports of dens evaginatus in the maxillary canine in their review.

TABLE 1-8 Root Number in the Maxillary Canine

Maxillary canines references		No. of teeth in studies	1 Root	
			%	No. of Teeth
Total number of root studies[5,7,11,14,31,78,298]	7	842	100	842

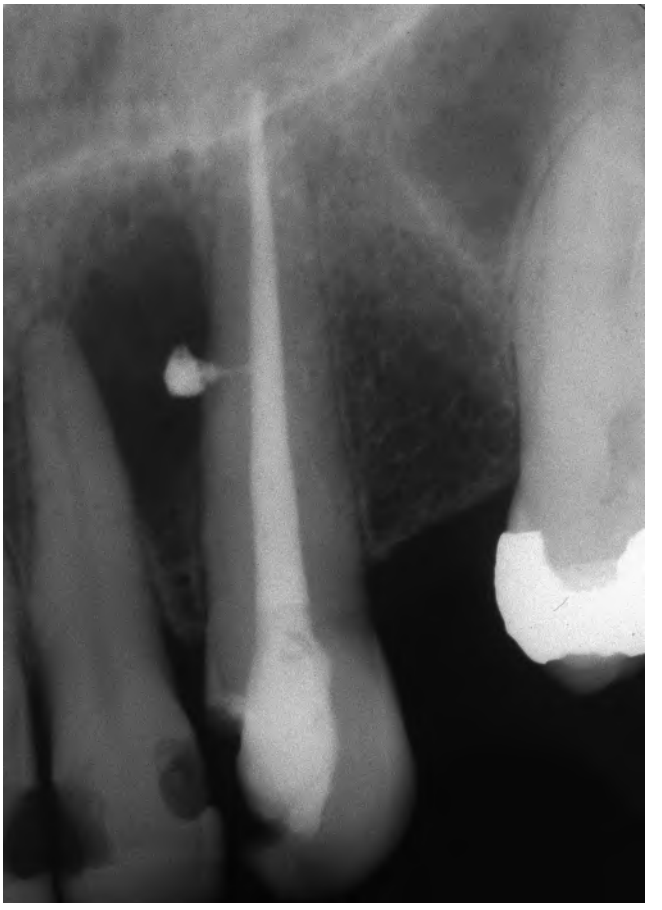


FIGURE 1-11 Maxillary left canine. Sealer extrusion demonstrates the presence of a large mid-root lateral canal system. (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

TABLE 1-9 Main and Apical Canal Number of the Maxillary Canine

Maxillary Canines References		No. of teeth in studies	1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[5,7,11,14,31,78,141,194,298]	9	2615	95.30	2492	4.70	123				
Total number of canal apices studies	5	745					98.02	1039	1.98	21

TABLE 1-10 Variations and Anomalies Associated with the Maxillary Canine

Maxillary Canines Anatomic Variation	No. of Cases	No. of References
Dens invaginatus Type 3[390–394]	5	5
Dens invaginatus[235,395]	2	2
1 root and 2 canals[396,397]	2	2
Talon cusp (dens evaginatus)[246,328]	2	2
2 rooted canine[388,387]	2	2
Bilateral supernumerary canines[398]	1	1
Canine gigantism and canine-premolar transposition[399]	1	1
Labial talon cusp (dens evaginatus)[400]	1	1
Macrodont[389]	1	1
Total number of case report references and cases	17	17

Other variations reported in the literature include case reports with one root and two canals,[396,397] two roots,[387,388] dens invaginatus,[235,390–395] and bilateral, supernumerary canines in a non-syndrome case.[398] The two-rooted maxillary canine should be considered extremely rare, unlike the two-rooted mandibular canine, which in some genetic populations can be a more frequent anomaly.[5,401]

MAXILLARY FIRST PREMOLAR

External Root Morphology

The root anatomy of the maxillary premolar can vary depending on whether one, two, or three roots are present (Figures 1-12, 1-13, 1-14). Also, a variation may exist in the depth of the bifurcation or trifurcation of the root trunk. The more common two-rooted form is illustrated in Figure 1-12. As with all multirooted teeth, two radiographs exposed at different angulations or even CBCT images (if indicated), may be needed to better understand the actual root and canal morphology.

The maxillary first premolar does exhibit ethnic variations with respect to root number (Table 1-11). There are some common features to the various forms. The overall length of the maxillary first premolar is 22.5 mm with an average crown length of 8.5 mm and an average root length of 14 mm.[4]

Prominent root concavities are present on both the mesial and distal surfaces of the root trunk. The mesial root concavity is more prominent and extends onto the cervical third of the crown.[1,3,4,6,13,15,17,402] This results in a root that is broad labiolingually and narrow mesiodistally with a kidney shape when viewed in cross-section at the cementoenamel junction.[4] These anatomical features have implications in restorative dentistry and in periodontal treatment, and are common areas for inadvertent endodontic root perforations.

Gher and Vernino[403] examined the external root shape as it is related to the development of periodontal defects. The

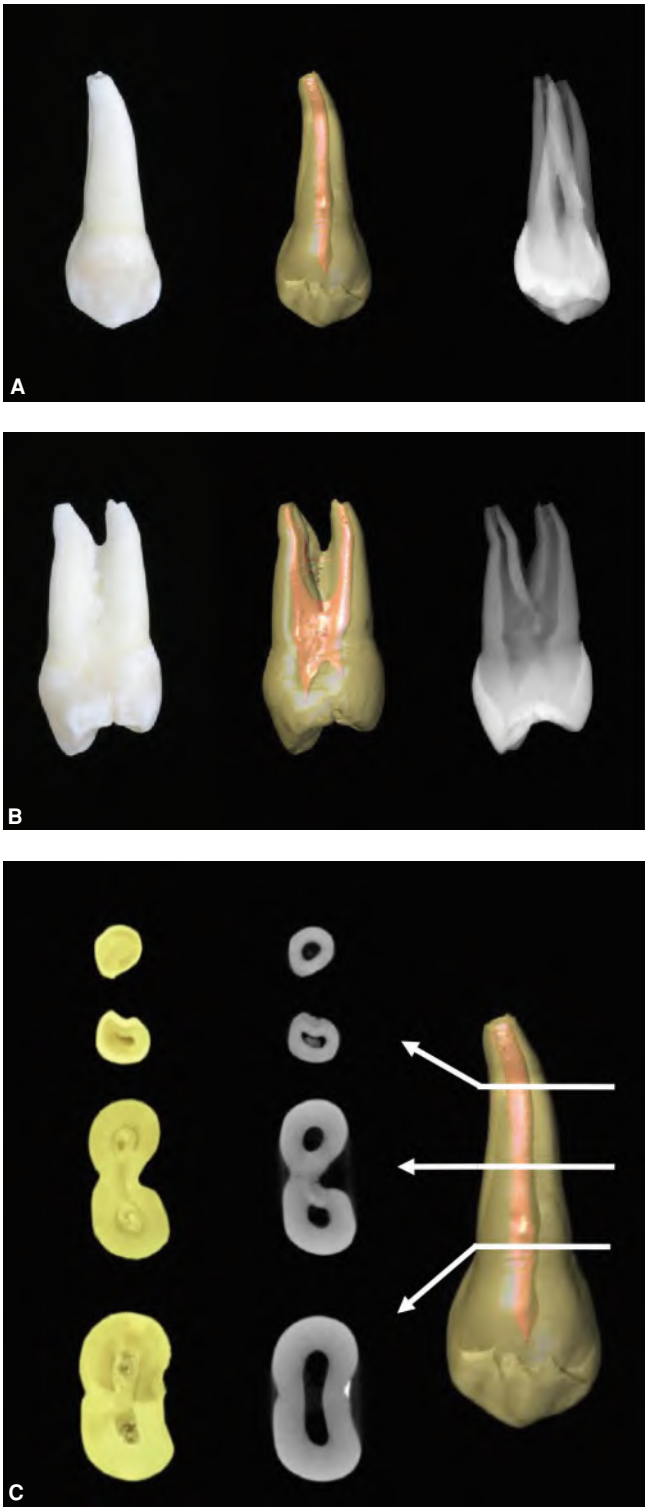


FIGURE 1-12 Maxillary right first premolar. ***A.*** Buccal view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Maxillary First Premolar- Rotations & Slices.)

palatal aspect of the buccal root tip of two-rooted maxillary first premolars usually has a deep longitudinal depression along its length. About 78% of the 45 teeth in their study

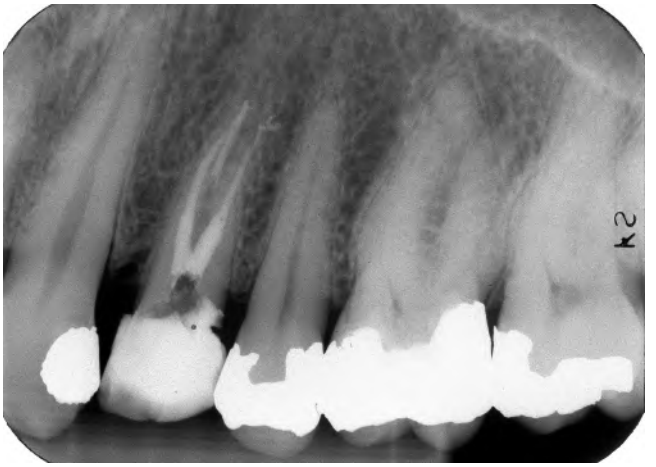


FIGURE 1-13 Maxillary left first premolar with three roots and three canals (MB, DB, and Palatal). Palatal root has a coronal post-space preparation. (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

exhibited a buccal furcation groove. They concluded that a buccal root furcation groove was a normal anatomical feature in those two-rooted maxillary first premolars that have well-formed buccal and lingual roots. The groove was not generally found in those two-rooted specimens with a furcation located in the apical third of the root. A study by Joseph et al.[404] found an incidence of the buccal furcation groove in 62% of teeth with bifurcated roots.

Root Number and Form

The majority of anatomic studies found that the most common form of the maxillary first premolar is the two-rooted form (Table 1-11). There was a wide variation in incidence of the number of roots in the anatomical studies cited. The incidence of three-rooted maxillary first premolars ranged from 0%[5,54,69] to 6%.[135] The ethnic background of the patients in many of the studies was not identified. The majority of studies reporting that the two-rooted form was most common had a probable Caucasian cohort, forming the majority of the population. Studies, which do identify ethnic background, have demonstrated distinct differences between Asian and Caucasian populations. Single-rooted maxillary first premolars are the dominant forms in Asian population[54,94,138,180] and three-rooted forms are rarer by 1:4 ratio.[54,69,138,180] Because of the significant influence of ethnic background in root number for this tooth, Table 1-11 reports the means for all studies and separates the data between Asian and North American Native populations (both Sundadont and Sinodont Mongoloid) versus non-Asian populations.[405,406]

FIGURE 1-14 Extracted maxillary right first premolar with three very fine roots (MB, DB, and Palatal). (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

In addition to ethnic differences, studies vary in their definition, or do not identify what constitutes a bifurcated root. Walker[54] utilized Turner's classification and does not consider a root to be separate unless it is a distinct and separate root for at least half of the overall root length. However, Loh[69] grouped teeth into one root, two roots (including two-root distinct and two-root fused), and three roots. The

TABLE 1-11 Root Number in the Maxillary First Premolar

Maxillary First Premolars References		No. of teeth in studies	1 Root		2 Roots		3 Roots		>3 roots	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of root studies (all studies)	18	7628	53.2	4055	45.6	3480	1.2	93	0.0	0
Total number of root studies (Non-Asian and NA Native populations)[5,76,88,89,103,109,113,125,135,407–410]	13	2647	37.0	978	60.8	1608	2.3	61	0.0	0
Total number of root studies (Asian and NA Native populations)[54,69,94,180,138]	5	4981	61.8	3077	37.6	1872	0.6	32	0.0	0

TABLE 1-12 Main and Apical Canal Number of the Maxillary First Premolar

Maxillary First Premolars References		No. of teeth in study	1 canal		2 canals		3 canals		Other canal configurations		1 canal at apex		2 canals at apex		3 canals at apex		Other configurations at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies (all studies)	21	6618	16.0	1061	80.8	5348	1.3	89	1.8	120								
Total number of canal apices studies (all studies)	11	2429									24.2	587	73.6	1787	0.5	11	0.2	4
Total number of root studies (Non-Asian and NA Native populations)[5,7,8,11,31,76,88,89,103,109,113,135,167,185,408–410]	17	5044	10.4	526	86.3	4353	1.6	83	1.6	82								
Total number of root studies (Asian and NA Native populations)[54,69,78,94]	4	1574	34.0	535	63.2	995	0.4	6	2.4	38								

two-root fused category was defined as teeth with roots joined almost to the apex and with two canals at their origin. The normal external three-root anatomy configuration is that of a mesiobuccal, distobuccal, and palatal root with morphology similar to that of a miniaturized maxillary three-rooted molar.

Sabala et al.[22] studied all aberrant root and root canal morphology in 501 patient records. The occurrence of the same aberration on the contralateral tooth varied according to the type of anomaly. In four of 501 patients, who were found to have three-rooted maxillary first premolars, all were bilateral. Their study found that the more rare the anomaly, the greater the incidence of the anomaly occurring bilaterally. Anomalies occurring <1% were found to occur bilaterally up to 90% of the time.

Canal System

The majority of maxillary first premolars were found to have two canals, whether the tooth had a single- or double-root morphology (Table 1-12). In 21 anatomical reports assessed, over 80% of the teeth had two canals.[5,7,8,11,31,54,69,76,78,88,89,94,103,109,113,135,167,185,408–410] Ethnicity was a factor affecting the canal number. The incidence of a Vertucci Type I single canal was significantly higher in Asian populations compared to mixed non-Asian populations.[54,69] The Vertucci and Weine classification systems are useful in exploring any canal systems with double canals in single roots for endodontic therapy.[14,122]

All the three-rooted first premolars in the anatomical studies were found to have a single canal in each root. The incidence of three canal maxillary premolar teeth ranged from 0%[54,69,409,410] to 6%.[135] This finding is a rare occurrence in Asian populations.[54,69]

Regardless of the number of roots, the majority of maxillary first premolars (80.8%) had two separate canals and 73.6% had two separate foramina at the apex.

TABLE 1-13 Variations and Anomalies Associated with the Maxillary First Premolar

Maxillary First Premolars Anatomic Variation	No. of Cases	No. of References
3 roots and 3 canals[411–423]	20	13
Furcation groove on the palatal aspect of the buccal root of 2-rooted maxillary first premolars[123,404,403]	3	3
Dens evaginatus[424,425]	2	2
2 roots and 3 canals[426]	1	1
Gemination[427]	1	1
Total number of case report references and cases	27	20

Variations and Anomalies

The most common variation reported for the maxillary first premolar was three roots and three canals (Table 1-13). Ethnic differences were found (Table 1-11). The study by Loh[69] reported a much lower incidence (50.6%) of the two-rooted maxillary first premolars in a Singaporean population compared to Sabala et al.'s study[22] (98.4%) based in the United States. Tratman[53] reported that the two-rooted first premolar was very uncommon in Asian stock as did

Petersen[51] in his study of the East Greenland Eskimo dentition. A more recent large anatomical study of 3202 maxillary first premolars in Japan by Aoki,[138] found that 65.7% of the specimens were single rooted.

Three-rooted forms were reported to be rare variations in Asian populations.[418,419,423] Mattuella et al.[426] reported a variation of the maxillary first premolar with a longitudinal sulcus on the buccal surface of the buccal root. This variation resulted in a two-canal system in the buccal root as the root canal system narrowed with age and thus, three canals in teeth with two roots. Gemination of the maxillary first premolar is rarely reported.[427]

Taurodontism is another rare anomaly in premolars, as a group, and less common in the maxillary premolars compared to their mandibular counterparts. Taurodontism of roots and canal systems is explained in greater detail in the section on maxillary first molars. Llamas et al.[428] found only three taurodont-like maxillary premolars in a sample of 379 extracted maxillary and mandibular premolars. Of the 16 cases of taurodontism in Shifman et al.'s review of radiographs, over a five-year period, none were maxillary first premolars.[429] Madeira et al.[430] studied 4459 premolars and found a total of 11 taurodont mandibular premolars (seven mandibular first premolars and four mandibular second premolars), but none in the maxillary premolars. Llamas et al.[428] reported three taurodont maxillary first premolars of the 379 teeth.

The Weine Type IV root canal system (Vertucci's Type V), with a wide buccolingual canal that branches into two apical canals and foramina in the apical third, may sometimes be confused as taurodont-like root canal anatomy, when it occurs in single rooted maxillary premolar teeth.

MAXILLARY SECOND PREMOLAR

External Root Morphology

The cross-sectional root anatomy of the maxillary second premolar in the mid-root area is described as oval or kidney shaped.[1,3,4,6,13,15–17] Developmental depressions are often present on the mesial and distal aspects of the root, but generally less pronounced than on the maxillary first premolar. The typical single root trunk is broad buccolingually and is narrower mesiodistally (Figure 1-15). The root tip usually ends as a single blunt apex, but may be fine and divide into two or more, rarely three, apices. Curvature in the apical third is also not uncommon. The overall average length of the maxillary second premolar is 22.5 mm with an average crown length of 8.5 mm and an average root length of 14 mm.[4]

Root Number and Form

All of the anatomical studies cited found that the most common form of the maxillary second premolar is a single root (Figure 1-15). The mean for data from eight anatomical studies of 9033 teeth is summarized in Table 1-14, resulting in a 91.5% incidence of single-rooted teeth. The incidence of two-rooted maxillary second premolars ranged from 1.6%[410] to 20.4%,[408] while the three-rooted form was a rare finding and ranged from 0%[5,87,410,431,432] to 1%.[408] Bilateral

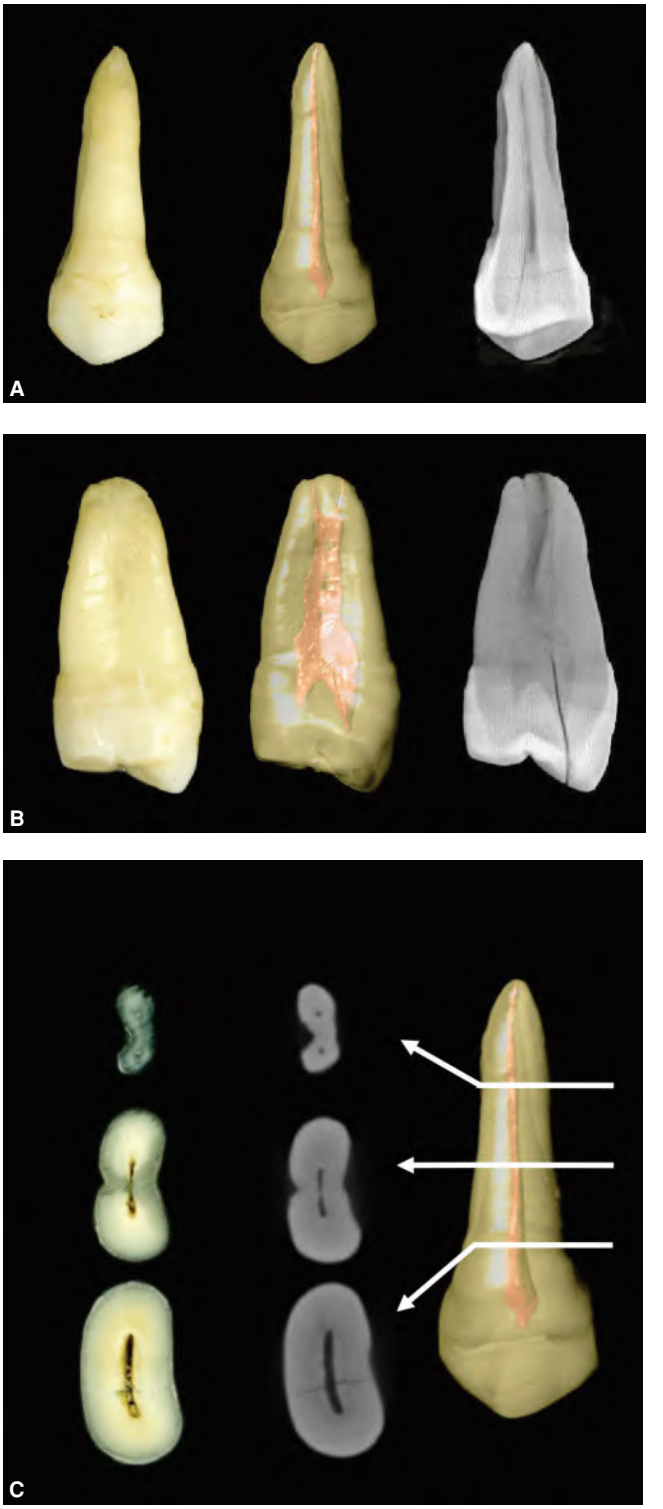


FIGURE 1-15 Maxillary left second premolar. ***A.*** Buccal view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Maxillary Second Premolar- Rotations & Slices.)

TABLE 1-14 Root Number in the Maxillary Second Premolar

Maxillary Second Premolars References		No. of teeth in studies	1 Root		2 Roots		3 Roots	
			%	No. of teeth	%	No. of teeth	%	No. of teeth
Total number of root studies[35,43,87,138,387,408,410,431]	8	9033	91.5	8263	8.4	756	0.2	14

TABLE 1-15 Main and Apical Canal Number of the Maxillary Second Premolar

Maxillary Second Premolars References		No. of teeth in studies	1 canal		2 canals		3 canals		Other canal configurations		1 canal at apex		2 or more canals at apex	
			%	No. of teeth	%	No. of teeth	%	No. of teeth	%	No. of teeth	%	No. of teeth	%	No. of teeth
Total number of canal studies[7,11,14,31,78,80,87,89,167,185,408–410,431,433]	16	4829	51.8	2502	46.2	2233	0.8	38	2.0	56				
Total number of canal apices studies	12	2470									63.6	1571	36.0	890

symmetry of the three-rooted form may be expected in many patients, as well as this anomaly occurs in both first and second premolar teeth.[22]

Canal System

The maxillary second premolar has a single canal in ~51.8% of the 4829 teeth examined in 16 anatomical studies (Table 1-15).[7,11,14,31,78,80,87,89,167,185,408–410,431,433] Therefore, even though over 90% of these teeth have a single root, a high proportion will have two canals present. Canal exploration of maxillary second premolar teeth should be done with fine curved files, keeping in mind many types of the Vertucci's[14] or Weine's[122] classification of "two canals in one root" that may not always be apparent on the radiograph. The incidence of three canals was low in each of the anatomical studies. The majority of the teeth studied (63.6%) had a single canal and foramen at the apex.

Variations and Anomalies

Relatively few case reports of variations and anomalies in the maxillary second premolar have been published (Table 1-16). The most common variation reported is the maxillary second premolar with three roots and three canals.[419,433–440] These case reports also include examples of dens invaginatus,[441] a deep distal root concavity,[442] dens evaginatus,[443,425] taurodontism,[429] two roots, and three canals.[6]

Three-rooted maxillary second premolar teeth do not seem to be as common as in first premolars. This anomaly is often bilateral and should be considered by exposing radiographs taken at different angles. Both first and second premolar triple-canal system anomalies do occur, but rarely.[419]

TABLE 1-16 Variations and Anomalies Associated with the Maxillary Second Premolar

Maxillary Second Premolars Anatomic Variation	No. of Cases	No. of References
3 roots (MB, DB and P) and 3 canals[419,433–440]	13	9
Dens evaginatus[425,443]	2	2
Dens invaginatus[441]	1	1
1 canal and multiple foramina[437]	1	1
2 canals and 1 foramen[437]	1	1
2 canals and 2 foramina[437]	1	1
2 canals, 1 S-shaped canal[444]	1	1
Deep distal root concavity[442]	1	1
Taurodontism[429]	1	1
Total number of case report references and cases	22	18

MAXILLARY FIRST MOLAR

External Root Morphology

The maxillary first molar normally has three roots (Figure 1-16). The mesiobuccal root is broad buccolingually and has prominent depressions or flutings on its mesial and distal surfaces.[1,3,4,6,15–17] The internal canal morphology is highly variable but the majority of the mesiobuccal roots contain two canals. The distobuccal root is generally rounded or ovoid in cross-section and usually contains a single canal. The palatal root is more broad mesiodistally than buccolingually and ovoidal in shape but normally contains only a single large canal. Although the palatal root generally appears straight on radiographs, there is usually a buccal curvature in the apical third.[445] Depressions on the

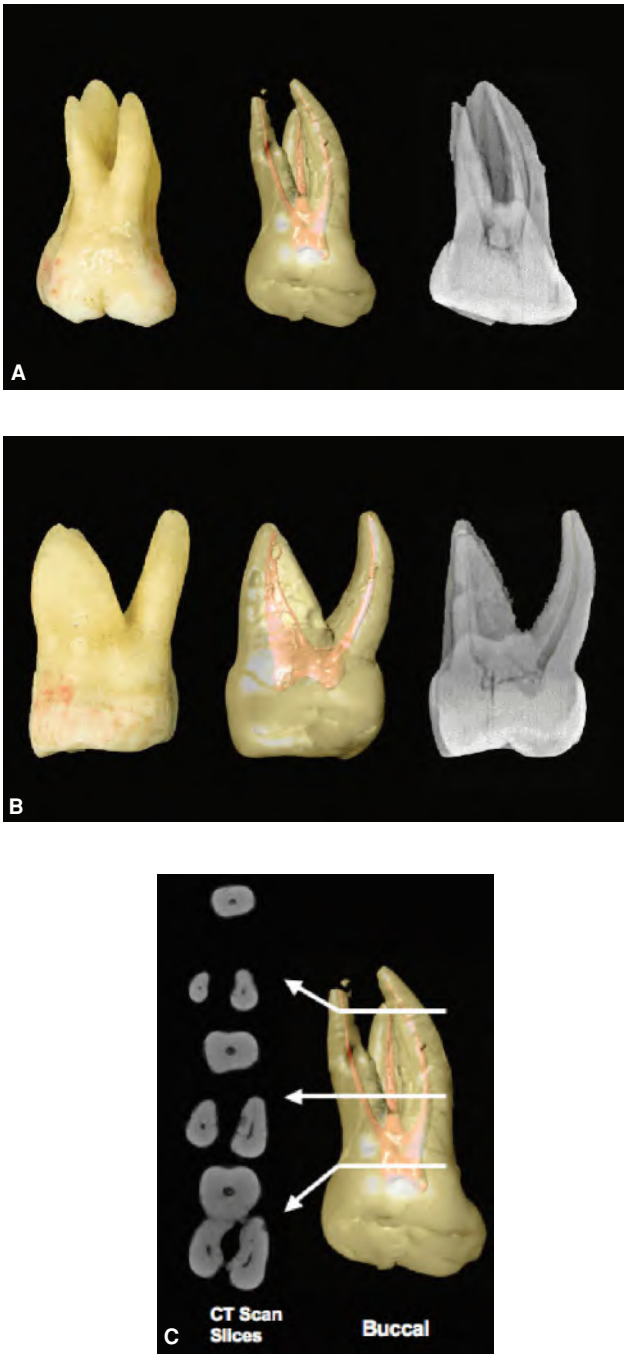


FIGURE 1-16 Maxillary right first molar. ***A.*** Buccal view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Maxillary First Molar- Rotations & Slices.)

buccal and palatal surfaces of the palatal root can be present but are generally shallow. Gher and Vernino[403] found prominent depressions not only on the mesial aspect but also on the distal aspect of the mesiobuccal roots. Shallow depressions could also be found on the furcal side of the distobuccal and palatal roots.

The overall average length of the maxillary first molar is 20.5 mm with an average crown length of 7.5 mm and an average root length of 13 mm.[4]

Root Number and Form

The maxillary first molar root anatomy is predominantly a three-rooted form, as shown in all anatomic studies of this tooth (Table 1-17). The two-rooted form is rarely reported, and may be due to fusion of the distobuccal root with palatal root or the distobuccal root with the mesiobuccal root. The single root or conical form of root anatomy in the first maxillary molar is very rarely reported.[5] Over 97.7% of maxillary first molars had three roots and 2.1% had two roots in 12 studies that included 2744 teeth. The four-rooted anatomy in its various forms is also very rare in the maxillary first molar and is more likely to occur in the second or third maxillary molars.[446,447]

The incidence of fusion (Table 1-18) of any two or three roots was ~5.9%.[18–23,182,448,449] The "C" shape canal morphology in the maxillary first molar is also a rare anomaly with an incidence varying from 0.09% to 0.3%.[449,450]

Canal System

Internal root canal system morphology reflects the external root anatomy. The mesiobuccal root of the maxillary first molar contains a double root canal system more often than a single canal according to most of the anatomical studies. This review contains data on the canal morphology of the mesiobuccal root that included a total of 14,346 teeth from 71 studies (Table 1-19). The incidence of two canals in the mesiobuccal root was 59.4% and of one canal was 40.6%. The incidence of two canals in the mesiobuccal root was somewhat higher in laboratory studies (64.0%) compared to clinical studies (56.8%).[11,30,132,139,143,148,183,451–454] Less variation was found in the distobuccal and palatal roots (Table 1-19). One canal was found in the distobuccal root in 98.8% of teeth, whereas the palatal root had only one canal in 99.3% of the teeth studied.[62]

Earlier *in vitro* studies of the mesiobuccal root canal system were slightly more likely to report two canals in the maxillary first molar than the *in vivo* clinical studies, but the incidence of location of a two-canal system in both clinical

TABLE 1-17 Root Number in the Maxillary First Molar

Maxillary First Molars References		No. of teeth in studies	1 Root %		2 Roots %		3 Roots %	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of teeth in root studies[5,18,19,23,107,142,171,174,175,182,184,193]	12	2744	0.15	4	2.1	58	97.7	2682

and laboratory studies appears to be increasing with the routine use of the SOM and other aids during the modified endodontic access opening procedure.[12,459] The diagnostic tool that has had the greatest impact in both clinical and laboratory studies is CBCT. The Clearing Technique has been the "gold standard" in laboratory studies, and the CBCT is rapidly becoming the "gold standard" in clinical studies as well as in laboratory studies.[198–201,203–207,462–464] However, it should be noted that nine of the *in vitro* laboratory studies reported an incidence of second mesiobuccal canal (MB-2) in 80% to 96% of the time.[11,132,139,143,148,451–454] Four were clearing studies,[11,139,451,454] two utilized CT,[143,148] and three used either an SOM or surgical telescopes.[132,452,453] Two of the *in vivo* clinical studies reported an incidence of MB-2 of >80%.[30,183] One study was a clinical RCT study[30] and the other utilized CBCT.[183]

The two-canal system of the mesiobuccal root of the maxillary first molar has a single apical foramen ~59.7% of the time (Table 1-19).

The single-canal system and single apical foramen in the distobuccal and palatal root of the maxillary first molar is the most predominant form, as reported in all studies, but

TABLE 1-18 Incidence of Fused Roots in the Maxillary First Molar

Maxillary First Molars References		No. of teeth in studies	Total Incidence of Fused Roots	
			%	No. of Teeth
Total number of teeth in fused root studies[18–23,182,448,449]	9	2014	5.9	119

TABLE 1-19 Main and Apical Canal Number of the Maxillary First Molar

Mesiobuccal Root of the Maxillary First Molar Number of Canals and apices References		No. of teeth in studies	1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of teeth	%	No. of teeth	%	No. of teeth	%	No. of teeth
Total number of canal studies (lab studies)[7–11,14,18,19,23,27,31,58,63,73,78,81,106,107,122,127,132,133,139,143,148,152,157,158,451–458]	41	5083	36.0	1828	64.0	3255				
Total number of canal apices studies (lab studies)	27	2956					60.6	1790	39.4	1166
Total number of canal studies (clinical studies)[12,25–28,30,64,127,170–172,174,175,182-184,188,189,193,197,198,408,459–461]	30	9263	43.2	3998	56.8	5265				
Total number of canal apices studies (clinical studies)	11	4883					58.7	2865	41.3	2018
Total number of canal studies (all studies)	71	14346	40.6	5826	59.4	8520				
Total number of canal apices studies (all studies)	38	7839					59.4	4655	40.6	3184
Distobuccal Root of the Maxillary First Molar Number of Canals and Apices (clinical and laboratory studies) References										
Total number of canal studies (all studies)[7,8,11,14,18,19,23,31,63,78,81,107, 127,171,174,175,182,184,189,193,408,456-458,460]	26	5758	98.8	5687	1.2	71				
Total number of canal apices studies (all studies)	16	3540					98.5	3487	1.5	53
Palatal Root of the Maxillary First Molar Number of Canals and Apices (clinical and laboratory studies) References										
Total number of canal studies (all studies)[7,8,11,14,18,19,23,31,63,78,81,107,127,171, 174, 175, 182, 184, 189, 193, 408,456–460]	26	5758	99.3	5717	0.7	41				
Total number of canal apices studies (all studies)	16	3540					99.1	3507	0.9	33

multiple canals and more than one apical foramen variation does exist in ~1% of these roots in the studies reported (Table 1-19).

Variations and Anomalies

The root and canal morphology of molar teeth varies greatly. Many studies provided no information on ethnic background, age or gender, or possible explanations for variation observed. Walker[54,55,79] reported on the ethnic differences in the root anatomy of maxillary first premolars, mandibular first premolars, and the high incidence of three-rooted mandibular first molars in Asian patients. However, he did not report on the incidence of a MB-2 in the maxillary first molar. A study by Weine et al.[58] determined that the incidence of MB-2 in a Japanese population was similar to the incidence reported for other ethnic backgrounds.

Age was found to have an effect on the reported incidence of MB-2. Fewer canals were found in the MB root due to increasing age and calcification.[28,30,139]

Sert and Bayirli[11] conducted a clearing study that identified gender in a sample of 2800 teeth from Turkish patients. Although only 100 of each type of tooth for each gender was included in their study, a single Vertucci's Type I canal was present in the mesiobuccal root in only 3% of men compared to 10% of women. There are conflicting results with respect to gender and the number of canals in other studies.[7,11,28,30]

Some studies[27,64] compared *in vivo* versus *in vitro* techniques. Seidberg et al.,[27] in an *in vivo* study, reported 33.3% of the 201 teeth studied had an MB-2 canal. This increased to 62% in their *in vitro* study of 100 teeth. Similar results were reported in a study by Pomeranz and Fishelberg.[64] Only 31% of 100 teeth studied had an MB-2 canal in their *in vivo* study compared to 69% of 100 teeth in their *in vitro* study. The *in vitro* portion of this study described the samples as extracted maxillary molars and may represent pooled data instead of maxillary first molar data alone. The definition of a canal as a treatable canal used in clinical studies[12,28] versus the more complex canal configurations visible through clearing studies[7,11,14,18,63] can also lead to different results.

The more common use of SOM or loupes in recent clinical studies has resulted in an increased prevalence of the clinical detection of the MB-2 canal on the floor of the pulp chamber.[12,25,132,197] The effect of magnification on the incidence of clinical location of MB-2 was assessed in a clinical study by Buhrley et al.[197] The MB-2 canal was found in 71.1% of teeth when using SOM. The group using loupes found MB-2 in 62.5%. The lowest incidence of MB-2 was in the group performing RCT without any magnification. MB-2 was found in only 17.2%. A study by Sempira and Hartwell[25] found that use of an SOM did increase the incidence of finding MB-2. They attributed relatively low incidence in their study due to their characterization of a canal as one that must be negotiated and obturated to within 4 mm of the apex.

CBCT is being utilized more frequently now in both clinical and laboratory studies as well as in clinical practice to assist in identifying both root and root canal morphology.[107,148,173–175,182–184,188,189,193,198,207,465–488] As a result, the incidence of MB-2 as well as other canals detected is increasing. Eder et al.[143] reported an incidence of 94.1% of MB-2 in an *in vitro* study, while Reis et al.[183] reported an incidence of 88.5% in a recent clinical study using CBCT.

The incidence of many of the anomalies cannot be determined due to the lack of data collection and population size. However, although rare, these anomalies can and do occur. Ninety-six case reports were reviewed and the most common anomalies associated with the maxillary first molar were two palatal canals in three-rooted teeth, taurodontism (Figure 1-17), and various forms of root fusion (Table 1-20).

Of all the canals in the maxillary first molar, the MB-2 can be the most difficult to find and negotiate in a clinical situation. Knowledge from laboratory studies is essential to provide insight into the complex root canal anatomy. A study by Davis et al.[536] compared the post debridement anatomy of the canals of 217 teeth. Injection of silicone impression material into the instrumented canals revealed that standard instrumentation left a significant portion of the canal walls untouched. Fins, webbing, and portions of the canal were sometimes found not fully instrumented. Clinical instrumentation of this tooth, especially with respect to the mesiobuccal root, can be complicated. Failure to detect and treat the second MB-2 canal system will result in a decreased long-term prognosis.[170] Stropko[12] observed that by scheduling adequate clinical time, by using the recent magnification and detection instrumentation aids, and by having thorough knowledge of how and where to search for MB-2, the rate of location can approach 93% in maxillary first molars.

Taurodontism or "bull-like" teeth was first described by Keith.[537] Initially, it was considered to be a primitive trait found mainly in Neanderthal skulls from Krapina and other archaeological sites. Shaw et al.[495,538,539] classified the "modern" molar form as cynodont and classified taurodontism into hypo-, meso- and hypertaurodontism. There are several case reports that have described successful endodontic treatment on taurodont-like teeth.[540–545] Other case reports linked

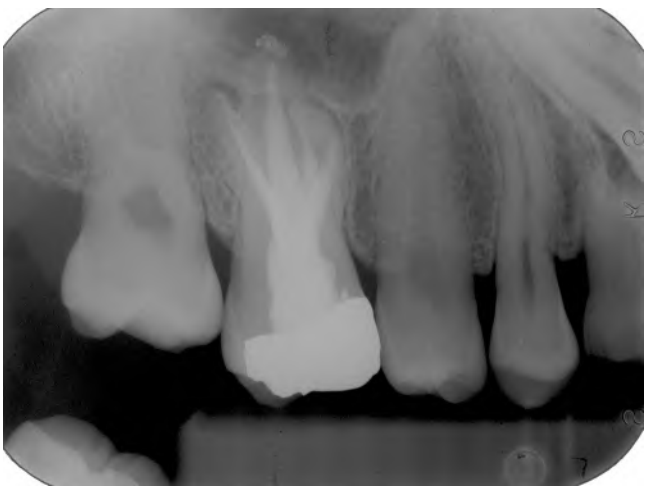


FIGURE 1-17 Mesotaurodontism in a maxillary right first molar. (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

TABLE 1-20 Variations and Anomalies Associated with the Maxillary First Molar

Maxillary First Molars Anatomic Variation	No. of Cases	No. of References
3 roots (MB, DB and Palatal) and 4 canals (MB, DB and 2 Palatal)[446,489–494]	13	7
Taurodontism[429,478,495–497]	10	5
Fused roots and C-shaped canal[471,479, 498–501]	9	7
4 roots (MB, DB and 2 Palatal) and 4 canals (MB, DB and 2 Palatal)[447,474,490, 502–505]	8	7
3 roots (MB, DB and Palatal) and 6 canals (2MB, 2DB and 2 Palatal)[466,472,506]	7	3
1 root and 1 canal[470,483,507–511]	7	7
2 roots (1B and 1 Palatal) and 2 canals (1B and 1 Palatal)[481,512,513]	5	3
3 roots (MB, DB and Palatal) and 5 canals (3MB, DB and Palatal)[465,514–516]	5	4
3 roots (MB, DB and Palatal) and 5 canals (3MB, DB and Palatal)[517–519]	3	3
3 roots (MB, DB and Palatal) and 5 canals (2MB, DB and 2 Palatal)[520–522]	3	3
3 roots (MB, DB and Palatal) and 7 canals (3MB, 2DB and 2 Palatal)[477,468,475]	3	3
3 roots (MB, DB and Palatal) and 5 canals (2MB, 2DB and Palatal)[523,524]	2	2
3 roots and (MB, DB and Palatal) and 5 canals (2MB, DB and 2 Palatal)[482,486]	2	2
3 roots (MB, DB and Palatal) and 6 canals (3MB, 2DB and 1 Palatal)[525]	2	2
2 roots (B and Palatal)[526]	1	1
2 roots (B and Palatal) and 3 canals (1B and 2 Palatal)[467]	1	1
3 roots (1 fused B root and 2 Palatal roots) and 4 canals (2B and 2 Palatal)[527]	1	1
3 roots (MB, DB and Palatal) and 5 canals (MB, DB and 3 Palatal)[528]	1	1
3 roots (MB, DB and Palatal) and 6 canals (2MB, 2DB and 2 Palatal)[529]	1	1
3 roots (MB, DB and Palatal) and 6 canals (3MB, DB and 2 Palatal)[530]	1	1
3 roots (MB, DB and Palatal) and 6 canals (2MB, 3DB and 1 Palatal)[472]	1	1
3 roots (MB, DB and Palatal) and 6 canals (2MB, DB and 3 Palatal)[531]	1	1
3 roots (MB, DB and Palatal) and 8 canals (3MB, 3DB and 2 Palatal)[474]	1	1
4 roots (MB, DB and 2 Palatal)[532]	1	1
4 roots (2MB, DB and Palatal) and 4 canals (2MB, DB and Palatal)[473]	1	1
4 roots (2MB, DB and Palatal) and 5 canals (2MB, 2DB and 1 Palatal)[533]	1	1
4 roots (MB, DB and 2 Palatal) and 6 canals (2MB, 2DB and 2 Palatal)[485]	1	1
4 roots (MB, DB, Palatal and MP roots) and 6 canals (MB, 2DB, Palatal and 2MP)[534]	1	1
5 roots (2MB, DB and 2 Palatal) and 5 canals (2MB, DB and 2 Palatal)[504]	1	1
Fusion with a supernumerary tooth[484]	1	1
Fusion with a maxillary second premolar[535]	1	1
Total number of case report references and cases	96	75

taurodontism to certain inherited genetic syndromes.[543,546] (For more details, see Chapter 26, "Endodontic retreatment and management of mishaps.")

MAXILLARY SECOND MOLAR

External Root Morphology

The maxillary second molar normally has three roots (Figure 1-18 and Table 1-21). The relative shape of each of the roots is similar to the maxillary first molar but the roots tend to be closer and there is a higher tendency toward fusion of two or three roots.[16] There is usually more of a distal inclination to the root or roots of this tooth compared to the maxillary first molar.[16] There is also a general reduction

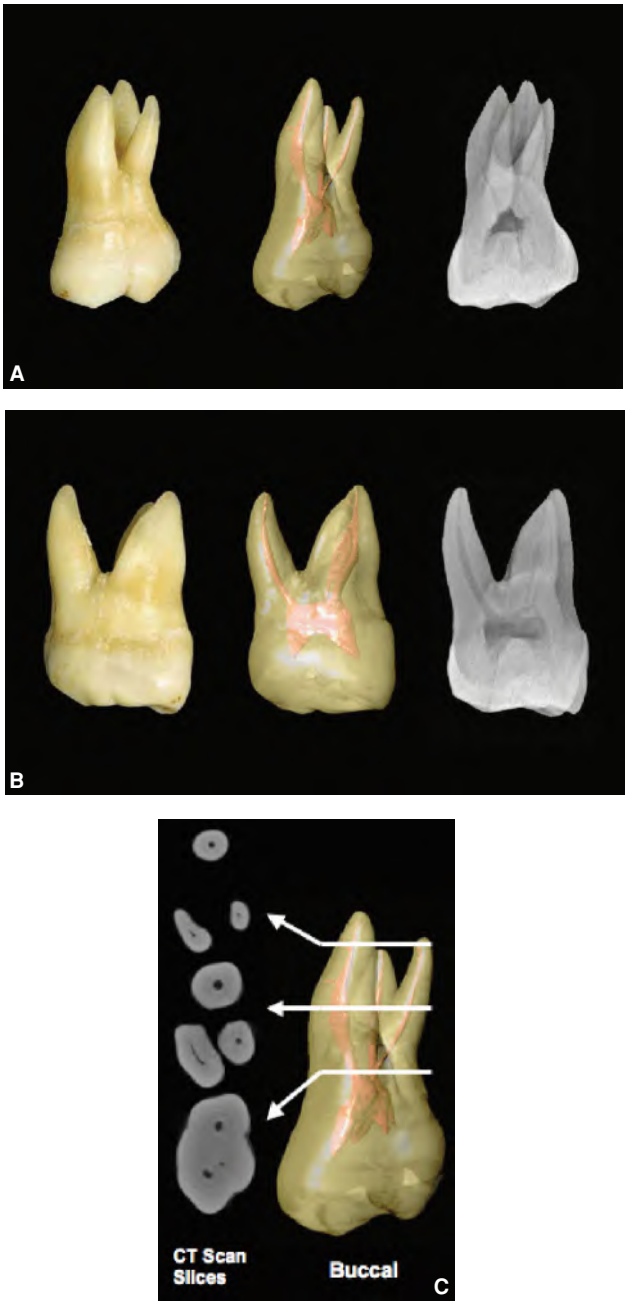


FIGURE 1-18 Maxillary left second molar. ***A.*** Buccal view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Maxillary Second Molar- Rotations & Slices.)

in crown and root size in the posterior arch consistent with the reduction in facial profile of modern *Homo sapiens*.[406]

The mesiobuccal root is broad buccolingually and has prominent depressions or flutings on its mesial and distal surfaces.[1,3,4,6,15–17] The internal canal morphology is variable and anatomical studies indicate that the mesiobuccal root has almost an equal incidence of one or two canals. The distobuccal root is generally rounded or ovoid in cross-section and usually contains a single canal. The palatal root is more broad mesiodistally than buccolingually and ovoidal in shape but normally contains only a single canal. Depressions on its buccal and palatal surfaces can be present but are usually shallow. Gher and Vernino[403] found prominent depressions on the distal aspect of the mesiobuccal roots. Lesser depressions could also be found on the furcal side of the distobuccal and palatal roots. The overall average length of the maxillary second molar is 19 mm with an average crown length of 7 mm and an average root length of 12 mm.[4]

Root Number and Form

The majority of maxillary second molars (85.8%) were three-rooted (Table 1-21). However, this is a lower incidence than that found in the maxillary first molar. The closer proximity of the roots results in a higher incidence of root fusion (26.4%) (Table 1-22) and C-shaped canals (4.9%) when compared to the maxillary first molar.[449]

Canal System

Mesiobuccal Canal System

In the most common three-rooted maxillary molar form, the shape of the root provides an indication of the shape of the internal canal morphology.[14] The broad buccolingual and narrow mesiodistal dimension of the mesiobuccal root of the maxillary second molar may have one or two canals (Table 1-23). A wide range of canal incidence in the mesiobuccal root exists. Kulild and Peters[132] reported that the norm would be a double-canal system and a low incidence of a single canal of 5.3%, while Hartwell and Bellizzi[460] found a single canal in 81.8% of their specimens. It should be noted that the former paper was a laboratory study while the latter was a clinical finding.

The single canal is usually described as being kidney or ribbon-shaped. Eskoz and Weine[74] suggested that age and continued deposition of secondary dentin in the isthmus can cause narrowing and possible occlusion in the mid portion resulting in two canals. There was a single apical foramen found in the mesiobuccal root over 64.7% of the time.

The Palatal and Distobuccal Canal Systems

The distobuccal and palatal roots exhibit a single canal over 99% of the time (Table 1-23). Both these roots are rounder in cross-section and a single-canal system is the expected normal finding. In a retrospective clinical treatment paper by Peikoff et al.[165] on the different forms of canal anatomy found in maxillary second molar teeth, six variants were described. In a random selection of 520 clinical treatments in a specialist office, Variants 1 to 6 reported and depicted second molar anatomy types from; single canal in a fused root to four canals in four roots (Figures 1-19 and 1-20).

Variations and Anomalies

Fifty case reports of anomalies associated with the maxillary second molar were reviewed (Table 1-24). The most common anomaly cited for this tooth is the four-rooted variation with two palatal roots. Other variations such as two canals within one large palatal root have been reported, but with a much lower frequency.[549,550]

A review article on the double palatal root by Christie et al.[446] studied a retrospective collection of 16 clinical cases from their practice and eight teeth from six published case reports[445,447,461,494,547,551] during a 25-year period. The highest occurrence of two palatal canals in double palatal roots (21/24 teeth) was found in the maxillary second molar tooth. The anomaly seemed to be grouped as three-root anatomy types; first, Type I, the two palatal roots being long and divergent; second, Type II, the two palatal roots being shorter, nearly parallel and comparable in shape and size to the two buccal roots (Figure 1-20); and third, Type III and IV were variations of root fusion that included a two-canal system, one entering the root from the palatal aspect. Subsequent case reports have tended to support this observation.[493,505,552–557]

TABLE 1-21 Root Number in the Maxillary Second Molar

Maxillary Second Molars Reference		No. of teeth in study	1 Root		2 Roots		3 Roots		4 Roots	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of teeth in root studies[5,18,142,175,182,184,189,547]	9	2845	4.4	124	9.0	255	85.8	2442	0.8	24

TABLE 1-22 Incidence of Fused Roots in the Maxillary Second Molar

Maxillary Second Molars References		No. of teeth in study	Total Incidence of Fused Roots	
			%	No. of Teeth
Total number of teeth in fused root studies[18,20–22,147,165,182,448,449]	9	2453	26.4	647

TABLE 1-23 Main and Apical Canal Number of the Maxillary Second Molar

Mesiobuccal Root Maxillary Second Molars (3-rooted teeth) References		No. of teeth in studies	1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[7,8,11,12,14,18,26,31,63,64,74,78,132,139,175,182,183,184,189,192,408,451,453,456,460,548]	27	5016	54.8%	2748	45.2%	2268				
Total number of canal apices studies	18	2254					64.7%	1458	35.3%	796
Distobuccal Root Maxillary Second Molars (3-rooted teeth) References										
Total number of canal studies[7,8,11,14,18,31,63,78,175,182,184,189,408,456,460,548]	16	3085	99.6%	3074	0.36%	11				
Total number of canal apices studies	10	1200					99.75%	1197	0.25%	3
Palatal Root Maxillary Second Molars (3-rooted teeth) References										
Total number of canal studies[7,8,11,14,18,31,63,78,175,182,184,189,408,456,460]	15	3035	99.8	3030	0.2	5				
Total number of canal apices studies	9	1150					99.8	1148	0.2	2

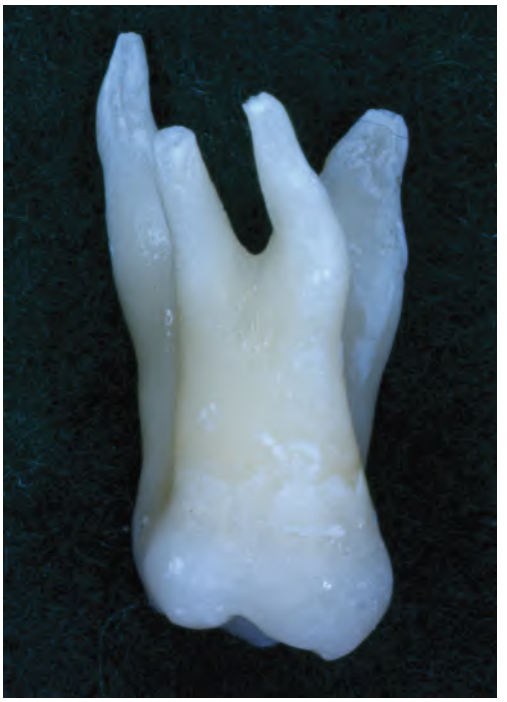


FIGURE 1-19 Maxillary right second molar (buccal view) with bifurcated double palatal root Type I. (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

In a study of 2636 posterior teeth in 875 Jordanian patients, a 4.4% incidence of taurodontism was found in the maxillary or mandibular molar teeth.[558] The maxillary second molar had the highest incidence of taurodontism. Of the 116 teeth identified in the study, 31% were permanent maxillary second molars. Treatment of hypertaurodont teeth was described in two case reports.[497,559] The ethnicity, if any, has not been established in this relatively rare root anomaly but it is no longer considered an exclusive primitive dental trait of *Homo Neanderthal* teeth.[538] (For more details, see Chapter 26, "Endodontic retreatment and management of mishaps.")

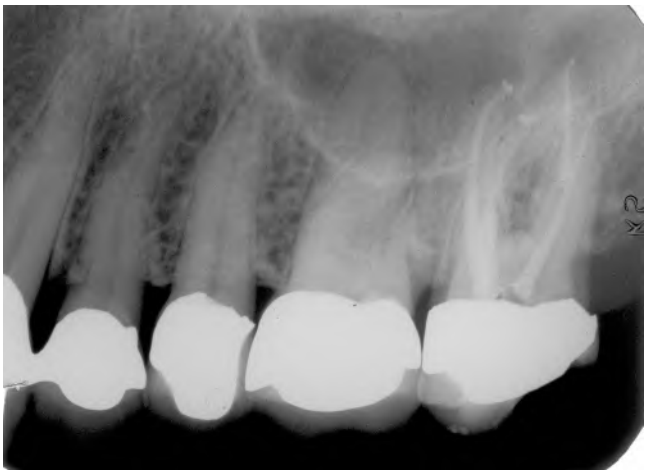


FIGURE 1-20 Maxillary left second molar (Type II) with four roots and four canals (1 MB root, 1 DB root, 1 MP, and 1 DP root, with post space. (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

MANDIBULAR CENTRAL INCISOR

External Root Morphology

The mandibular central incisor is single rooted (Figure 1-21). The external form of the root is broad labiolingually and narrow mesiodistally. Longitudinal depressions are present on both the mesial and distal surfaces of the root. A cross-section of the root is ovoid to hourglass in shape due

TABLE 1-24 Variations and Anomalies Associated with the Maxillary Second Molar

Maxillary Second Molars Anatomic Variation	No. of Cases	No. of References
4 roots and 4 canals (MB, DB and 2 palatal roots; Each root contained 1 canal)[446,493, 504,552,554–557,560–567]	55	17
Hypertaurodont[497, 559]	2	2
3 roots and 4 canals (MB, DB and 2 Palatal canals)[549,550]	2	2
3 roots and 5 canals (3 MB, DB and Palatal canals)[568,569]	2	2
4 roots and 4 canals (3 separate buccal roots and a Palatal root)[570, 571]	2	2
1 root and 1 canal[572]	1	1
C-shaped palatal canal with 2 apical foramina[573]	1	1
2 roots and 2 canals (2 MB and 2 DB canals)[574]	1	1
3 roots and 4 canals (2 MB, DB and Palatal canals)[575]	1	1
3 roots and 4 canals (MB 2 DB and Palatal canals)[576]	1	1
3 roots and 6 canals (2 MB, DB and 3 Palatal canals)[577]	1	1
3 roots and 6 canals (2 MB, 2 DB and 2 Palatal canals)[578]	1	1
4 roots and 6 canals (2 Palatal roots; 2 MB, 2 DB and 2 Palatal canals)[579]	1	1
5 roots and 5 canals (2 MB, DB, MP and DP)[580]	1	1
Gemination or fusion[581]	1	1
Gemination (5 canals – 2 MB, DB, Palatal and GB or geminated buccal canal)[582]	1	1
Dens invaginatus Type II[583]	1	1
Dens evaginatus[584]	1	1
Taurodontism[429]	1	1
Total number of case report references and cases	77	39

to the developmental depressions on each side.[1,3,4,6,13,15,17]. The overall average length of the mandibular central incisor is 21.5 mm with an average crown length of 9 mm and an average root length of 12.5 mm.[4] In general, the mandibular central incisor is just slightly smaller than the mandibular lateral incisor in all dimensions.

Root Number and Form

The anatomical studies reviewed here reported that 100% of the mandibular central incisors were single-rooted teeth (Table 1-25). Variations from this form have either not been reported or not found in a review of the literature. An early study of the anatomy of the mandibular anterior teeth by Rankine-Wilson and Henry[585] established that even with a single root, these teeth may contain a "bifurcated" or double-canal system.

Canal System

The shape of the canal system is either rounded or ribbon sha ped.[1,3,4,6,13,15,17,589] The majority of mandibular central incisors have a single canal. Table 1-26 shows that out of 7455 pooled incisor teeth, a single canal is found in 81.1%. Two separate canals were found in 18.8%, which is less frequent than that found in the mandibular lateral incisor. The incidence of three or more canals was quite rare (0.2%).

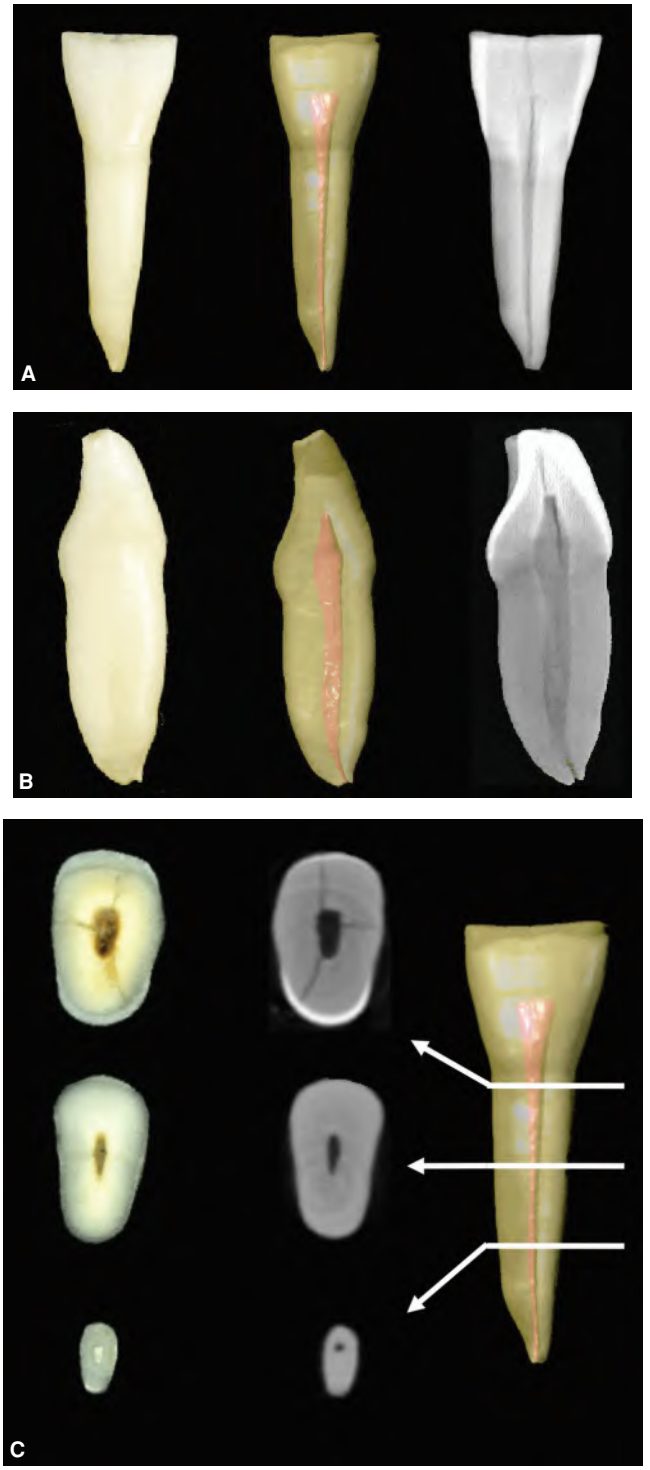


FIGURE 1-21 Mandibular right central incisor. ***A.*** Labial view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Mandibular Central Incisor- Rotations & Slices.)

TABLE 1-25 Root Number in the Mandibular Central Incisor

Mandibular Central Incisors References		No. of teeth in studies	1 root		2 roots	
			%	No. of Teeth	%	No. of Teeth
Total number of teeth in root studies[5,7,11,31,187,586,587,588]	8	3286	100	3286	0	0

TABLE 1-26 Main and Apical Canal Number of the Mandibular Central Incisor

Mandibular Central Incisors References		No. of Teeth in Studies	1 canal		2 canals		> 2 canals		1 apical foramen		2 or more apical foramina	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[5,7,11,31,168, 186,187, 194,586,587,588, 591–597]	18	7455	81.1	6043	18.8	1399	0.2	13				
Total number of canal apices studies	12	4412							96.5	4256	3.5	156

A single apical foramen was found in 96.5% of the teeth. Therefore, even when two separate canals have been found, the majority of these canals will join and exit near or at a single foramen (Figure 1-22). Even the single canal system in all lower anterior teeth is wide in a labiolingual direction and narrow mesiodistally, matching the external root outline.[1,3,4,6,13,15,17] Fluting of the mesial and distal surfaces produces a figure eight shape single canal in cross section. As secondary dentin forms with age, the canal system takes on the shape of a double canal with a connecting isthmus, and should be treated as if it was a double canal when the root canal treatment is performed.[589] (Figure 1-23). Clements and Gilboe[590] and others have even recommended an incisal edge access opening for a straight-line approach to the apical foramen, when incisal edge wear is present or when a restorative crown is planned.

Green[213] found that the average diameter of the major foramen in 200 pooled mandibular incisors was 0.3 mm while the accessory foramina were 0.2 mm or less in diameter. Approximately 12% of the pooled mandibular incisors exhibited accessory foramina. The average distance of the apical foramen from the anatomical root apex was 0.2 mm.

Variations and Anomalies

Few anomalies are reported for this tooth (Table 1-27). Of the 15 case reports reviewed, the most frequently reported anomaly was dens evaginatus (talon cusp).

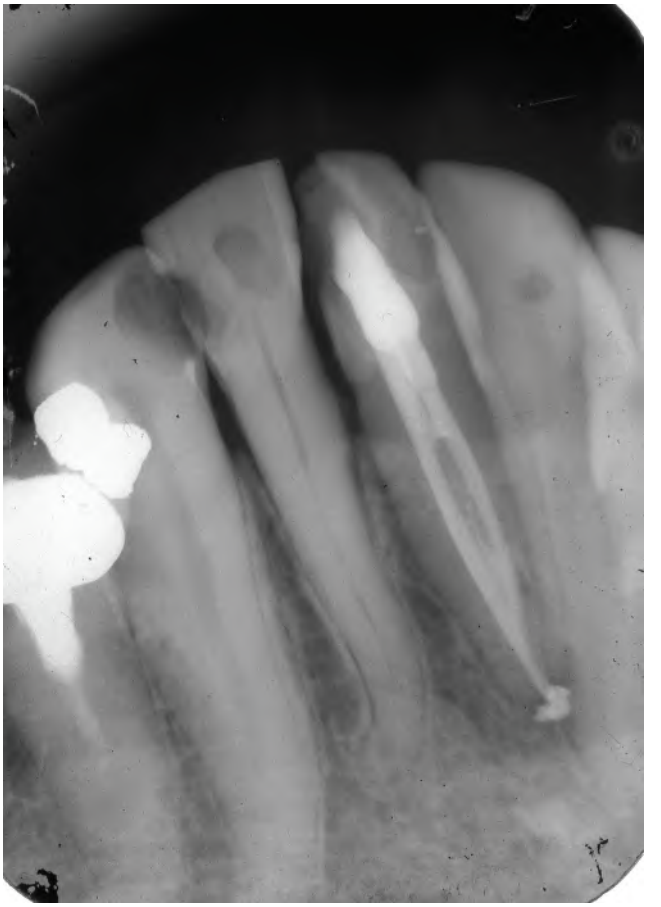


FIGURE 1-22 Mandibular left central with two canals and one apex. (Courtesy of Dr. Marshall D. Peikoff, Winnipeg, MB, Canada.)

MANDIBULAR LATERAL INCISOR

External Root Morphology

The mandibular lateral incisor is single rooted (Figure 1-24) and comparable in form and shape to the mandibular central incisor. The external form of the root is broad labiolingually and narrow mesiodistally. Longitudinal depressions are present on both the mesial and distal midroot surfaces.

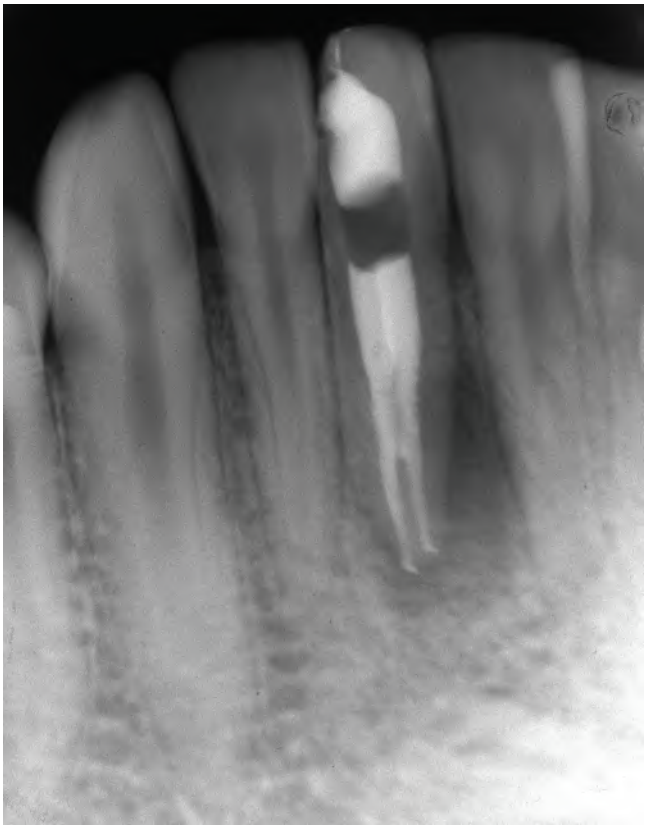


FIGURE 1-23 Mandibular left central incisor with two canals and two separate apical foramina. (Courtesy of Dr. Marshall D. Peikoff, Winnipeg, MB, Canada.)

TABLE 1-27 Variations and Anomalies Associated with the Mandibular Central Incisor

Mandibular Central Incisors Anatomic Variation	No. of Cases	No. of References
Dens evaginatus (talon cusp)[327, 598, 599, 600]	4	4
Dens invaginatus and talon cusp[601, 602]	2	2
Dens invaginatus Type III[603, 604]	2	2
2 canals (Vertucci Type V)[605]	1	1
3 canals with a single apex (Type XV)[606]	1	1
Fusion with a supernumerary tooth associated with a talon cusp[607]	1	1
Dens invaginatus Type I and talon cusp, short root and macrodont crown[608]	1	1
2 canals and 2 separate foramina[609]	1	1
Dens evaginatus (labial talon cusp)[400]	1	1
Dens invaginatus[235]	1	1
Total number of case report references and cases	15	15

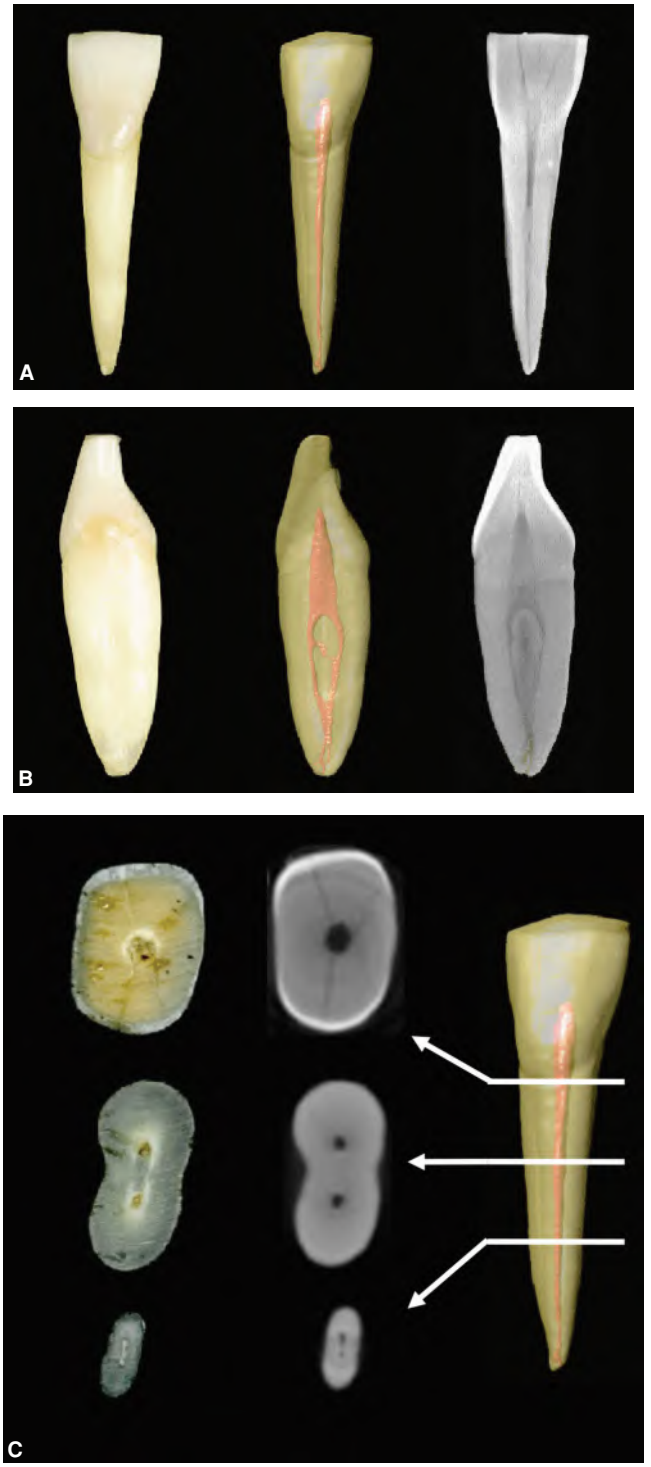


FIGURE 1-24 Mandibular left lateral incisor. ***A.*** Labial view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Mandibular Lateral Incisor- Rotations & Slices.)

A cross-section of the root is ovoid or hourglass in shape due to the developmental depressions on each side.[1,3,4,6,13,15,17] The overall length of the average mandibular lateral incisor is 23.5 mm with an average crown length of 9.5 mm and an average root length of 14 mm.[4]

In addition to being slightly larger in all dimensions, the major difference in tooth anatomy, compared with the mandibular central incisor, is at the incisal edge coronal anatomy. Slight angulation to the distolingual and mesiolabial of the mandibular incisor's incisal edge should be compensated for when preparing an endodontic access opening

TABLE 1-28 Root Number in the Mandibular Lateral Incisor

Mandibular Lateral Incisors References	No. of teeth in studies	1 root	
		%	No. of Teeth
Total number of teeth in root studies[5,7,11,31,187,586–588]	3266	100	3258

TABLE 1-29 Main and Apical Canal Number of the Mandibular Lateral Incisor

Mandibular Lateral Incisors References		No. of teeth in studies	1 canal		2 canals		> 2 canals		1 apical foramen		2 apical foramina	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[5,7,11,31,168,186,187,194,586–588,591–597]	18	7200	75.4	5427	24.6	1770	0.04	3				
Total number of canal apices studies	12	4374							95.8	4190	4.2	184

and searching for the more frequently encountered, broader labiolingual canal system.

Root Number and Form

All eight studies reviewing a pooled average of 3266 teeth, reported that 100% of the mandibular lateral incisors were single-rooted teeth (Table 1-28). Variations in root number from this form have either not been reported or not found in a review of the literature, except that reported by Slowey.[445]

Canal System

The shape of the canal system is comparable to the mandibular central incisor and is either rounded or ribbon shaped.[1,3,4,6,13,15,17,589] The majority of mandibular lateral incisors have a single canal, which is less than the percentage found in the average mandibular central incisor (81.1%) (Table 1-29). Two canals were found in 24.6% of the specimens (Figure 1-25). The incidence of more than two canals was quite rare (0.04%). A single apical foramen was found in 95.8% of teeth. Therefore, similar to the mandibular central incisors, even when two separate canals have been found, the majority of the canals will join and exit through a single foramen.

Variations and Anomalies

Very few anomalies have been reported for this tooth (Table 1-30). Dens evaginatus (talon cusp), dens invaginatus, and fusion were the main anomalies. Slowey[445] described a radiograph of a rare mandibular lateral incisor with two distinct root tips.

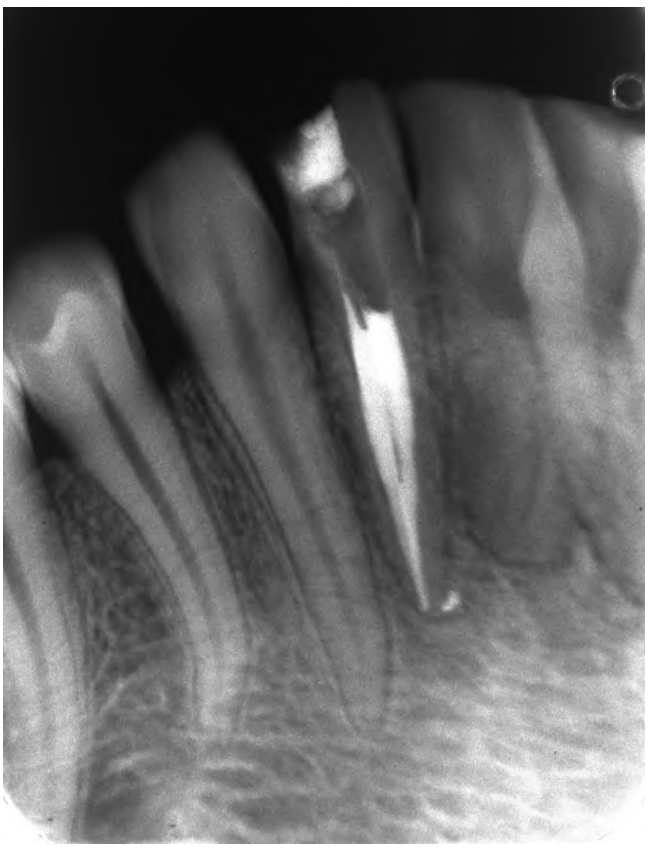


FIGURE 1-25 Mandibular right lateral incisor with two canals and one apical foramen. (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

MANDIBULAR CANINE

External Root Morphology

The root of the mandibular canine is wider labiolingually and narrower mesiodistally in cross-section, which is larger and longer than, but similar to the shape, the other mandibular anterior teeth (Figure 1-26). The projecting cusp tip of the mandibular canine is usually lingual to the long axis of the tooth length, while the cusp tip of a maxillary canine is labial to its long axis. Both canine roots are usually straight but may occasionally have fine and curved tips.

Developmental depressions are normally present on both the mesial and distal surfaces of the mid-root. The depressions can be relatively deep. Normally a single-rooted tooth,

TABLE 1-30 Variations and Anomalies Associated with the Mandibular Lateral Incisor

Anatomic Variation	No. of Cases	No. of References
Dens invaginatus Type 3[610, 611]	2	2
Dens invaginatus Type 2[612]	1	1
Dens evaginatus (talon cusp)[326]	1	1
Dens invaginatus[613]	1	1
Fusion with a supernumerary tooth[614]	1	1
Fusion with a supernumerary tooth[615]	1	1
Fusion between a mandibular lateral incisor and canine[616]	1	1
2 roots[445]	1	1
Total number of case report references and cases	9	9

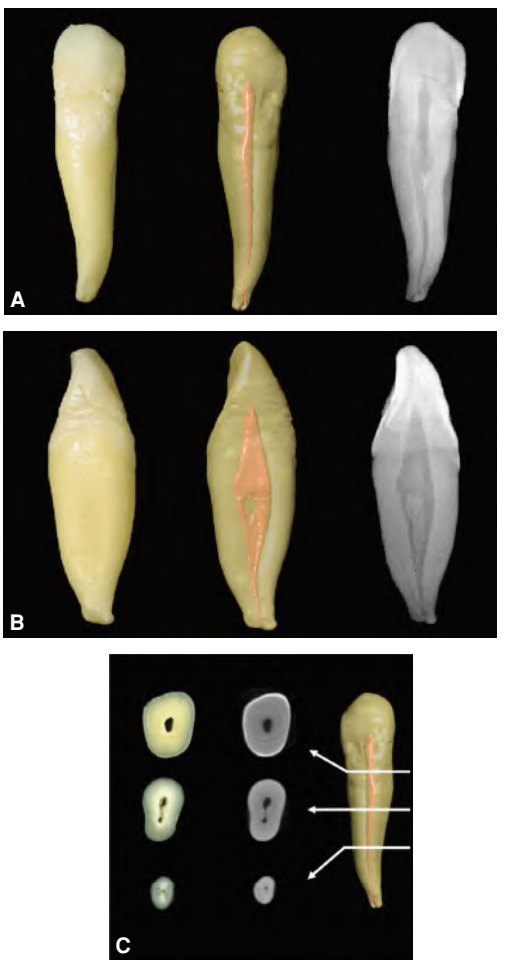


FIGURE 1-26 Mandibular left canine. ***A.*** Labial view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Mandibular Canine-Rotations and Slices.)

one variation of root morphology is a bifurcated root that may resemble a premolar root.[1,3,4,6,13,15,17] At other times, when this bifurcation occurs, the furcation dividing the labial and lingual roots can be at any level on the root trunk and usually results in a smaller lingual rootlet at the apical third region. In rare occasions, even two separated roots can be found (Figure 1-27).

The overall average length of the mandibular canine is 27 mm with an average crown length of 11 mm and an average root length of 16 mm[4] that is comparable to the overall length of the maxillary canine.

Root Number and Form

The most common form of the mandibular canine is one with a single root (95.4%) (Table 1-31). The studies cited found an incidence of two roots ranging from 1.3%[586] to 6.2%.[5] A recent paper by Lee and Scott[617] suggested that this trait is largely a European ethnicity trait (5.7%–9.2%), found most frequently in Basque, Spain, and is rarely found in Asian or sub-Sahara African populations. An earlier article by Scott and Alexandersen[618] estimated the incidence of two-rooted mandibular canines in European samples to be ~5% to 10% and very rare in Asian and African populations.

Canal System

The mandibular canine usually presents with a single root canal system[5,7,14,22,31,83,85,168,409,594,596] (Table 1-32). The incidence of a single canal is 90.5%. However, even in a single canal system, the root trunk is broad in a labiolingual direction and therefore very ovoid or figure-eight shaped in cross-section.[620] In the single-canal system, 97.9% have a single apical foramen.[7,11,14,31,85,131,141,586] Therefore, when two canals are present (9.5%) in a single-rooted mandibular canine, the most common configuration is the joining of the two canals before

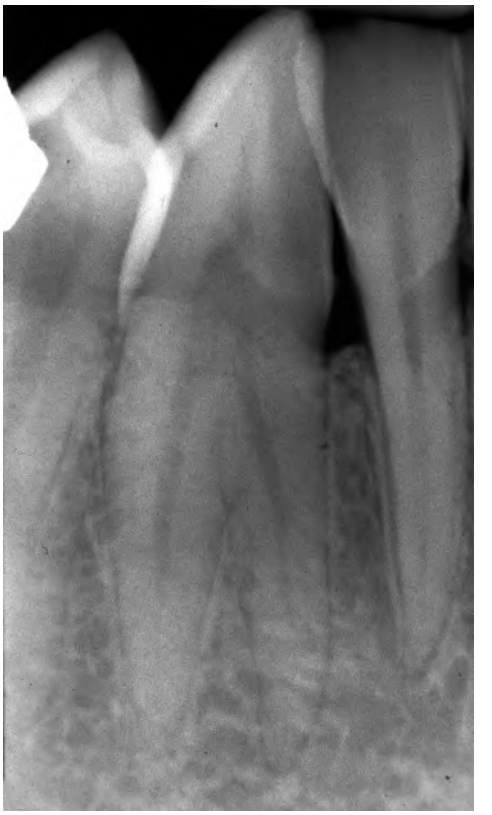


FIGURE 1-27 Rotated two-rooted mandibular right canine with radicular third root bifurcation. (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

TABLE 1-31 Root Number in the Mandibular Canine

References	No. of teeth in study	1 root		2 roots	
		%	No. of Teeth	%	No. of Teeth
Total number of teeth in root studies[5,85,401,586,619]	5	8356	95.4	7975	4.6	381

TABLE 1-32 Main and Apical Canal Number of the Mandibular Canine

Mandibular Canines References		No. of teeth in study	1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[5, 7, 11, 14, 31, 85, 131, 141, 168, 194, 409, 586, 594, 596]	14	6081	90.5	5502	9.5	579				
Total number of canal apices studies	8	3058					97.9	2995	2.1	63

TABLE 1-33 Variations and Anomalies Associated with the Mandibular Canine

Anatomic Variation	No. of Cases	No. of References
2 roots and 2 canals[622–627]	8	6
2 roots and 3 canals[628–630]	3	3
1 root and 2 canals[631, 632]	3	2
1 root and 3 canals[633]	1	1
Fusion between a mandibular lateral incisor and canine[616]	1	1
Total number of case report references and cases	16	13

exiting at the apex [Vertucci Type II (2-1) or Vertucci Type III (1-2-1)].

Green[213] found that the average diameter of the major foramen was 0.3 mm, while the accessory foramina were 0.2 mm or less. The average distance of the major apical foramen from the anatomical root apex was found to be 0.35 mm. Approximately 10% of the mandibular canines exhibited accessory foramina.

VARIATIONS AND ANOMALIES

The most frequent variation found in the mandibular canine is the presence of two roots and two canals (Table 1-33).[13] A report by Alexandersen[401] showed a high incidence (5.6%) of two-rooted mandibular canine teeth in an Iron-Age Danish population.

Although, the data from anatomical studies varies greatly with respect to the incidence of two roots, the mandibular canine has the highest incidence of all of the anterior teeth at 4.5%. The human primary canine teeth are more likely to exhibit two separate (bifurcated) roots.[621] Other variations reported in the literature include two canals and two roots, two canals with a single apical foramen, three canals, dens invaginatus, and gemination.

MANDIBULAR FIRST PREMOLAR

External Root Morphology

The mandibular first premolar is typically a single-rooted tooth that is wider buccolingually and narrower mesiodistally, although two-rooted varieties do occur fairly frequently (Figure 1-28).[1,3,4,6,15–17] Developmental depressions or grooves are frequently found on both the mesial and distal surfaces of the root resulting in an ovoid or slight hourglass-shaped root that tapers to the lingual. The depression on the distal root surface has been described as being deeper than the mesial root depression.[634]

The overall average length of the mandibular first premolar is 22.5 mm with an average crown length of 8.5 mm and an average root length of 14 mm.[4]

Root Number and Form

The mandibular first premolar is normally a single-rooted tooth (Table 1-34); however, studies revealed an incidence of ~2.7% of bifurcated teeth.[5,7,11,14,408,635–637] Three-rooted mandibular first premolars are extremely rare with an incidence of 0.2%.

Trope et al.[638] found significant ethnic variations in root anatomy when comparing African American and Caucasian patients. Their study found an incidence of two root canals in 5.5% in Caucasian and 16.2% in the African American group of patients. Three-rooted mandibular first premolars are rare but are occasionally found in case reports (Figure 1-29).[435,639–642]

Scott and Turner[406] described the accessory root as "Tome's root." Their anthropological review of ethnic differences indicates that aboriginal Australians and sub-Sahara African populations have the highest incidence (>25%) of accessory roots. The lowest incidence (0%–10%) of Tome's

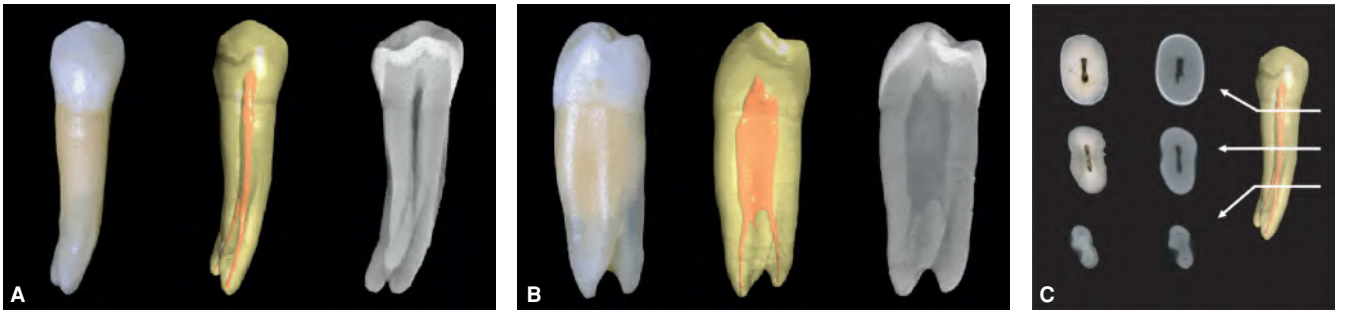


FIGURE 1-28 Mandibular right first premolar with two roots and two canals. Note, the tooth pictured is a variation from normal (incidence circa 2.7%). ***A.*** Buccal view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Mandibular First Premolar- Rotations and Slices.)

TABLE 1-34 Root Number in the Mandibular First Premolar

References		No. of teeth in study	1 Root		2 Roots		3 Roots		4 Roots	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of teeth in root studies[5,7,11,100,108,114,120,121,129,177,178,408,635,636,637]	15	6348	97.1	6163	2.7	173	0.2	10	0.0	2

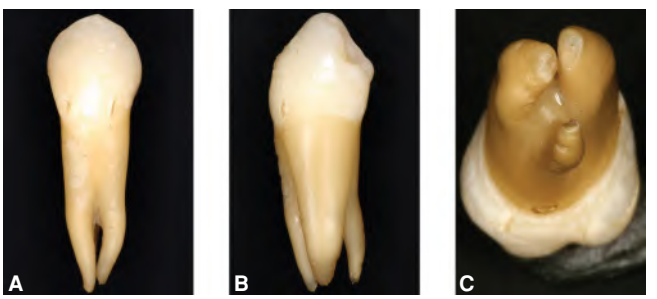


FIGURE 1-29 Rare three-rooted mandibular first premolar. ***A.*** Buccal view. ***B.*** Mesial view. ***C.*** Apical view. (Reprinted with permission from RightsLink December 20, 2014. Cleghorn B, Christie W, et al. The root and root canal morphology of the human mandibular first premolar: a literature review. *J Endod*. 2007a;33:509-516, Figures 2a, 2b and 2c.)

root occurred in American Arctic, New Guinea, Jomon, and Western Eurasian populations.

The complexity of the root and root canal system is related to the presence and severity of radicular grooves.[151,154,155] Tome's root ranges from a shallow concavity to a deep groove, to complete furcation(s) of the root(s). The deeper the concavity, the more likely the presence of a C-shaped canal system within.[151,154,155] Chen et al. studied the relationship of radicular grooves and root canal morphology in the mandibular first premolars using Micro-CT and found that 40.9% of the teeth in their study sample had radicular grooves. These grooves were primarily located on the mesial root surface (69.5%) and were more frequently associated with those teeth that had more complex root canal morphology.[156]

Canal System

Slowey[445] suggested that the mandibular premolars may present with the greatest difficulty of all teeth to treat endodontically. A University of Washington study assessed the failure rate of non-surgical root canal treatment in all teeth. It was highest for the mandibular first premolar at 11.45%.[643] Possible reasons for a high failure rate are the numerous variations in root canal morphology and difficult access, cleaning and sealing of a second canal. There is usually straight-line access to the buccal canal while the lingual canal branches below the chamber at a sharp angle, potentially resulting in a missed canal. Kartal and Yanikoglu,[644] using pooled data that included first and second premolars, reported a 27.8% incidence of mandibular premolars with more than one canal. Serman and Hasselgren[645] found that 15.7% of patients had at least one mandibular first premolar with either a divided canal or root. The second premolars had a lower but still notable incidence of 7% of the teeth with a double-canal system in this study.

The data from 29 anatomical studies of the canal system that included only mandibular first premolars but no ethnic attribute resulted in an average of a single canal in 77.3% of cases (Table 1-35). Two or more canals were found in 22.7% of the teeth. A single canal at the apex is found 76.7% of the time. Almost 25% of teeth had more than one foramen.

Trope et al.[638] found significant ethnic differences between African American and Caucasian patients. The African American group had an incidence of two or more canals in 32.8%, while the incidence in the Caucasian group was 13.7%. A study of 1000 full mouth radiographic surveys also found ethnic differences between African American and

TABLE 1-35 Main and Apical Canal Number of the Mandibular First Premolar

Mandibular First Premolars References		No. of teeth in study	1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[5,7,11,22,31,56,68,75,100,102,108,114,117,120,121,124,126,129,149,156,177,178,185,408,409,410,596,635,646]	29	8538	77.3	6601	22.7	1937				
Total number of canal apices studies	20	4651					76.7	3568	23.3	1083

Caucasian patients.[647] Although the number of patients in each ethnic group was not identified, the study reported that 16% of the Caucasian patients had bifurcated canals compared to 21.6% of the African American patients. Sert and Bayirli[11] found an incidence of two or more canals in 35% of men and 44% of women of Turkish descent, further reinforcing the importance of ethnic differences as well as possible gender differences.

Variations and Anomalies

Twenty-five case reports of anomalies or variations of the mandibular first premolar were identified in a review of the literature (Table 1-36). The most common variation associated with the mandibular first premolar was the three-rooted variation with three canals (Figure 1-29). However, only five case reports in total were identified.[435,639–642] Other reported developmental anomalies included "C"-shaped root canals in premolars,[151,154–156] three canals in a single root,[648–650] and three canals and two roots.[649]. Discussion of dens evaginatus, particularly in premolar teeth of patients of Asian ethnicity is also found under mandibular second premolar tooth anomalies.

A study of 45,X chromosome women (and 45,X/46,XX chromosome) found more than one canal in one or more of the mandibular premolars in almost half of the 87 patients studied who had the genetic syndrome. Separate canals were found in 23% of the mandibular first premolars and 25% of the mandibular second premolars.[666] The study concluded that X-chromosomes might have a gene or genes with a regulatory function for root development.

TABLE 1-36 Variations and Anomalies Associated with the Mandibular First Premolar

Anatomic Variation	No. of Cases	No. of References
3 roots and 3 canals[435, 39–642]	5	5
Dens evaginatus*[425, 651–653]	4	4
1 roots and 2 canals[650, 654, 655]	3	3
1 root and 3 canals[648–650]	3	3
2 roots and 2 canals[656–658]	3	3
3 canals[659]	1	1
1 root and 4 canals[660]	1	1
2 roots and 3 canals[661]	1	1
4 roots and 4 canals[662]	1	1
4 canals[663]	1	1
Gemination[664]	1	1
Multiple roots[665]	1	1
Total number of case report references and cases	25	25

MANDIBULAR SECOND PREMOLAR

External Root Morphology

The mandibular second premolar is normally a single-rooted tooth (Figure 1-30) like the mandibular first premolar.[1,3,4,6,15–17] The root is described as flat or convex on its mesial surface while the distal surface often (73%) has a longitudinal developmental depression.[634] A cross-section of the root is usually ovoid in shape, generally tapering to the lingual.[16]

The overall average length of the mandibular second premolar is 22.5 mm with an average crown length of 8 mm and an average root length of 14.5 mm.[4]

Root Number and Form

The mandibular second premolar is normally a single-rooted tooth. Trope et al.[638] compared root and canal morphology in Caucasian and African American patients and found 1.5% incidence of two roots in mandibular second premolar teeth in Caucasian patients and 4.8% in African American patients. These differences were lower and not statistically significant in contrast to the differences (as high as one in six) found between these two groups in the mandibular first premolars.

Studies found multi-rooted mandibular second premolars to be quite rare (Table 1-37). Two-rooted varieties comprised 0.5% of the teeth. Three-rooted forms were rarely found and comprised 0.1% of teeth.

Canal System

Studies found a single canal in 91.3% of mandibular second premolars (Table 1-38). When a second canal system is located, it is usually fine and branches toward the lingual surface in the middle or apical third of the main canal. The incidence of two or more canals was 8.7%. The characteristic appearance of such a tooth on a periapical radiograph is of a clearly visible coronal canal that seems to abruptly fade-out in the apical region beyond the branching canal system.

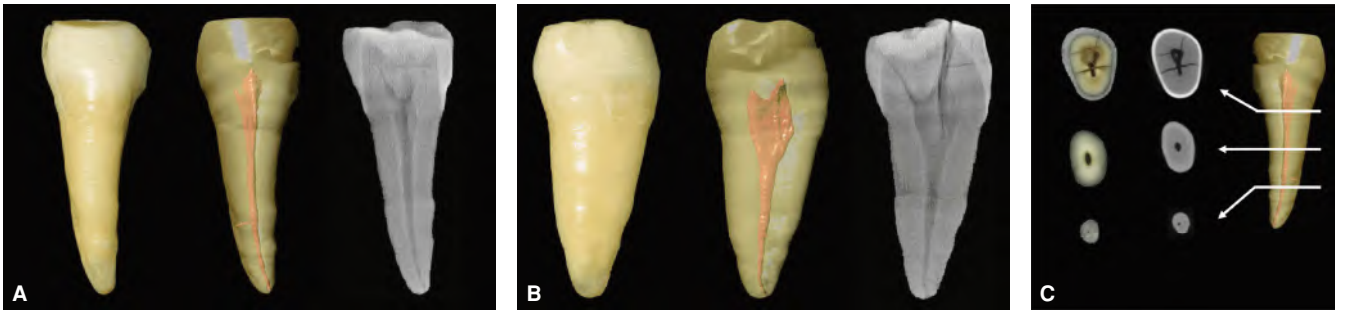


FIGURE 1-30 Mandibular left second premolar. ***A.*** Buccal view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Mandibular Second Premolar- Rotations and Slices.)

TABLE 1-37 Root Number in the Mandibular Second Premolar

References		No. of teeth in study	1 Root		2 Roots		3 Roots	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of teeth in root studies[5,7,11,99,100,102,108,114,130,177,408,635,646,667]	14	5055	99.4	5026	0.5	23	0.1	6

TABLE 1-38 Main and Apical Canal Number of the Mandibular Second Premolar

References		No. of teeth in study	1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[5,7,11,31,75,90,99,100,102,108,114,130,177,185,408,409,596,635,646]	19	5521	91.3	5043	8.7	478				
Total number of canal apices studies	14	3123					90.6	2828	9.4	295

There was a single apical foramen 90.6% of the time. Serman and Hasselgren[645] found that second mandibular premolars had a 7% incidence of having a divided canal or root. Trope et al.[638] found ethnic differences between African American and Caucasian patients. African American group had a higher incidence (7.8%) of mandibular second premolars with two or more canals, while the incidence in the Caucasian group was lower (2.8%). These differences were not numerically significant in contrast to the results found with the mandibular first premolar canal systems.

Sert and Bayirli[11] study of Turkish patients found an incidence of two or more canals in 43% of men and 15% of the women. The gender differences in this population were significant and this ethnic group, as a whole, demonstrated a higher incidence of multiple canals than the averages of the remaining anatomical studies reported.

Variations and Anomalies

The most common anomaly reported was the variation with three canals in one or more roots (Table 1-39). The next most common anomaly was two canals in one or two roots

TABLE 1-39 Variations and Anomalies Associated with the Mandibular Second Premolar

Anatomic Variation	No. of Cases	No. of References
3 canals[70,648,678–685]	12	10
2 roots and 2 canals[650,658,674,686–691]	10	9
Dens evaginatus[672,692–694,]	6	4
3 roots and 3 canals[435,641,695–697]	5	5
2 roots and 3 canals[642,698,699]	3	3
2 roots and 4 canals[675,700,701]	3	3
4 roots and 4 canals[676,702]	2	2
C-shaped canal[640,703]	2	2
1 root and 2 canals[704]	1	1
1 root and 3 canals[705]	1	1
1 root and 4 canals[706]	1	1
1 root and 5 canals[677]	1	1
3 roots and 4 canals[691]	1	1
2 canals[707]	1	1
4 canals[708]	1	1
Hypertaurodont[709]	1	1
Multiple roots[665]	1	1
Total number of case report references and cases	52	47

(Figure 1-31), although this could be considered a variant from the one canal in one root.

Another anomaly includes mandibular second premolars with dens evaginatus (odontome) (Figure 1-32). Dens evaginatus is a fairly common occurrence in Asian populations and is usually found in the mandibular premolars, although not exclusively.[159,668,669] Merrill[670] reported a high incidence (4.5%) of this anomaly in Alaskan Eskimos (Inuit) and American Indians, an observation serving to illustrate Alaskan natives' ties to their more recent Asian heritage.

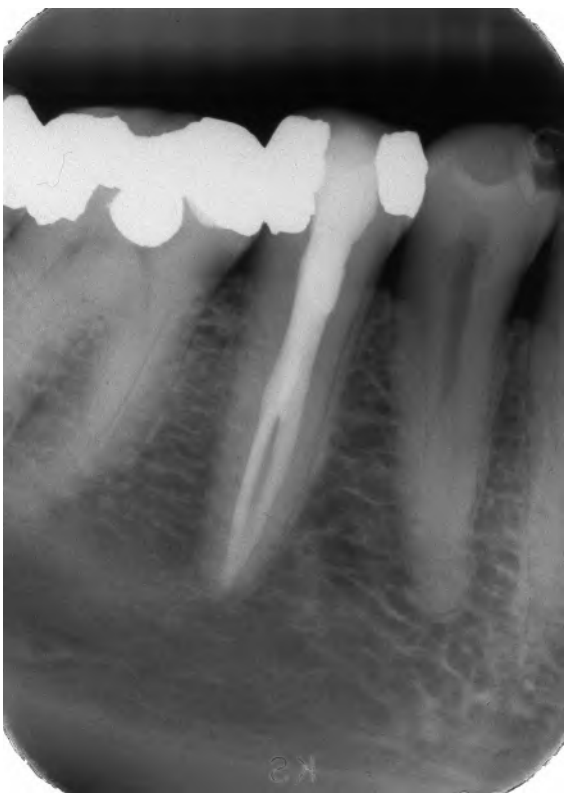


FIGURE 1-31 Mandibular right second premolar with two canals and one apical foramen. Complex canal anatomy can also be seen in the first premolar. (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

Very often, when these teeth, with an occlusal enamel projection, erupt into occlusion, the pulp is exposed by attrition of the tubercle and it becomes nonvital. Root development at this age usually presents with a large or blunderbuss apex. Various prophylactic treatments have been suggested over the decades.[671–673] However, endodontic therapy and/or apexogenesis is the most frequent outcome of this coronal anomaly.[674]

C-shaped canal is a rare occurrence.[675,676] The hypotaurodont-like canal system may also appear in the mandibular second premolar tooth.[677]

MANDIBULAR FIRST MOLAR

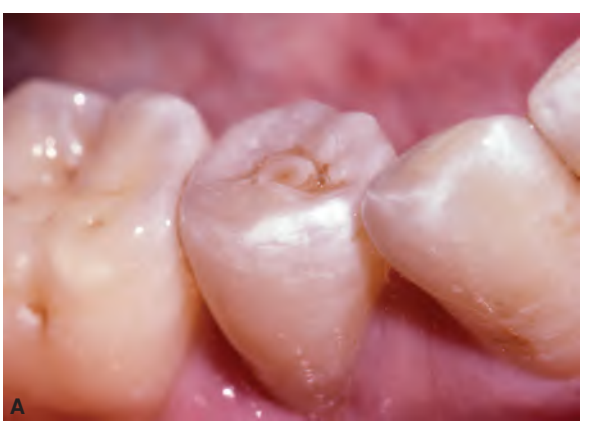
External Root Morphology

The mandibular first molar is typically a two-rooted, three-canal tooth (Figure 1-33).[1,3,4,6,15–17] The mesial and distal roots are normally widely separated, with a furcation level at ~3 mm buccally and lingually and 4 mm apical to the cementoenamel junction, respectively.[4] Both roots are broader buccolingually than mesiodistally, with the mesial root wider buccolingually than the distal root. The mesial root has concavities on both its mesial and distal surfaces and is angled slightly mesially before curving to the distal approximately mid-root. The mesial root is slightly rotated and tapers distally from buccal to lingual. The distal root is generally more ovoid in its cross-sectional shape, although a mesial concavity in the mid-root may make it appear "kidney-bean" shaped in cross-section.[4]

The overall average length of the mandibular first molar is 21.5 mm with an average crown length of 7.5 mm and an average root length of 14 mm.[4] Root length may vary considerably in some individuals, with overall tooth length in excess of 25 mm in both first- and second-mandibular molar teeth.

Root Number and Form

The mandibular first molar is typically a two-rooted form with a mesial and a distal root (Table 1-40).[5,55,84,91,92,134,137,160,161,408,710–717] This anatomic form has an overall incidence of 85.6%. However, there are significant root anatomy differences when comparing Asian[55,91,92,137,160,710-712,714,717] to non-Asian



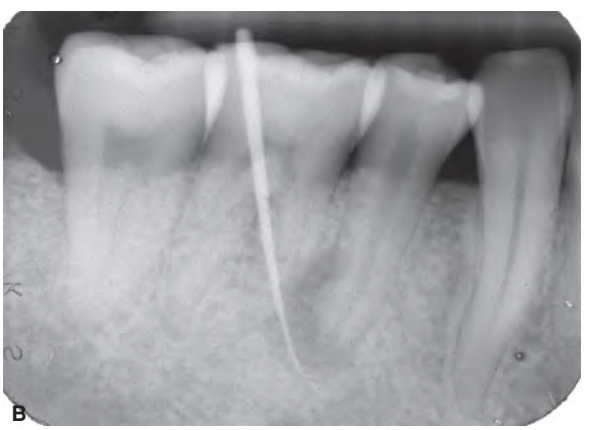


FIGURE 1-32 Non-vital mandibular right second premolar exhibiting dens evaginatus. ***A***. Clinical view. Note, worn tubercle in central fossa. ***B***. Radiographic view. Gutta-percha point in sinus tract pointing to apex of the affected tooth. (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

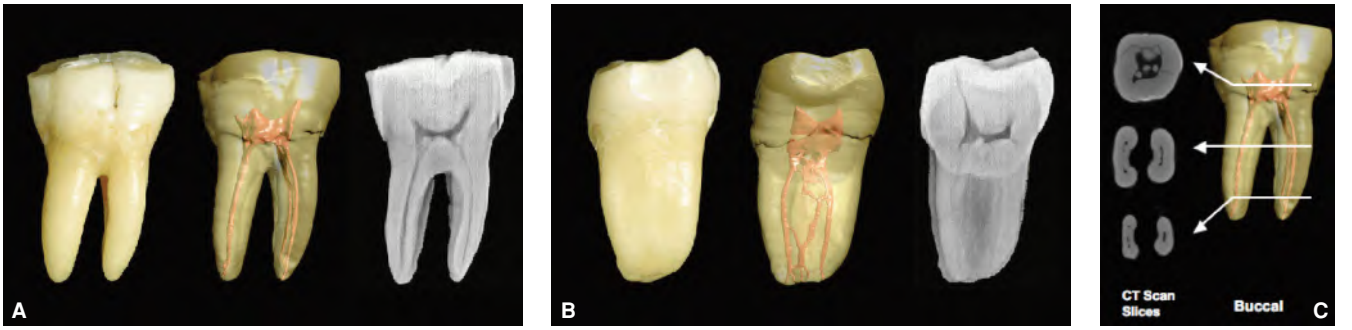


FIGURE 1-33 Mandibular right first molar. ***A.*** Buccal view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Mandibular First Molar-Rotations and Slices.)

TABLE 1-40 Root Number in the Mandibular First Molar

References		No. of teeth in study	1-Fused Roots						2 Roots		3 Roots		4 Roots	
			Total Incidence of Fused Roots		1 Root (conical)		1 Root (C-shaped)							
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of root studies (all studies combined)	36	17780	0.3	49	0.1	24	0.1	13	85.6	15218	14.1	2510	0.0	3
Total number of root studies (Non-Asian populations)[5,71,72,84,97,110,111,116,119,134,144,161,163,171,408,710,713,715–717]	23	7615	0.1	11	0.1	4	0.0	1	96.1	7321	3.7	283	0.0	0
Total number of root studies (Asian and NA Native populations)[35,47,55,91,92,137,160,176,190,191,710,711,712,714,717]	13	10165	0.4	38	0.2	20	0.1	12	77.7	7897	21.9	2227	0.0	3

populations.[5,84,134,161,408,710,713,715–717] Non-Asian populations have a higher incidence of two roots (96.1%) while the Asian populations have a lesser incidence (77.7%). Therefore, three roots may occur in ~20% to 25% of Asian populations. Anatomical studies found that the three-rooted variety, with a bifurcated mesial or distal root or an additional supplementary root, had an overall incidence of 14.1%. Single-rooted forms, fused and four-rooted forms, were extremely rare and occurred in <1% of teeth.

There is a higher incidence of three roots (DLi supplementary) occurring in Asian populations, that includes North American Aboriginal peoples (Mongoloid groups). Thirteen of the 36 anatomical studies that specifically included Asian-ethnicity populations, showed a variation in incidence even within these population groups. The incidence of three-rooted mandibular first molars ranged from 10.1% in the Burmese[92] to 29.3% in the Chinese.[176] Non-Asian groups ranged from 0%[5,716] to 13.3%.[72] *Radix entomolaris* (as well as the single-rooted maxillary first premolar) may be used as one of the markers in following Asian origins in both ancient and modern individuals or groups.[405,710,716,718] The incidence of single, fused, or C-shaped roots increases in the mandibular molar teeth from first to third molars.[1,3,4,6,15–17]

Canal System

The mandibular first molar typically has two mesial and one distal canals (Table 1-41). The two-rooted forms have two canals in the mesial root 95.6% of the time. The mesial root canals may have a common exit foramen 1:3 (35.6%) or can exit separately as two or more apical foramina 2:3 (64.4%).

Slowey[461] indicated that the mesiobuccal canal usually has a distinct buccal curvature at the floor of the chamber while the mesiolingual canal is straighter in the long axis to the root. The distal root usually has a single, broad, oval or kidney

bean-shaped canal, but a two-canal system can occur in nearly one third of the distal roots, resulting in a four-canal system.

The three-rooted forms had two canals in the mesial root 97.8% of the time in an average of the three anatomical studies reported (Table 1-42). However, there was a single apical foramen 29.9% of the time. The distobuccal root had one canal 98.6% of the time and the distolingual or third root had a single canal 100% of the time.

TABLE 1-41 Main and Apical Canal Number of the 2-rooted Mandibular First Molar

Mesial Root References		No. of teeth in study	1 canal		2 canals		3 canals		Other canal morphology		1 canal at apex		2 or more canals at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[7,11,14,31,81,84,91,92,110,128,134,144,161,166,190,191,408,452,460,714]	20	5824	3.2	188	95.6	5567	1.1	66	0.1	3				
Total number of canal apices studies	15	3483									35.6	1239	64.4	2244

Distal Root References		No. of teeth in study	1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[7,11,14,31,81,84,91,92,110,128,134,144,153,161,166,171,190,191,408,460,714]	21	5965	69.3	4131	30.7	1834				
Total number of canal apices studies	16	3657					83.1	3039	16.9	618

TABLE 1-42 Main and Apical Canal Number of the 3-rooted Mandibular First Molar

Mesial Root References	Number of teeth in study		1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[91,92,110,190,714]	5	724	2.2	16	97.8	708				
Total number of canal apices studies	4	545					29.9	163	70.1	382

Distobuccal Root References	No. of teeth in study		1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[91,92,110,190,714]	5	724	98.6	714	1.4	10				
Total number of canal apices studies	4	545					99.1	540	0.9	5

Distolingual Root References	Number of teeth in study		1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[91,92,110,116,190,714]	6	732	100	732	0	0				
Total number of canal apices studies	5	553					100	545	0	0

Variations and Anomalies

Eighty-six case reports are included in Table 1-43. The most frequent reported anomalies relate to additional canals in one or more of the roots. However, the most frequently reported variation in root form is *radix entomolaris* (an anthropologic term for third root distolingually positioned). The additional reports of extra canals, supernumerary roots and canals, and taurodontism were not included in Table 6-43 (see Mandibular second molar and Chapter 26 for additional reports of taurodontism).

The mid-mesial canal is a concept that was described by Weine.[719] It is a term that is gaining acceptance with numerous case reports from the more frequent use of CBCT examination and increased use of microscope and guide-path probing techniques in endodontic treatments.[196,720–742] Table 1-41 shows that it was recorded in 1.1% of the mesial roots of the 5824 teeth in all studies.

TABLE 1-43 Variations and Anomalies Associated with the Mandibular First Molar

Anatomic Variation	No. of Cases	No. of References
3 roots (Radix entomolaris)[41,50,720, 743–752]	23	13
2 roots and 5 canals (3M and 2D)[196, 721–732]	19	13
2 roots and 4 canals (3M and D)[719, 733–739]	10	8
2 root and 5 canals (2M and 3D)[166, 753–756]	8	5
C-shaped canal[499, 757–759]	4	4
2 roots and 6 canals (3M and 3D)[726, 760, 761]	4	3
Taurodontism[429, 495, 497]	3	3
3 M canals[740]	2	1
2 roots and 6 canals (4M and 2D)[741, 742]	2	2
2 roots and 4 canals[762]	2	1
2 roots and 2 canals[763]	1	1
2 roots and 6 canals (2M and 4D)[764]	1	1
3 roots (2 mesial and 1 distal)[688]	1	1
4 roots and 4 canals[765]	1	1
4 roots (2 mesial and 2 distal)[688]	1	1
4 roots (M and 3D) and 5 canals (2M and 3D)[766]	1	1
4 roots and 6 canals (MB, DB, 3D and DLi)[767]	1	1
4 roots (Radix paramolaris and radix entomolaris) and 6 canals[768]	1	1
5 canals (2M and 3D)[769]	1	1
Total number of case report references and cases	86	67

MANDIBULAR SECOND MOLAR

External Root Morphology

The mandibular second molar normally has two roots (Figure 1-34).[1,3,4,6,15–17] The mesial and distal roots are usually closer or have a longer root trunk and slightly shorter radicular root ends, and are more frequently fused, compared to the mandibular first molar. The roots are broader buccolingually than mesiodistally. Root concavities are usually present on the mesial surfaces of both the mesial and distal roots and the distal surface of the mesial root.[16]

The overall average length of the mandibular second molar is 20 mm with an average crown length of 7 mm and an average root length of 13 mm.[4] Therefore, the overall dimensions of the second molar are smaller than the first molar.

Root Number and Form

The mandibular second molar is two rooted ~67% of the time (Table 1-44).[5,79,84,91,92,408,712,770,771] Root fusion, that becomes a single-root, conical or "C-shape" form, has an incidence of ~31.3%.

The incidence of a third root, usually the distolingual root, in mandibular second molars (1.3%) is not as high as in first mandibular molars (14.1%) (Table 1-44). However, patient of Asian ethnicity seemed to have a higher incidence of single or conical root.

Canal System

The mandibular second molar typically has two mesial and one distal canal (Tables 1-45 and 1-46). The mesial root of the mandibular second molar has a higher incidence of one canal (18.1%) than does the mesial root of the mandibular first molar (3.2%). Therefore, the two-rooted form may have only a two-canal system. On the other hand, since a distal root may have a double-canal system about 15% of the time, a four-canal system is not uncommon.

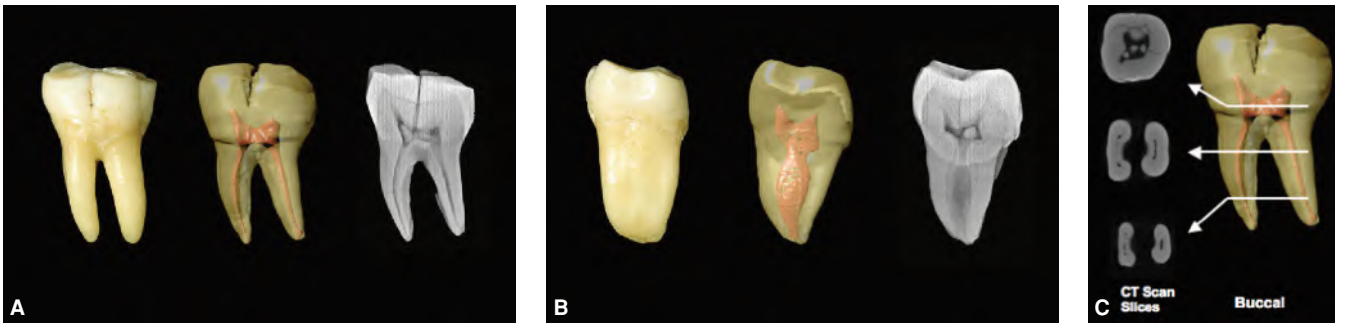


FIGURE 1-34 Buccal view of Mandibular left second molar (vertical crack in crown is an artifact). ***A.*** Buccal view. ***B.*** Mesial view. ***C.*** Root cross-sections. (Reprinted with permission from Brown P, Herbranson E. Dental Anatomy and 3-D Tooth Atlas Version 3.0. Illinois: Quintessence, 2005: Mandibular Second Molar- Rotations and Slices.)

TABLE 1-44 Root Number in the Mandibular Second Molara

References		No. of teeth in study	1-Fused Roots						2 Roots		3 Roots		4 Roots	
			Total Fused Roots		1 Root (conical)		1 Root (C-shaped)							
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of teeth in studies[5,36,67,79,84,91,92,101,104,110,111,118,119,176,181,408,712,770,771]	19	6210	31.3	1946	1.2	74	2.5	157	67.3	4181	1.3	83	0.0	0

TABLE 1-45 Main and Apical Canal Number of the 2-rooted Mandibular Second Molar

Mesial Root References		No. of teeth in study	1 canal		2 or more canals		1 canal at apex		2 or more canals at apex	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[7,11,14,84,91,92,104,110,181,408,460,772]	12	2061	18.1	374	81.9	1687				
Total number of canal apices studies	9	1160					58.0	673	42.0	487
Distal Root References		**No. of teeth in study**	**1 canal**		**2 or more canals**		**1 canal at apex**		**2 or more canals at apex**	
			%	No. of Teeth	%	No. of Teeth	%	No. of Teeth	%	No. of Teeth
Total number of canal studies[7,11,14,84,91,92,104,110,181,408,460,771]	12	2061	84.6	1743	15.4	318				
Total number of canal apices studies	9	1160					93.7	1087	6.3	73

TABLE 1-46 C-Shaped Canals in the Mandibular Second Molar

References		No. of Teeth in Study	C-shaped canals	
			%	No. of Teeth
Total number of C-shaped canal studies (all studies)	25	7985	20.6	1642
Total number of C-shaped canal studies (Non-Asian populations)[101,104,110,111,118,119,145,169,181,770–775]	15	4022	8.4	336
Total number of C-shaped canal studies (Asian populations)[57,176,61,36,776,777,91,92,60]	10	3963	33.0	1306

Mesial root canals may have a common foramen or may exit separately as two or more foramina, but the joining of the two canals is the most common form (Vertucci Type II).

Variations and Anomalies

Due to the higher incidence of root fusion in the mandibular second molar, C-shaped canals are not infrequent (Table 1-47). An extensive literature review by Fernandes et al.[45] found that the incidence of C-shaped canals ranged from 0.6% to 41.3% in the Chinese population and from 31.3% to 45.5% in the Korean population. The incidence of root fusion is generally higher in studies of Asian patients with Hou and Tsai,[448] reporting an incidence of fusion of 51.6% in the mandibular second molars in a Chinese population. In the same study, the maxillary second molar was found to have the highest incidence of fused roots (60.3%).

As would be expected, the incidence of C-shaped canals is often higher in Asian patients as well.[53] Seo and Park[777] reported an incidence of C-shaped canals in 32.7% of Korean patients and Gulabivala et al.[92] reported an incidence of 22.4% in the Burmese. Manning[49] reported a lower incidence of C-shaped canals at 12.7 but Caucasian ethnicity formed the majority of the patients. Studies in the United States by Weine et al.[771,772] and Sabala et al.[22] reported incidences of single root or C-shape of <10%. Ethnicity was not identified in either of these studies but it is likely that non-Asian patients formed the majority of

TABLE 1-47 Variations and Anomalies Associated with the Mandibular Second Molar

Anatomic Variation	No. of Cases	No. of References
C-shaped canal[38,49,784–790]	18	9
Taurodontism[429,495,497]	18	3
Fusion with a paramolar[791–796]	7	6
3 roots (MB, MLi, and D) and 3 canals[688, 797,798]	6	3
1 root and 1 canal[572,799]	4	2
4 roots and 4 canals[800,801]	2	2
3 roots (Radix entomolaris) and 4 canals[802]	1	1
2 roots and 5 canals (3M and 2D)[803]	1	1
Gemination or fusion?[804]	1	1
3 canals in the M root (mid-mesial canal)[730]	1	1
2 roots and 5 canals (3M and 2D)[731]	1	1
2 distinct pulp chambers and 5 canals were present[805]	1	1
2 roots and 3 canals (1M and 2D)[806]	1	1
Total number of case report references and cases	62	32

the patients in both the studies. The data from the studies that differentiated single conical and C-shaped roots indicate that the incidence of these two canal systems is approximately equal (8.3% and 8.5%, respectively).[49,91,92,408,712,771]

A variation in canal and root morphology was first termed as "C-shaped canal" by Cooke and Cox.[38] This canal shape results from fusion of the mesial and distal roots on either buccal or lingual root surface. A deep radicular groove is formed by Hertwig's root sheath that extends to the apical root end. The pulp canal system is of a "C" shape, which may vary in size with age of the patient.

Melton et al.[778] described three categories of C-shaped canals and Haddad et al.[775] added to the initial description. Category I is described as a continuous C-shaped canal from the pulp chamber to the apex. Category II is described as a "semi-colon" where one canal was separated by dentin from the C-shaped canal. The Category III, C-shaped anatomy, is described as having a C-shaped orifice with two or more distinct and separate canals. Although there have been reported cases of C-shaped canals in other teeth such as the maxillary lateral incisor, maxillary first molar,[450,498–501] maxillary second molar,[779,780] maxillary third molar,[82] mandibular first premolar,[126,781] mandibular first molar,[499,757,759] and the mandibular third molar,[38,82,782] with the most case reports are of the C-shaped mandibular second molar (Table 1-46). The incidence of C-shaped canals is the highest in mandibular second molars[36,38,45,57,60,61,79,783] ranging from 2%[119] to 44.5%.[776]

Other reported anomalies of mandibular second molars include fused or single roots, additional canals in one or more of the roots and two canals.

Munir et al.[807] found 12% incidence of taurodontism in 500 patients of a Punjab Dental Hospital. Two-thirds of the cases were meso-taurodontism (Figure 1-35) and <2% were considered hypertaurodont.

Just as in the other maxillary or mandibular molar teeth, hyper-taurodontism may occur in single instance in mandibular second molar teeth, which require endodontic treatment.[808]

SUMMARY

Maxillary Teeth

The preceding data aims to bring together the available knowledge of the anatomy of teeth in the human dentition in the dental literature. Because it comes from studies done with somewhat different methodologies and sources, it may not be amenable to meta-analysis, as such. However, some general observations and conclusions may be drawn about root and root canal anatomy of each tooth. Just as it is imperative to know what is the normal anatomy of a tooth, the range of variation and anomalous forms that a human tooth may take is equally

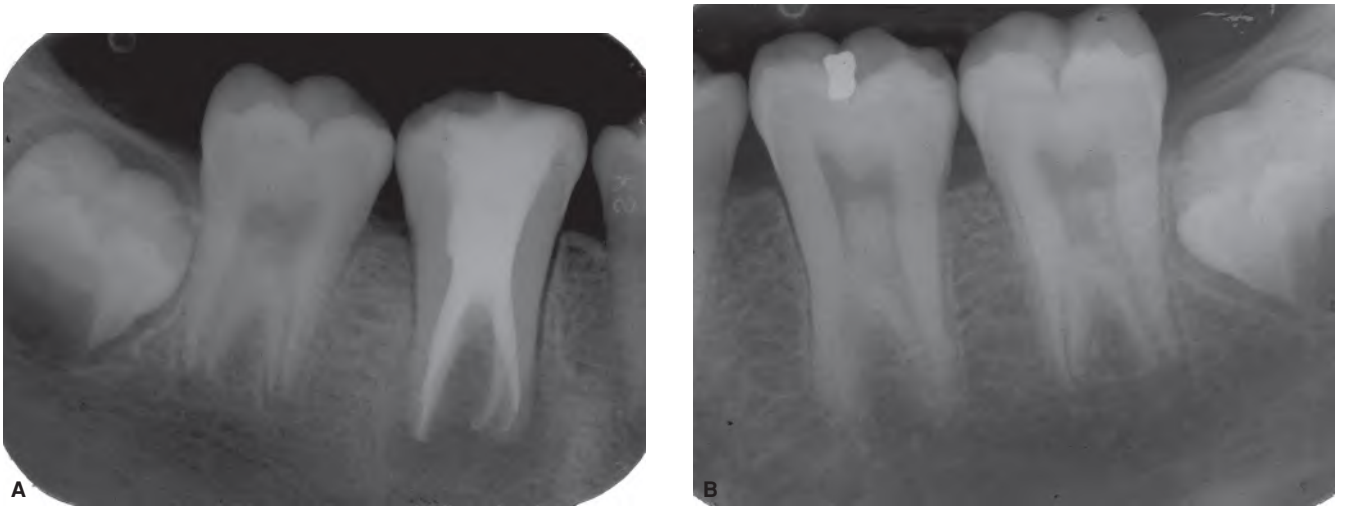


FIGURE 1-35 Bilateral mesotaurodont of mandibular second molars in the same patient. ***A.*** Right side. ***B.*** Left side. (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

important. The tooth bud and eventually the developing tooth come from genes of each parent. Therefore, each patient may present with a dentition that is unique and individual.

The maxillary incisor and the maxillary canine teeth are all represented as a single-rooted tooth when data from all studies are combined for analysis. Despite this superficial similarity in anatomy, there are some distinctive differences in the morphology and variations in these anterior teeth.

After compensating for access alignment on the lingual side of midline, a root canal system of a *maxillary central incisor* is usually the largest and straightest of all human teeth. Even the palatal root of a first maxillary molar generally has a curvature to the buccal in the apical region. Lateral canals and apical delta canal branches are common. Nonetheless, gemination and fusion in the incisors occurred at the highest rate of all the teeth in one study. Developmental anomalies may include talon cusps or dens evaginatus, dens invaginatus, and fusions, which produced "double teeth." These and other anomalies outside the normal appearance of the maxillary central incisor are found in individual case reports.

The *maxillary lateral incisor* has a root canal system that is finer and oval in size and may curve in its apical portion to the distal, lingual, or both. Uncommon two rooted forms are reported up to 4% of the teeth, often a result of fusion of root forms with a root groove resulting. A lingual groove anomaly may be associated with both pulpal and periodontal pathology as first reported by Simon et al.[221] The developmental lingual groove anomaly may occur in maxillary central incisor and maxillary canine teeth, but are much rarer.

The lateral incisor may show anomalies such as dens invaginatus, followed by dens evaginatus (talon cusp), palatogingival grooves, and two roots and two canals fusion with peg-laterals and rarely three or four canals.

The *maxillary canine* was formerly known simply as the "cuspid." It has been shrinking in size in the human dentition since the times of the late Miocene hominids,[809] with closure of the canine diastema, as well. The canine is oval with a taper to the lingual as seen in cross-section, and a single-rooted tooth unless afflicted with developmental anomalies such as dens invaginatus or gemination. Dens evaginatus or "talon cusps," usually located on the lingual, may also occur. There may be two canals within one root in <5% of teeth, but the canal usually exits as a single foramen. Double-rooted maxillary canines occur rarely, compared to the mandibular canine teeth.

The *maxillary first premolar* tooth in its classic form is a "bicuspid" with two cusps and two roots of approximately equal size when viewed in standard dental anatomy and endodontic texts. Thus, the root trunk is wider in a buccolingual dimension. Both a double-rooted form and a single-rooted morphology occur, but the triple-rooted variant occurs with a frequency of 0.6% in Asian ethnicity and 2.3% in Caucasian populations.

Regardless of root number and ethnicity, the majority of maxillary first premolar teeth (80%) had a two-canal system. Anomalies such as taurodontism and dens evaginatus are relatively rare in maxillary premolar teeth.

The *maxillary second premolar* at times may resemble the first premolar in a slightly diminished size, and with similar ethnicity differences in root and canal numbers. It has a single root form over 90% of the time. The two-rooted form comprises a range of 1.6% to 20% of the time. Although three-rooted forms exist, it is found in lesser numbers but often concurrent with a three-rooted first premolar. All studies showed the majority of maxillary second premolars that had one root with either one- or two-canal system, terminated in one foramen ~64% of the time. Taurodontism or multi-foramen Vertucci Type IV–VIII and so forth canals were also noted in case reports.

Nearly 98% of *maxillary first molar* teeth have three roots. Only 2% are found as two-rooted varieties and single or conical root anatomy is rare. The palatal root is larger in diameter as is the internal root canal, often curving in the buccal direction that is rarely noted in periapical radiographs. Fusion may occur with any of the two roots, but is more likely encountered in the second maxillary molar, and more so with the third molar tooth.

The mesiobuccal root has two canals in >60% of the teeth. The incidence of conical C-shaped roots in a maxillary first molar is relatively low at 0.1% to 0.3%. Other anomalies may include two canals in the palatal root, taurodont molar (both genetic syndromes and otherwise), and multiple canal systems beyond four canals at five-, six-, and even seven-canal molars.

The *maxillary second molar* will still most likely be a three-rooted tooth, but only 86% of the time. Incidence of fusion of roots into a two-rooted form may occur in 10% of the teeth, and interestingly the single-rooted form is at 4% and a four-rooted tooth may occur just <1% of the time. The incidence of double palatal roots is rare. Other anomalies include teeth with five or more than six canals, fusions and C-shape canals, and taurodontism in its hypo-, meso-, and hyper-taurodont forms. Taurodont teeth are no longer considered a trait specific to *Homo neanderthalensis* in Ice Age Europe, but genetic syndromes, recessive familial inheritance and no specific ethnicity have been implicated.

MANDIBULAR TEETH

Just as the maxillary teeth of the human dentition have their own range of variation of crown, root, and root canal systems, the mandibular arch has its own unique characteristics. The opposing tooth and its antimere may be within the normal range or may be an anomaly in root number or canal number. The genetic characteristics of size and root shape of the mandibular teeth also have evolved to fit the occlusion of the upper arch. The reduction in the size of teeth and profile of both alveolar processes seem to be happening more quickly in human evolution than the base of the lower mandible, thus leaving the protruding chin and jaw that is not found in other

hominids. The roots of the mandibular teeth may also exhibit some interesting evolutionary characteristics.

The *mandibular central incisor* is typically a single-rooted tooth with a smaller incisal ridge to fit inside the smaller mandibular arch. The incisor roots in cross-section are far from circular, being most often a wide, ovoid shape like in the maxilla with concavities on the mesial and distal surfaces. It may present a double or bifurcated root canal system about 1:5 times. Despite the double canal internally, only about 4% of the canals existed in more than one foramen. Developmental anomalies are relatively rare and include "talon cusp" or dens evaginatus, dens invaginatus, and some fusions with supernumerary teeth.

The *mandibular lateral incisor* is a slightly larger, but very similar in root morphology to the central incisor. It also contains a double-root canal system slightly more often at a 1:4 ratio, and most often ends in a single foramen (~96%). Root anomalies are rare.

The *mandibular canine* is usually, but not always, a single-rooted tooth with a unique blend of mandibular incisor root morphology and characteristic canine tooth size and function. Deep developmental grooves on both sides of the root may sometimes be visible in a buccolingual radiograph. Even though the incidence of a single canal may be 90%, it would be prudent to treat the canal system as a wide, buccolingual double-canal shape. The tooth bud may form only a single, long cusp during the development of the canine crown, but the prominence of mid-root grooves may contribute to a two-rooted apical formation. Developmental anomalies of the mandibular canine, other than double canals and two-rooted forms are rare.

The *mandibular first premolar* is mainly a single-rooted tooth (Vertucci type I), but may have two roots with a 3% incidence and even a triple-root or canal with <0.2% incidence. However, this extra root on the lingual aspect, known as "Tome's Root" may occur much more frequently if ethnicity of the patient is considered. The missed canal and complex anatomy may be a reason for many attempts at retreatment of a failing initial treatment that assumed only one canal is present.

The *mandibular second premolar* root morphology may resemble the first premolar as a single-rooted tooth, but ethnicity differences in incidence have been reported. Vertucci Type I single canal in a single root is predominant. The two-root and two-canal morphology was rare at 0.5%, and triple-root forms at <0.1%. Even in the single-root morphology, a single canal compared to two or more canals ratio was about 9:1.

A coronal anomaly that may afflict premolar teeth is the dens evaginatus, or dental tubercle on the mid-occlusal surface. Since this delicate enamel projection often includes a fine pulp horn, occlusal attrition may create a pulp exposure at a young age. This developmental anomaly may be present predominantly in patients of Asian ethnicity. Preventive restorative measures or apexogenesis treatment may be required as a consequence to retain the tooth or teeth.

The *mandibular first molar* is usually a two-rooted tooth with three canals, two mesial and one distal. However, many more complex canal numbers and shapes can exist. The morphology of the two-rooted form may also vary from long, well-developed, and recurved roots that may resemble a "cow-horn," to short and parallel, almost fused double roots. Genetics play a role in root anatomy as studies show, with Asian populations having 78% of mandibular first molar teeth being two-rooted and up to 22% may have a third, or distolingual root. Other studies show that 96% of non-Asian populations are likely to be two-rooted and a three-rooted variant occurs <4% of the time. In average, two roots were observed in 86% of cases. The mesial root anatomy is of great interest. A single Vertucci Type I canal may occur >3% of the time, any of the two-canal types may occur 96% of the time, and a three-canal system in one root has about 1% incidence. The "mid-mesial canal" is now more routinely recognized if the SOM and ultrasonic trough techniques are used.

The *mandibular second molar* is usually a two-rooted tooth (2:3 ratio) with the roots usually smaller and developing closer together, to the point where they may appear fused externally, but retaining molar-like or three-canal root canal anatomy inside. Conical root, single canal form (Figure 1-36) and "C-shaped" root and canal system are found more frequently. Taurodontism, or the "bull-shaped" root trunk and pulpal anatomy may be occasionally found.

Tables 1-48 to 1-51 summarize the most relevant data on root number and root canal number of each tooth in the maxillary and mandibular arch.

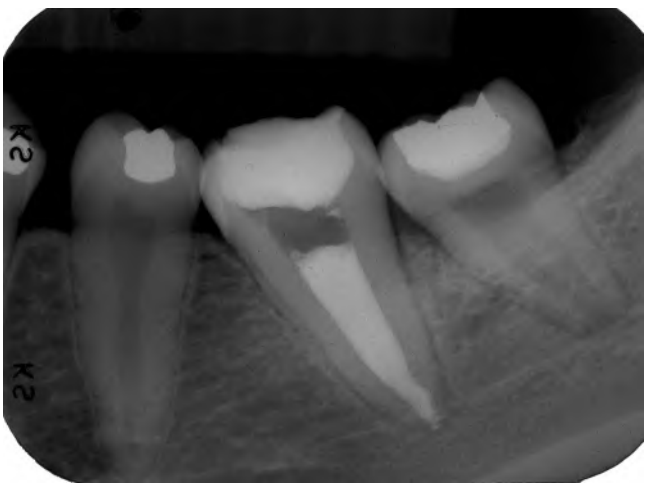


FIGURE 1-36 Mandibular left second molar with one conical and one canal root. (Courtesy of Dr. William H. Christie, Winnipeg, MB, Canada.)

TABLE 1-48 Summary Table of the Root Numbers of the Permanent Maxillary Teeth

Tooth Type	No. of Roots						No. of Studies	No. of Teeth	Most Common Anomaly or Variation (number of case reports in brackets)
	Most Common	1	2	3	4	5			
Central Incisors	1 Root	100					7	892	Dens evaginatus (17) 2 roots and 2 canals (14) 1 root and 2 canals (9) Fusion (9)
Lateral Incisors	1 Root	100					7	827	Dens invaginatus (46) Dens evaginatus (talon cusp) (16) Palatogingival groove (12) 2 roots and 2 canals (10)
Canines	1 Root	100					7	842	Dens invaginatus (7) 1 root and 2 canals (2) Dens evaginatus (talon cusp) (2) 2 roots (2)
First Premolar							18	7628	3 roots and 3 canals (20) Furcation groove (palatal of B root) (3) Dens evaginatus (2)
Caucasian	2 Roots	37.0	60.8	2.3			13	2647	
Asian and NA Native (Mongoloid)	1 Root	61.8	37.6	0.6			5	4981	
Second Premolar	1 Root	91.5	8.4	0.2			8	9033	3 roots and 3 canals (13) Dens evaginatus (2)
First Molar	3 Roots (MB, DB and Li)	0.2	2.1	97.7			12	2744	3 roots (MB, DB and Palatal and 4 canals (MB, DB and 2 Palatal) (13) Taurodontism (8) Fused roots and C-shaped canal (9) 4 roots (MB, DB and 2 Palatal) and 4 canals (MB, DB and 2 Palatal) (8)
Second Molar	3 Roots (MB, DB and Li)	4.4	9.0	85.8	0.8		9	2845	4 roots (MB, DB and 2 Palatal) and 4 canals (MB, DB and 2 Palatal) (28)
Third Molar	3 Roots (MB, DB and Li)	31.8	27.0	38.1	3.1	0.1	5	778	4 roots (3) C-shaped canal (1)

TABLE 1-49 Summary Table of the Root Canal Systems of the Permanent Maxillary Teeth

	No. of Canals						No. of Studies	No. of Teeth	Most Common Anomaly or Variation (number of case reports in brackets)
	Most Common	1	2	3	4	Other			
Central Incisors *2 or more canals	1 Canal	99.2	0.8*				8	2435	Dens evaginatus (17) 2 roots and 2 canals (14) 1 root and 2 canals (9) Fusion (9)
Lateral Incisors *2 or more canals	1 Canal	97.4	3.6*				8	2331	Dens invaginatus (46) Dens evaginatus (talon cusp) (16) Palatogingival groove (12) 2 roots and 2 canals (10)
Canines *2 or more canals	1 Canal	95.3	4.7*				9	2615	Dens invaginatus (7) 1 root and 2 canals (2) Dens evaginatus (talon cusp) (2) 2 roots (2)
Caucasian	2 Canals	10.4	86.3	1.6		1.6	17	3405	
Asian and NA Native (Mongoloid)	2 Canals	34	63.2	0.4		2.4	4	1574	

TABLE 1-49 Summary Table of the Root Canal Systems of the Permanent Maxillary Teeth (*Continued*)

Second Premolar	1 Canal	51.8	46.2	0.8		2.0	16	4829	3 roots and 3 canals (13) Dens evaginatus (2)
First Molar (three roots) *2 or more canals									3 roots (MB, DB and Palatal and 4 canals (MB, DB and 2 Palatal) (13) Taurodontism (8) Fused roots and C-shaped canal (9) 4 roots (MB, DB and 2 Palatal) and 4 canals (MB, DB and 2 Palatal) (8)
MB	2 Canals	40.6	59.4*				71	14346	
DB	1 Canal	98.8	1.2*				26	5758	
Palatal	1 Canal	99.3	0.7*				26	5758	
Second Molar (three roots) *2 or more canals									4 roots (MB, DB and 2 Palatal) and 4 canals (MB, DB and 2 Palatal) (28)
MB	1 Canal	54.8	45.2*				27	5016	
DB	1 Canal	99.6	0.4*				16	3085	
Palatal	1 Canal	99.8	0.2*				15	3035	
Third Molar	3 canals	6	9.2	60.1	24.3	2.5	3	436	4 roots (3) C-shaped canal (1)

TABLE 1-50 Summary Table of the Root Numbers of the Permanent Mandibular Teeth

Tooth Type	No. of Roots						No. of Studies	No. of Teeth	Most Common Anomaly or Variation (number of case reports in brackets)
	Most Common	1	2	3	4	Other			
Central Incisors	1 Root	100					8	3286	Dens invaginatus (5) Dens evaginatus (talon cusp) (4)
Lateral Incisors	1 Root	100					8	3266	Dens invaginatus (4)
Canines	1 Root	95.5	4.5				4	7550	2 roots and 2 canals (8) 2 roots and 3 canals (3) 1 root and 2 canals (3)
First Premolar	1 Root	95.5	2.7	0.2			15	6348	3 roots and 3 canals (5) Dens evaginatus (4) 1 root and 2 canals (3) 1 root and 3 canals (3) 2 roots and 2 canals (3)
Second Premolar	1 Root	99.4	0.5	0.1			14	5055	3 canals (12) 2 roots and 2 canals (10) Dens evaginatus (6) 3 roots and 3 canals (5)
First Molar (all studies)	2 Roots (M&D)	0.3	85.6	14.1	0.0		36	17780	Radix entomolaris (23) 2 roots and 5 canals (3M and 2D) (19) 2 roots and 4 canals (3M and D) (10) 2 roots and 5 canals (2M and 3D) (8)
Caucasian	2 Roots (M&D)	0.1	96.1	3.7	0.0		23	7615	
Asian and NA Native (Mongoloid)	2 Roots (M&D)	0.4	77.7	21.9	0.0		13	10165	
Second Molar	2 Roots (M&D)	31.3	67.3	1.3	0.0		19	6210	C-shaped canal (18) Taurodontism (18) Fusion with a paramolar (7) 3 roots (MB, MLi, and D) and 3 canals (6) 1 root and 1 canal (3)
Third Molar	2 Roots (M&D)	43.2%	53.5%	3.8%	0.1%	0	9	13753	Highly variable; variation is the norm

TABLE 1-51 Summary Table of the Root Canal Systems of the Permanent Mandibular Teeth

Tooth Type	No. of Canals						No. of Studies	No. of Teeth	Most Common Anomaly or Variation (number of case reports in brackets)
	Most Common	1	2	3	4	Other			
Central Incisors	1 Canal	81.1%	18.8%			0.2%	18	7455	Dens invaginatus (5) Dens evaginatus (talon cusp) (4)
Lateral Incisors	1 Canal	75.4%	24.6%			0.04%	18	7200	Dens invaginatus (4)
Canines * 2 or more canals	1 Canal	90.5%	9.7*				14	6081	2 roots and 2 canals (8) 2 roots and 3 canals (3) 1 root and 2 canals (3)
First Premolar * 2 or more canals	1 Canal	77.3%	22.7*				29	8538	3 roots and 3 canals (5) Dens evaginatus (4) 1 root and 2 canals (3) 1 root and 3 canals (3) 2 roots and 2 canals (3)
Second Premolar * 2 or more canals	1 Canal	91.3%	8.7*				19	5521	3 canals (12) 2 roots and 2 canals (10) Dens evaginatus (6) 3 roots and 3 canals (5)
First Molar (Two Roots) * 2 or more canals									Radix entomolaris (23) 2 roots and 5 canals (3M and 2D) (19) 2 roots and 4 canals (3M and D) (10) 2 roots and 5 canals (2M and 3D) (8)
Mesial	2 Canals	3.2%	95.6%	1.1%		0.1%	20	5824	
Distal	1 Canal	69.3%	30.7*				21	5865	
First Molar (Three Roots) * 2 or more canals									
Mesial	2 Canals	2.2%	97.8*				5	724	
Distobuccal	1 Canal	98.6%	1.4*				5	724	
Distolingual	1 Canal	100					6	732	
Second Molar (Two roots) * 2 or more canals									C-shaped canal (18) Taurodontism (18) Fusion with a paramolar (7) 3 roots (MB, MLi, and D) and 3 canals (6) 1 root and 1 canal (3)
Mesial	2 Canals	18.1%	81.9*				12	2061	
Distal	1 Canal	84.6%	15.4%				12	2061	
Third Molar	2-3 Canals	7.0%	43.2%	37.9%	7.7%	4.1%	3	414	Highly variable; variation is the norm

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Chapter 2

Structure and Function of the Pulp–Dentin Complex

Sivakami R. Haug, Syngcuk Kim, Karin J. Heyeraas

The dental pulp is a connective tissue. It consists of nerves, blood vessels, interstitial fluid, odontoblasts, fibroblasts, and other cellular components. As late Dr. I.B. Bender said, "The pulp is a small tissue with a big issue." The dental pulp resides in a rigid capsule consisting of enamel and dentin layers. The uniqueness of the pulp can be appreciated only when provoked. Unlike other connective tissues, this encasement creates a low-compliant environment in which a small increase in tissue volume, due to inflammation, has serious consequences. Moreover, the pulp receives its blood supply from end arteries, and the main blood supply goes through the apex such that there is no collateral blood circulation. Another uniqueness of the pulp is that it is densely innervated by sensory and sympathetic nerve fibers.

Despite its confinement by enamel and dentin, the pulp–dentin complex functions as an exquisitely responsive sensory system, using unusual mechanisms to defend itself from insults. Sensory fibers together with its microcirculatory vasculature make the dental pulp a unique organ to study the neurovascular interactions during various stages of inflammation. When the dental pulp is involved, dentists face diagnostic dilemmas, especially when presented with thermal sensitivity, sensitivity to osmotic pressure, and various grades of non-localized pain. Therefore, it is important that clinicians have a sound biological and physiological knowledge for diagnosis and treatment planning. As such, one must understand the very tissue frequently eliminated by endodontic therapy.

This chapter discusses the available information on the development, structure, and function of the dentin–pulp complex, with the aim that this knowledge provides a firm biological basis for clinical decision-making.

EMBRYOLOGY

Human tooth development begins as early as the fifth week of gestation for primary dentition. Mineralization of the primary dentition begins during the 14th week of gestation at the same time the permanent teeth start to develop. The first primary teeth erupt at the age of 6 months and the first permanent teeth at 5–6 years. The third molars are the last teeth to be formed with the completion of crown development between 12 and 16 years. Thus, tooth development starts from the embryonic stage and continues into early adulthood. Recent findings have revealed multiple growth and transcription factors, from several signaling families, acting as critical regulators at the initiation, and subsequently throughout all stages of tooth development. Gene-targeting experiments have demonstrated the biological function of transcription facts in regulating tooth morphogenesis. Today, over 200 genes have been demonstrated to be active in the developing tooth. This chapter overviews the topic of tooth development while referring to excellent reviews in this field.[1–4]

Teeth derive from two principal cell types: the ectoderm and the cranial neural crest-derived ectomesenchyme. The ectodermal component gives rise to the ameloblasts that form the enamel of the tooth crown while the ectomesenchyme gives rise to the dentin, pulp, and periodontal tissue. The neural crest cells migrate from the crest of neural ectoderm. Only neural crest-derived mesenchyme has the ability to respond to the initial signals from the dental epithelium and contribute to tooth formation.[5–7] Initiation of tooth development begins with the formation of dental lamina, a thickening of the oral epithelium. Localized proliferation within the dental lamina leads to the formation of a series of epithelial outgrowths or buds at sites where the tooth should be, thus marking the time and position of tooth development. The underlying mesenchyme underneath, called the dental papilla, forms the future dentin and pulp. The dental follicle, or sac, surrounds both the dental papilla and the enamel organ and gives rise to the supporting tissue.

There are three stages in the early tooth development: the bud, the cap, and the bell stage. During the bud stage, the mesenchyme underneath condenses and begins to appear like a cap on the dental papilla, thus leading to the stage known as the cap stage. The dental epithelium comes to be known as the enamel organ from the cap stage. The enamel organ, the dental papilla, and the dental follicle constitute the dental organ, or the tooth germ (Figure 2-1). Transition from the bud to the cap stage is a critical step in tooth morphogenesis involving multiple signaling molecules within the enamel organ epithelium. These have been implicated to regulate the expression of various transcription factors in the surrounding mesenchyme.[4,8,9] During the cap stage, a critical signaling center, the enamel knot, is formed within the enamel organ epithelium.

The enamel knot is a dense population of epithelial cells, without any proliferative activity, and is marked by the expression of multiple signaling molecules for the proper development of the tooth organ. The enamel knot serves as a transient signaling center and is removed by programmed

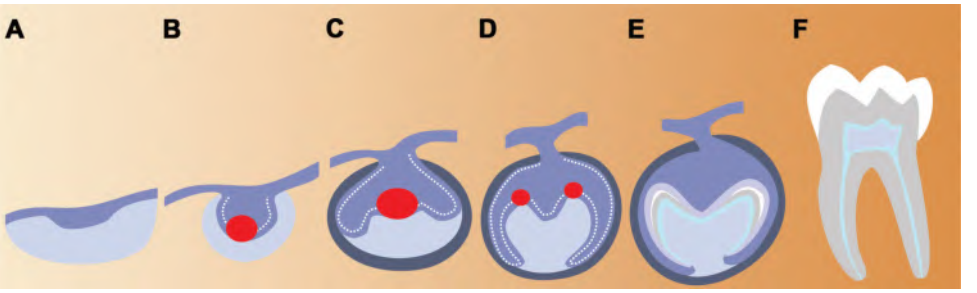


FIGURE 2-1 Schematic presentation of the embryonic molar tooth development and the adult tooth. ***A.*** Due to localized proliferation of the oral epithelium, thickening of the dental epithelium is formed at the beginning of tooth development. ***B.*** The ingrowth of dental epithelium into the condensed dental mesenchyme occurs and the dental bud is formed. At this bud stage, the primary enamel knot (PEK) (red dot), the signaling center, appears in the tip of the epithelial bud. ***C.*** The dental epithelium on the lingual and buccal sides of the PEK grows and cervical loop is forming at the cap stage. Dental mesenchyme becomes the dental papilla and dental follicle. ***D.*** The secondary enamel knots (SEK) (red dots) appear at the sites of the future cusp tips at the early bell stage. The epithelial cervical loop grows and surrounds the dental pulp. ***E.*** Tooth specific cells, odontoblasts and ameloblasts differentiate and secrete dentin (light grey) and enamel (white), respectively, at the late bell stage. Later the crown formation is followed by root formation (not shown) ***F.*** Mature molar tooth. ***A–E.*** coronal view. ***F.*** sagittal view (The inner and outer enamel epithelia are demarcated by a white dashed line. A, ameloblast; de, dental epithelium; dm, dental mesenchyme; df, dental follicle; o, odontoblast). (Courtesy of Dr. Paivi Kettunen, Bergen, Norway.)

cell death. In molar teeth, this primary enamel knot is replaced by secondary enamel knots that are responsible for inducing cusp development. The enamel knot is involved in the patterning process such as cusp shape and size during tooth morphogenesis.[4]

Determination of tooth identity depends on the expression of certain homeobox genes in the mesenchyme.[10,11] Continuous growth of the tooth germ leads to the bell stage during which the crown assumes its final shape (morphodifferentiation) and the cells that will be making the hard tissue (ameloblasts and odontoblasts) acquire their distinctive phenotype (histodifferentiation). Cranial neural crest-derived odontogenic mesenchyme and its adjacent inner enamel organ epithelium differentiate into preodontoblast and preameloblast, respectively. Dentin and enamel will begin to form at the junction between odontoblasts and ameloblasts to complete the tooth-forming process.

Blood supply to the developing tooth is via the dental follicle during the cap stage. The enamel organ is avascular, although there are numerous blood vessels in the adjacent follicle. These blood vessels ramify around the tooth germ. Their number in the dental papilla increases, reaching a maximum when matrix deposition begins. The vessels in the papilla are arranged in groups that coincide with the future root-forming site.

The first trigeminal nerve fibers are present in the vicinity of the tooth at the bud stage and around the tooth organ in the dental follicle during the cap and bell stages.[12–16] In the bell stage, sensory nerve fibers are present in the dental follicle but not in the dental papilla and enamel organ. In the advanced bell stage, after initiation of dentin and enamel formation, sensory nerve fibers enter the dental papilla. Sympathetic innervation appears later at the beginning of the root formation.[17] With the start of root development, a subodontoblastic nerve plexus gradually forms. Also with the start of root formation, nerves begin to appear in the developing periodontal ligament (PDL), although a mature distribution pattern does not occur until root formation is nearly completed.[17] The expression of growth factors such as nerve growth factor, brain-derived neurotrophic factors, gondotropin neurotrophic factor, neurotrophin 3, and neurotrophin has been shown at specific locations during various developing stages of the tooth.[18–20] Recent studies have shown that enamel and dentin formation is under circadian control. Clock genes may be involved in the regulation of ameloblast and odontoblast functions, such as enamel and dentin matrix protein secretion and biomineralization expressing incremental lines and cross-striations. Clock genes, such as Bmal1, Clock, Per 1, and Per 2, which are known to control circadian rhythms and related functions in many tissues, are expressed in teeth as well. Clock protein expression has been found preferentially in matrix secreting cells such as ameloblasts, odontoblasts, and osteoblasts and not in cementoblasts.[21,22]

STRUCTURES AND FUNCTIONS

Dental–Pulp Complex

As long as dentin is covered peripherally by enamel on coronal surfaces and cementum on radicular surfaces, the dental pulp will generally remain healthy for life, unless the apical blood supply is disrupted (e.g., severe impact trauma, surgical procedures, or excessive orthodontic forces). Most pathological pulp conditions begin with the removal of one or both these protective barriers via caries, fractures, or abrasion. The result is the communication of the pulp soft tissue with the oral cavity via dentinal tubules. A classic work by Pashley et al.,[23] using radioactive I^{131} in dog teeth, clearly demonstrated that once dentin tubules are opened, molecules can easily permeate to the pulp (Figure 2-2). This dentin permeability has a significant impact on the pathogenesis of the pulp in many clinical situations. This will be discussed later in this chapter.

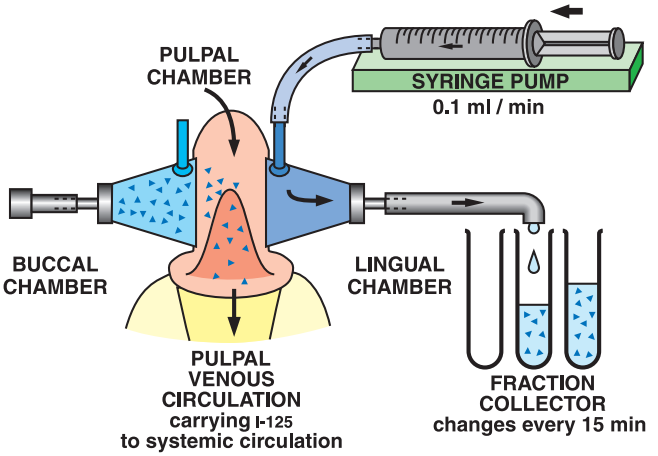


FIGURE 2-2 Pashley's experimental setup for the study of dentin permeability. ^{131}I, as the marker, was diffused through the buccal chamber attached to the naked dentin of the tooth. Blood was drawn from the venous system and checked for ^{131}I radioactivity. The appearance of ^{131}I in the blood demonstrates the degree of permeability of the dentin. (Reproduced with permission from Pashley DH, et al.[23])

It is apparent that substances easily permeate dentin, permitting thermal, osmotic, and chemical insults to act on the pulpal constituents. The initial stages involve stimulation or irritation of odontoblasts and may proceed to inflammation and often to tissue destruction. First, one must examine the dentin structure.

Dentin Structure

Dentin is a calcified connective tissue consisting of ~70% inorganic material and 10% water. Organic matrix accounts for 20% of dentin, of which about 91% is collagen. Most of the collagen is type I, but there is minor amount of component of type V. Noncollagenous matrix components include phosphoproteins, proteoglycans, γ-carboxyglutamaten-containing proteins, acidic glycoproteins, growth factors, and lipids. Dentin is penetrated by millions of tubules; their density varies from 40,000 to 70,000 tubules per square millimeter (Figure 2-3).[24] Tubules range in diameter from 1 μm at the dentinoenamel junction to 3 μm at their pulpal surface. These variations in the size of the tubules, in the dentin surface area, and in the surface area covered by the tubules are important in the pathogenesis of pulpal inflammation. For instance, permeation of toxic substances to the pulp created by caries or restorative procedures from the dentinoenamel junction is less likely compared to permeation from the middle of the dentin due to the degree of surface area covered by the tubules.

From a purely anatomical standpoint, possible pulpal damage increases exponentially the deeper toxins reach into the dentin. Dental tubules contain a fluid that has a composition similar to extracellular fluid.[25] If the fluid becomes contaminated with carious bacterial endotoxins and exotoxins, it becomes a reservoir of injurious agents that can permeate through dentin into the pulp to initiate inflammation.[26] It is useful to understand the important variables that control dentin permeability.

Dentin Permeability

Dentinal tubules in the coronal dentin converge from the dentinoenamel junction to the pulp chamber.[27] This tends to concentrate permeating substances into a smaller area at their terminus in the pulp. The surface area occupied by tubules, at different levels, indicates the effect of tubule density and diameter. One can calculate from Garberoglio and Brännström's[24] observations that the area of dentin occupied by tubules is only 1% at the dentinoenamel junction and it increases to 45% at the pulp chamber (Figure 2-3). Its clinical

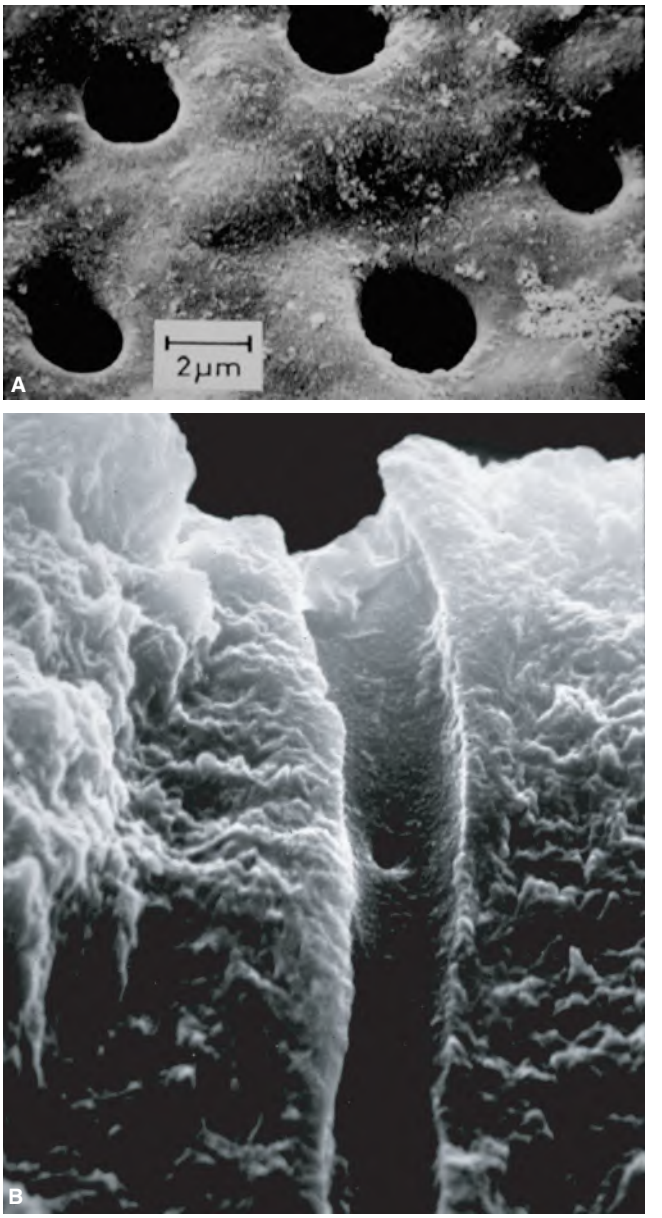


FIGURE 2-3 ***A.*** Heterogenous nature of the dentin tubular diameters along the dentin length and dentin surface. The tubular diameter increases (up to 4 μm) and the dentin surface area occupied by the tubules increases logarithmically as it approaches the pulp. ***B.*** Scanning electron micrograph (SEM) of a crosssection of tubules. (Courtesy of Dr. Henry Trowbridge, Philadelphia, PA, U.S.A. and Dr. David H. Pashley, Augusta, GA, U.S.A.)

implications are significant. As dentin is exposed to increasing depths, by restorative procedures, attrition, or disease, the remaining dentin becomes increasingly permeable.[28,29] Thus, dentin removal renders the pulp more susceptible to chemical or microbial irritation. This functional consequence of tubule area is also responsible for the decrease in dentin microhardness closer to the pulp.[30,31] As tubule density increases, the amount of calcified matrix between the tubules decreases. This relative softness of the dentin lining the pulp chamber facilitates canal enlargement during endodontic treatment.[32]

Overall dentin permeability is directly proportional to the total surface area of exposed dentin. Obviously, a leaking restoration over a full crown preparation provides more surface for diffusion of microbial products than a small occlusal restoration.[33] Restorations requiring extensive and deep removal of dentin (i.e., preparation for a full crown) would open more tubules and increase the rate of injurious substances diffusing from the surface to the pulp; thus, the importance of "remaining dentin thickness."[34,35]

The permeability of the root is 10 to 20 times less than that of a similar thickness of coronal dentin.[36] This may account for the lack of pulpal reactions to periodontal therapy that removes cementum and exposes root dentin to the oral cavity. Recent evidence indicates that dentin permeability is not constant after cavity preparation. In dogs, dentin permeability decreased over 75% in the first 6 hours following cavity preparation.[37] Although there were no histological correlates of the decreased permeability, dogs depleted of their plasma fibrinogen did not decrease their dentin permeability following cavity preparation.[38] These authors speculated that irritation to pulpal blood vessels caused by cavity preparation increased the leakage of plasma proteins from pulpal vessels into the dentinal tubules, where they absorb to the dentin, decreasing permeability. Future study of this phenomenon is required to determine if it occurs in humans.

The character of the dentin surface can also modify dentin permeability. Two extremes are possible: tubules that are completely open, as seen in freshly fractured[39] or acid-etched dentin,[40] and tubules that are closed, either anatomically or with microcrystalline debris.[41] This debris creates the "smear layer" that forms on dentin surfaces whenever they are cut with hand or rotary instruments.[42] The smear layer slows microbial penetration,[43,44] but permits a wide range of molecules to readily permeate dentin. Small molecules permeate much faster than large molecules. Smear layers are often slowly dissolved over months to years as oral fluids percolate around microleakage channels between restorative materials and the tooth. The removal of the "smear layer" by acid etching, or chelation, increases dentin permeability because the microcrystalline debris no longer restricts diffusion of irritants and also permits microorganisms to penetrate into dentin.[45,46] There is considerable debate as to whether smear layers created in the root canal during biomechanical preparation should be removed.[47] Its removal may increase the quality of the seal between endodontic filling materials and root dentin. It may also increase the bond strength of resin posts.[48]

Pulp Reaction to Permeating Substances

What happens when permeating substances reach the pulp chamber? Although microorganisms may not actually pass through dentin, their toxic by-products have been shown to cause severe pulp reaction.[49–51] The broad spectrum of pulp reaction, from no inflammation to abscess formation, may be related to the concentration of these injurious substances in the pulp. Although exposed dentin may permit substances to permeate, their concentrations may not reach levels high enough to trigger the cascade of events associated with inflammation. This would indicate that the interstitial fluid concentration of these substances can be maintained at low concentrations. As long as the rate of pulpal blood flow is normal, the microcirculation is very efficient at removing substances diffusing across dentin to the pulp chamber. This delicate balance between the dentin and the pulp is illustrated in Figure 2-4.

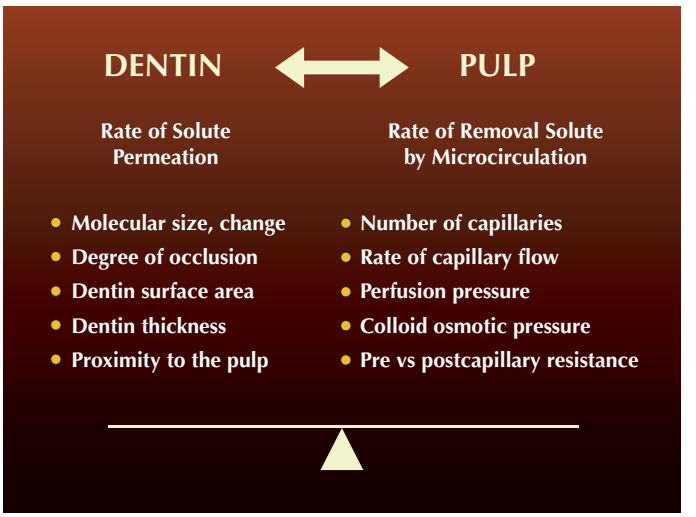


FIGURE 2-4 Diagram showing the delicate balance between dentin and the pulp. On the left side of the balancing beam are factors governing the dentin and on the right side are factors that govern the pulp.

Dentin Sensitivity

Clinicians recognize that dentin is exquisitely sensitive to certain stimuli. It is unlikely that this sensitivity results from direct stimulation of nerves in dentin (Figure 2-5). As previously stated, nerves have not been shown in peripheral dentin.[52,53] Another speculation is that the odontoblastic process may serve as excitable "nerve endings" that would, in turn, excite nerve fibers shown to exist in deeper dentin, closer to the pulp.[52–54]

The experiments by Anderson et al.[54] and Brännström[55] suggested that neither odontoblastic processes nor excitable nerves exist within dentin. They proposed the "hydrodynamic theory" of dentin sensitivity that sets forth that fluid movement through dentinal tubules, moving in either direction, stimulates sensory nerves in dentin or the pulp.[55,56]

Further support for the hydrodynamic theory came from electron microscopic examination of animal[57–59] and human dentin,[55,59–61] demonstrating that odontoblastic processes seldom extend more than one-third the distance of the dentinal tubules. A work by La Fleche et al.[62] suggested that the process may retract from the periphery during extraction or processing. Obviously, more investigation will be required before any definitive statement can be made regarding the distribution of the process. The tubules are filled with dentinal fluid, similar in composition to interstitial fluid.[25] The hydrodynamic theory satisfies numerous experimental observations. Although it cannot yet be regarded as fact, it has provided and will continue to provide a very useful perspective for the design of future experiments.[25]

Pulpal Microvasculatures

The arterial supply of the dental pulp has its origin from the posterior superior alveolar arteries and the infraorbital and the inferior alveolar branch of the internal maxillary arteries. Because the dental pulp itself is small, pulp blood vessels do not reach a large size. As such, the terms artery and vein cannot be used. Rather they are called arterioles (diameter in the range of 100 μm) and corresponding venules (diameter in the range of 200 to 300 μm).[63] Arterioles pass through the root pulp to supply the coronal pulp. As the arterioles travel straight to the coronal area, branching patterns develop. These branching vessels have very thin smooth muscle coating at the juncture and lose the coating as they reach toward the dentin, forming a capillary network. Near the dentin, around the odontoblastic area, they form a dense terminal capillary network in the subodontoblastic region[64] Figures 2-6 to 2-11 illustrate the basic microvascular units in the pulp. Terminal capillary networks are the key vessels in the pulp and are involved in transporting nutrients and O2 to the cells and removing waste products and CO_2 from the cells to maintain pulp homeostasis.

Corrosion cast studies have shown that numerous arterioles and venules pass through the apex of the root.[65] Vessels also enter and leave the pulp via accessory lateral canals that may be located anywhere on the root, but most commonly in the apical region.[66,67] The tooth lacks a collateral or alternative blood supply that makes the apical region a critical point. Thus, increased tooth movement in the apical area due to trauma, periodontitis, or excessive and uncontrolled

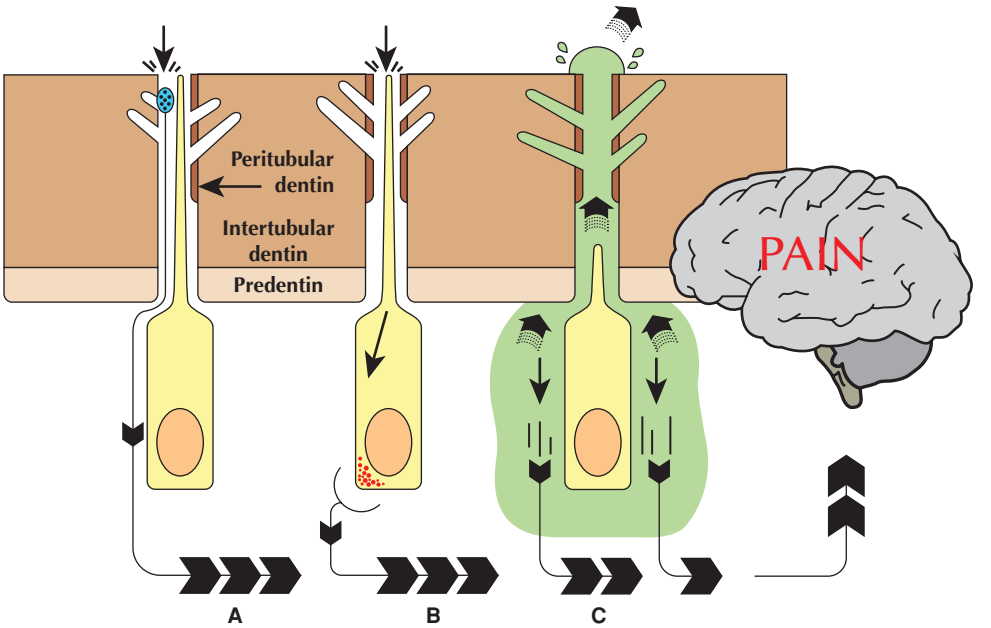


FIGURE 2-5 Diagram of essentials of three theories of dentin sensitivity. ***A.*** Classical theory proposed that stimuli applied to dentin caused direct simulation of nerves in dentin. ***B.*** Modified theory proposed that stimuli applied to the odontoblastic process would be transmitted along the odontoblast and passed to the sensory nerves via some sort of synapse. ***C.*** Hydrodynamic theory proposed that fluid movement within tubules transmits peripheral stimuli to highly sensitive pulpal nerves. (Modified with permission from Dr. Calvin D. Torneck, Toronto, Ontario, Canada.)

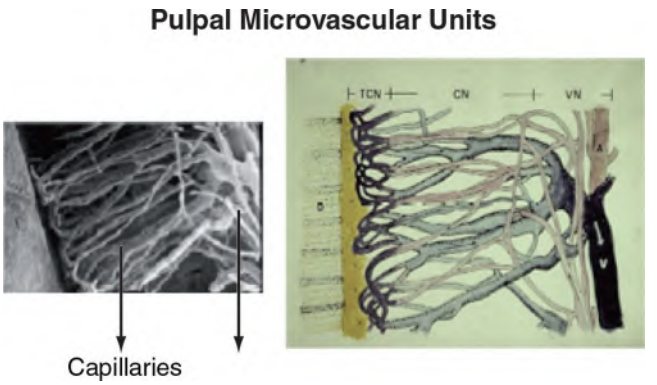


FIGURE 2-6 Pulpal microvascular units. Scanning electron micrograph (SEM) and a diagram based on the SEM are presented on the right side. The basic unit shows arterioles running parallel with venules in the center of the pulp. The small arterioles branch off almost 90° from the main arterioles. These small arterioles get smaller in diameter as they come near the dentin. These small arterioles also lose their smooth muscle coating and become capillaries forming the capillary network (CN). When they reach the odontoblastic layer, they become the terminal capillary network (TCN). They drain into the venular network (VN). (Courtesy of Drs. Kazuto Takahashi and Yoshiaki Kishi, Kanagawa, Japan.)

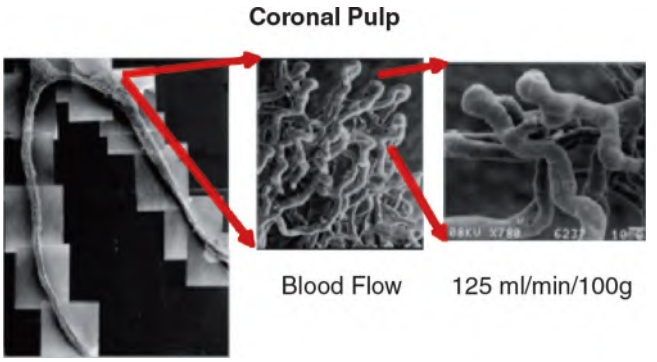


FIGURE 2-7 Corrosion resin casts of the pulp vessels of a dog premolar. These scanning electron micrographs (SEMs) clearly show heterogeneity of the pulpal microvascular architecture. Higher magnification of the coronal pulp region (arrows) reveals many hairpin capillary loops within the pulp horn. Higher magnification of the area (left) shows an individual loop with a diameter just over 10 mm. The blood flow per unit volume per minute is 125 mL/min/100 g tissue. Higher magnification of the root canal lesion in the apical pulp shows a very different architecture than the coronal pulp. Here, the microvessels show a net-type architecture surrounding the main arteriole and venules. The blood flow in this lesion is 22 mL/min/100 g, significantly less than that in the coronal pulp. (Courtesy of Drs. Kazuto Takahashi and Yoshiaki Kishi, Kanagawa, Japan, and Syngcuk Kim, Philadelphia, PA, U.S.A.)

orthodontic tooth movement may damage the pulpal blood supply. There is evidence that vessels have been identified connecting the pulp to the PDL (Figure 2-8). The degree to which these vessels play a role in pathogenesis remains to be investigated.

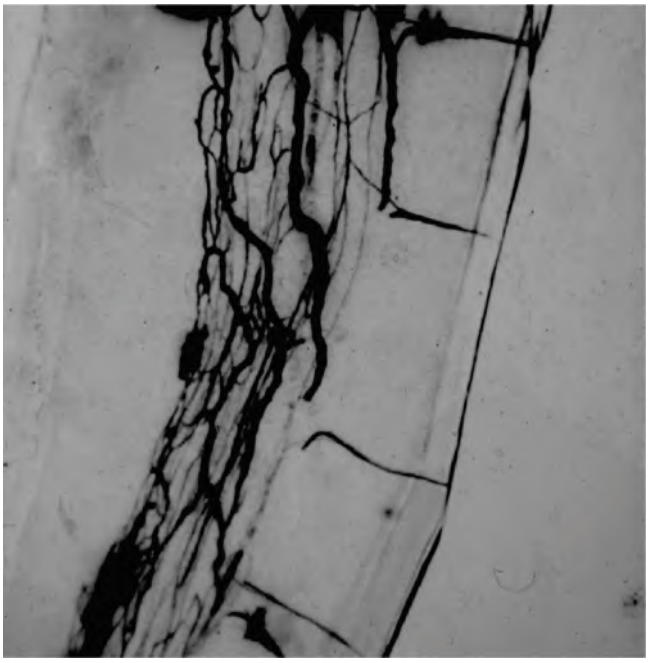
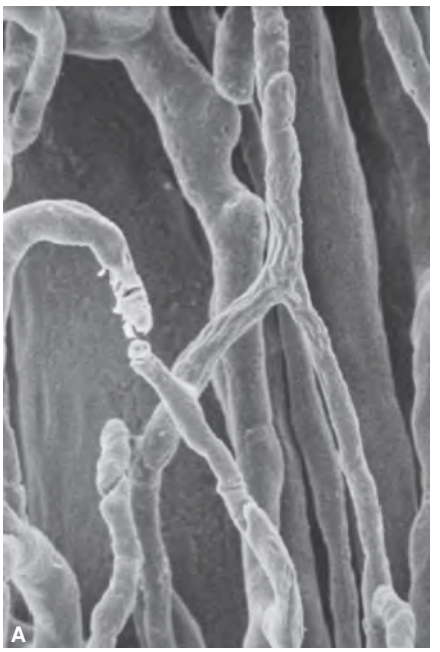


FIGURE 2-8 Vessel architecture in the pulp visualized by the India ink suction technique. Lateral canal vessels connecting the pulp and the PDL are observed.

Arteriovenous Anastomosis and "U"-Turn Loops

Before the arterioles break up into capillary beds, arteriovenous anastomosis (AVA) often arise to connect the arteriole directly to a venule.[68] The AVAs are relatively small vessels, having a diameter ~10 μm.[69] Their presence is more frequent in the radicular area of the pulp. The functional role of these vascular structures is not completely known. However, one can speculate that AVAs play a role in the regulation of blood flow. Theoretically, they could provide a mechanism for shunting blood away from the area of injury or inflammation, where damage to the microcirculation may result in thrombosis and hemorrhage. An interesting observation to support this view is during an intravital microscopic study, using the live rat incisor teeth. When the pulp is approached during tooth preparation, one sees a sudden appearance of AVA shunts and "U"-turn loops filled with streaming blood.[63] This might indicate that AVA shunts participate in temperature regulation within the pulp, as tooth preparation probably increases the pulp temperature and the AVA shunts will transport the heat out of the pulp. "U"-turn loops are frequently found in the pulp vascular network, and it is believed that their functions are similar to that of AVA's (Figures 2-9 and 2-10). Capillary density is highest in the subodontoblastic region with loops passing between odontoblasts (Figures 2-6, 2-10 and 2-11).[63,68,70] In the subodontoblastic region, capillaries with fenestrations occur frequently in both primary and permanent teeth.[68] However, the function of this fenestration is still disputed. Capillaries and the smallest postcapillary venules are the site of exchange between blood and the interstitial fluid. The terminal capillary network in the coronal area exhibits numerous short hairpin loops (Figures 2-6 and 2-7),[67] while the capillary network in



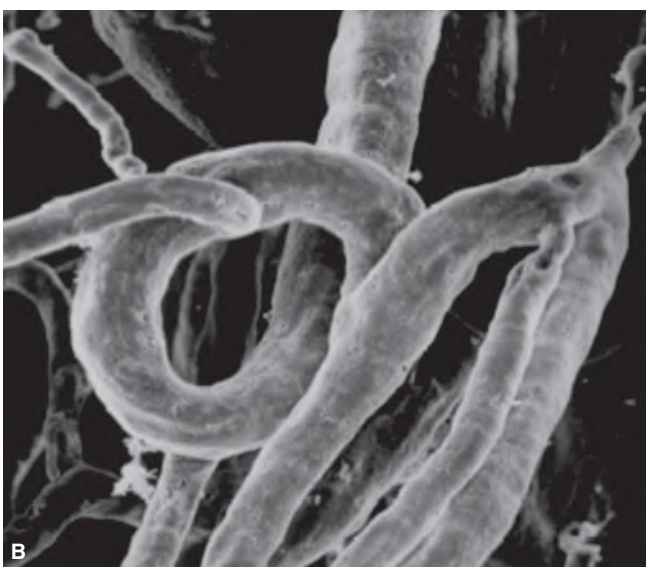


FIGURE 2-9 Corrosion resin casts of the AVA (A) and a "U"-turn loop (B). These unique features seem to play an important role in blood flow regulation, especially during the initial stages of inflammation. (Courtesy of Drs. Kazuto Takahashi and Yoshiaki Kishi, Kanagawa, Japan.)

the root area is similar to capillaries in the connective tissue elsewhere. Capillaries empty into small venules that connect with fewer and successively larger venules. At the apex, multiple venules exit the pulp. These venules connect with vessels that drain the PDL and adjacent alveolar bone. Vessels of the dental pulp have thinner muscular walls (tunica media) than vessels of comparable diameter in other parts of the body.[71] This is probably an adaptation to the surrounding protective and unyielding walls.

The vascular architecture can be divided into different vascular segments based on the morphology of periendothelial cells, such as smooth muscle cells and pericytes. These segments are muscular arterioles, terminal arterioles, precapillary arterioles, capillaries, postcapillary venules, and collecting or muscular venules. In most arterioles and in some venules, smooth muscle cells maintain a state of partial vasoconstriction at all times, and a variety of substances such as neurotransmitters, hormones, and local factors influence this muscle tone and, ultimately, the blood flow.[72] Nerve fibers have intimate association with the blood vessels, with arterioles being the most densely innervated. Compared with other connective tissues, the dental pulp has a high resting blood flow that is in the same range as blood flow in cerebral white matter, and four times as high as in resting skeletal muscles.[73] This is nearly as high as blood flow in the brain (Figure 2-12); however, the metabolic or functional requirements of this high resting blood flow in the pulp remains partly unexplained. Sometimes it is called a luxury perfusion. Figure 2-12 shows the comparison of blood flows among various tissues and organs, adjusted according to weight. Pulpal blood flow is intermediate between muscle and heart blood flow per unit weight.

Regulation of Pulpal Blood Flow

Pulpal blood flow is regulated by sympathetic α-adrenergic and neuropeptide Y (NPY) vasoconstriction,[74] β-adrenergic vasodilation,[75] sympathetic cholinergic vasoactive system, and an antidromic vasodilation, that is, a sensory axon reflex. A parasympathetic vasodilator mechanism is not shown in cat dental pulp.[76] The walls of arterioles and venules are coated with smooth muscles, innervated by unmyelinated sympathetic vasoconstrictor fibers as well as sensory fibers containing vasodilating neuropeptides substances such as calcitonin gene-related peptide (CGRP) and substance P (SP). When the sympathetic nerve fibers are stimulated, the muscle fibers contract, decreasing the diameter of the blood vessel and reducing blood flow. This phenomenon is called vasoconstriction. It has been shown experimentally that electrical stimulation of sympathetic fibers leading to the pulp results in a marked decrease in pulpal blood flow.[74,75] The sympathetic nerve-induced vasoconstriction of pulpal vessels is accomplished by the release of the α-adrenergic agonist noradrenalin, and NPY from sympathetic nerve endings. Activation of α-adrenergic receptors by the administration of an α-agonist norepinephrine results in a marked decrease in pulpal blood flow. This reduction in pulpal blood flow is blocked by pretreatment of the pulpal vessels with α-antagonist. These experiments clearly demonstrate that pulpal vessels are heavily equipped with sympathetic adrenergic receptors and their activation causes vasoconstriction. Activation of sensory nerves in the pulp cause increased blood flow, so-called vasodilation.

Blood circulation in an inflamed pulp involves very complex pathophysiological reactions that have not been fully elucidated, in spite of numerous studies.[77, 78]

A unique feature of the dental pulp is that it is rigidly encased within dentin. This places it in a low-compliant environment, much like the brain, bone marrow, and nail bed. Thus, the dental pulp tissue has limited ability to expand, so vasodilation and increased vascular permeability, evoked during an inflammatory reaction, result in an increase in pulpal hydrostatic

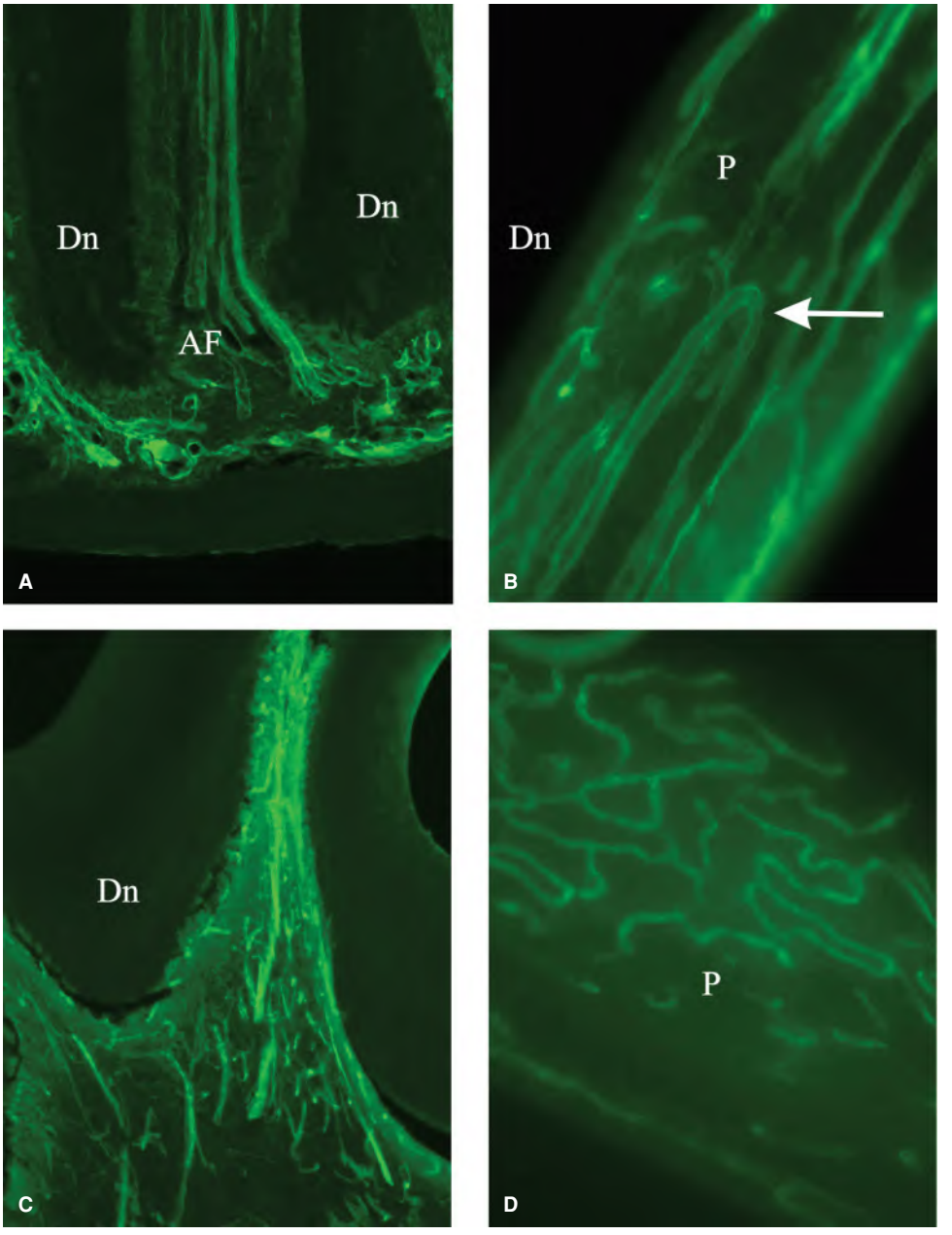


FIGURE 2-10 Immunohistochemical staining of dental pulp stained with fluorescence (green) antibody for laminin that stains mainly blood vessels. ***A.*** Large vessels enter the dental pulp via the apical foramen. ***B.*** Blood vessels in the root dental pulp showing U-turn loops (arrow). ***C.*** Numerous blood vessels in pulp horn region. ***D.*** Cross-section of the odontoblast layers showing capillary network. (AP: apical foramen, Dn: dentin, P: pulp). (Courtesy of Dr. Sivakami R. Haug, Bergen, Norway.)

pressure.[79,80] Presumably, any sudden increase in intrapulpal pressure would be distributed equally within the area of pressure increase, including the blood vessels. Theoretically, if tissue pressure increases to the point that it equals the blood pressure, the thin-walled venules would be compressed, thereby increasing the vascular resistance and reducing the pulpal blood flow.[72] This could explain why injection of vasodilators such as bradykinin into an artery leading to the pulp results in a reduction rather than an increase in pulpal blood flow.[72,75,79] However, Heyeraas[79] observed that an increase in intrapulpal tissue pressure promoted absorption of tissue fluid back into the blood and lymphatic vessels, thereby reducing the pressure. Thus, it appears that blood flow can increase, in spite of an elevation in tissue pressure as long as the permeability of the pulpal vessels is not increased. By normal vessel permeability, the increased pressure causes fluid absorption according to "the Starling forces" that lowers the tissue pressure.

Obviously, a combined multidisciplinary approach is needed to understand the intricate circulatory changes occurring during the development of pulpal inflammation. Using the laser Doppler technique to study pulpal blood flow in dogs, Sasano et al.[81] suggested that an increase or a decrease in pulpal blood flow is more dependent on systemic blood pressure than on local vasoconstriction or vasodilation.

FIGURE 2-11 Capillary loops passing under, or above, the odontoblastic layer. Capillaries that are not in focus represent their position in relation to the cells, either under or above them. Prepared by modified H&E technique. (Courtesy of Drs. Kazuto Takahashi and Yoshiaki Kishi, Kanagawa, Japan.)

The Stealing Theory

The dental pulp receives its blood supply through the so-called end arteries. These arterioles, entering the apical pulp, have a relatively low blood pressure. The pressure difference from the arterioles entering the pulp to the venules leaving is only about one-fourth of the total arteriovenous pressure difference (Figure 2-13).[80] Accordingly, a considerable part of the vascular resistance that regulates the pulpal blood circulation is located in the venules and also outside the pulp.[82] This implies that changes in circulation in the neighboring adjacent tissues, such as the gingiva, alveolar bone, and PDL, will change the blood flow to the pulp because it will affect the feeding pulpal arterial blood pressure. Any vasodilation in tissues that receive their blood supply through side branches of the end arterioles feeding the pulp will, according to the Poisseuille law, "steal" blood pressure from the pulp (Figure 2-14).[80] Therefore, it should be borne in mind that clinical treatment causing vasodilatation in adjacent tissues, may decrease the circulation to the dental pulp. Thus, the mere application of a matrix may affect the pulpal circulation and cause a fall in pulpal blood flow. The dental pulp may become ischemic.

Low-Compliance System Theory

As stated earlier, the dental pulp is encased in rigid structures, namely dentin, enamel, and cementum, creating a low-compliance system. In this system, any increase in blood flow or vasodilation has a limit, depending on the degree of increase in tissue pressure. In normal conditions, venular blood pressure is higher than the tissue pressure in the pulp. However, introduction of a known vasodilator, such as isoproterenol, causes an initial increase in pulpal blood flow that in turn causes a sudden increase in tissue pressure.[83] When the tissue pressure exceeds that of the venular pressure, a passive compression can cause a decrease in pulpal blood flow (Figure 2-15). Injection of various vasodilators caused a biphasic flow response, an increase followed by a decrease using the radioisotope microsphere injection technique.[84] In clinical situations, the effects of vasodilators produced by the initiation of inflammatory process on pulpal blood flow seem to be based on the low-compliance system theory. Thus, outpouring of inflammatory mediators, mostly vasodilators that

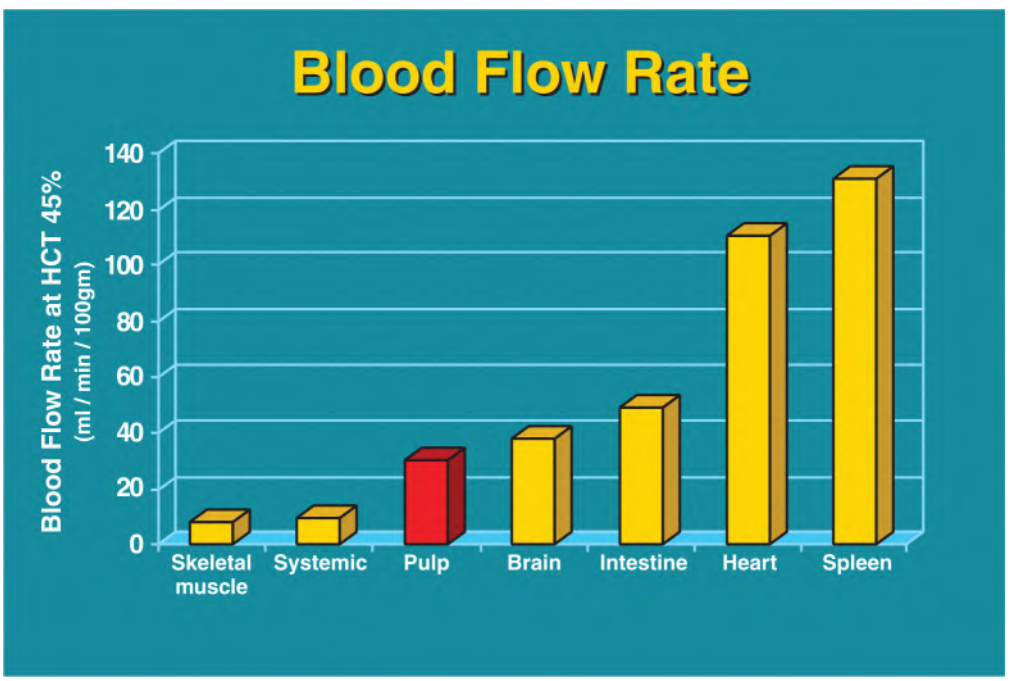


FIGURE 2-12 Blood flow rate in milliliters per minute per 100 g tissue of several important tissues and organs in a dog determined with a radioisotope-labeled microsphere technique. The pulpal blood flow is similar to that of the brain, but far less than in the major organs like the heart or the spleen. (Courtesy of Dr. Syngcuk Kim, Philadelphia, PA, U.S.A.)

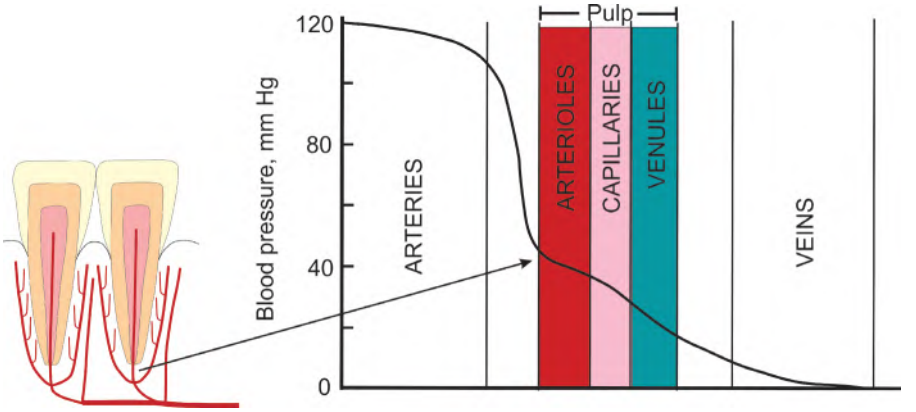


FIGURE 2-13 Fall in blood pressure along extra-pulpal and intra-pulpal blood vessels. Blood pressure in the small arteries entering the dental pulp is relatively low, about half of the aortic pressure. A considerable part of the vascular resistance that regulates pulpal blood flow is located in vessels outside the pulp (bone, gingiva, PDL). (Courtesy of Dr. Karin J. Heyeraas, Bergen, Norway.)

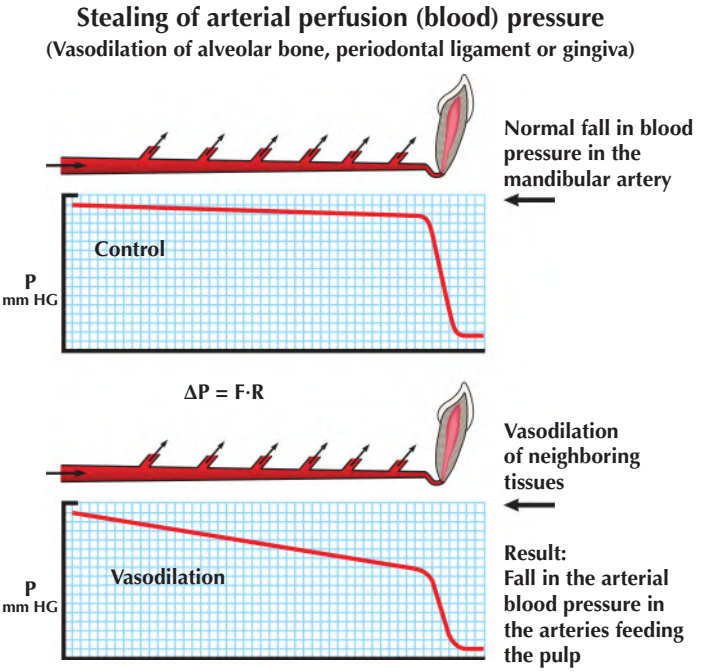


FIGURE 2-14 Stealing of arterial perfusion pressure during vasodilation in adjacent tissues. (P:blood pressure, F:blood flow, R:vascular resistance). (Courtesy of Dr. Karin J. Heyeraas, Bergen, Norway.)

also increases the vessel permeability, has grave consequences for pulpal survival due to passive compression of the low-pressure venules by a high tissue pressure (Figure 2-15).

Transcapillary Fluid Flow

At the same time as the exchange of substances occurs across capillaries, another completely distinct process also takes place, that is, the bulk flow of fluid across the capillary wall. The function of this bulk flow of fluid is not the exchange of nutrients and waste products, but the distribution of the extracellular fluid. This distribution is governed by the hydrostatic pressure difference and the colloid osmotic pressure difference across the capillary wall. These pressures determine the amount of the extracellular fluid located within the vessels as blood plasma and as interstitial fluid in the tissues. The difference in protein concentration between plasma and interstitial fluid is important because it creates a higher colloid osmotic pressure in plasma than in the interstitial fluid, thus favoring bulk fluid movement back into the capillary.

The hydrostatic pressure outside the vessels is normally considerably lower than the blood pressure inside the capillaries. Usually, this difference in hydrostatic pressure favors filtration, or bulk flow of fluid out of the vessels. However, in the low-compliant dental pulp, where a small increase in volume increases the tissue pressure, absorption may take place during vasodilation. In the low-compliant pulp, a nearly simultaneous increase in blood volume and tissue pressure, as a result of vasodilation and/or venous stasis,[78] has been

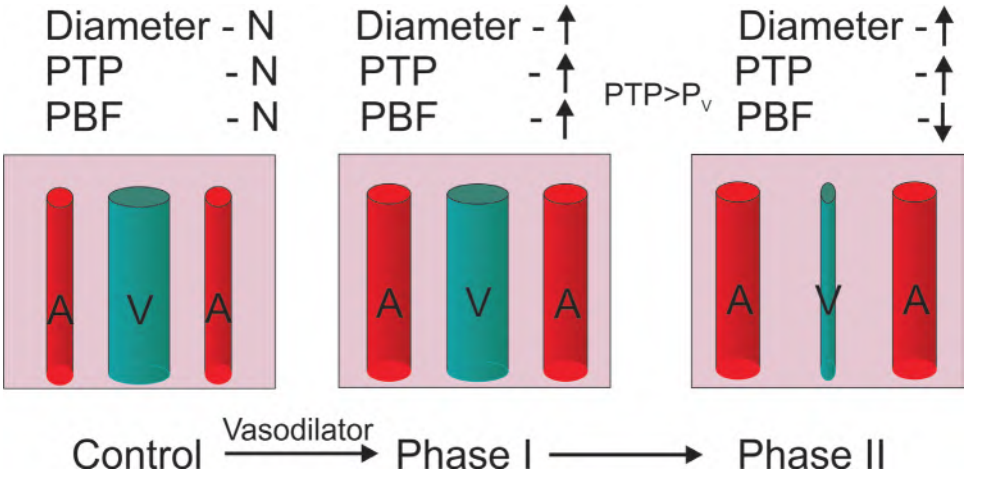


FIGURE 2-15 Diagram illustrating the low-compliance system during vasodilation (Phase I). The non-inflamed dental pulp has normal vessel permeability. Therefore, only a transient increased in pulpal tissue pressure (PTP) and fall in pulpal blood flow (PBF) will occur by vasodilation (Phase II). In non-inflamed pulp, the increased tissue pressure will cause absorption of interstitial fluid back into the vessels, and thus relatively quickly lower the tissue pressure and restore blood flow. On the contrary, in the inflamed dental pulp, the increased vessel permeability will prevent fluid absorption and a fall in PBF (Phase II) will prevail due to compression of veins. (Courtesy of Dr. Syngcuk Kim, Philadelphia, PA, U.S.A.)

recorded, showing that vascular distension with an increase in blood volume compresses the pulpal tissue and raises the tissue pressure. Thus, a rapidly responding hydrostatic counter pressure develops that lowers the transmural hydrostatic pressure difference. Consequently, capillary filtration is reduced and absorption occurs, and the tissue pressure is rapidly normalized, according to the Starling forces.[85]

The interstitial fluid in the pulp reaches out in dentinal tubules and forms a continuum that extends out to the dentinoenamel and cementoenamel junctions. The mere cutting of dentin, as it occurs during cavity and crown preparation, may, therefore, affect the dental pulp.[85] Furthermore, sensory nerve fibers are located in the subodontoblastic space and in ~50% of tubules in the crown. Research during the last decades has shown that sensory nerves have a strong impact on the blood circulation in the dental pulp, due to liberation of vasodilating neuropeptides. In the cat pulp, most of the sensory nerve fibers contain these neuropeptides. The majority of sensory nerve fibers containing the vasodilating neuropeptides in the main pulp seem to be located in the walls of the blood vessels, a finding that implicates a role for these neuropeptides in blood flow regulation.

Lymph Vessels

The only known mechanism for the removal of proteins and macromolecules that may leak out from the blood vessels in any tissue is the lymphatics. The existence of lymph vessels in the dental pulp is well established.[86, 87] Recovery of inflammation depends on the removal of macromolecules and plasma proteins from the tissue, thus a reversible pulpitis is dependent on functional lymph vessels.[88] Most lymphatic vessels are located in the root pulp, whereas in the coronal area, lymph vessels are observed in the more central part. Some studies conclude that in the coronal part, real lymph vessels are lacking and that lymph is collected in interstitial clefts and drains toward lymphatic vessels in the apical region of the pulp.[89] The lymphatics in the peripheral pulp zone join to form larger collecting vessels.[90] These vessels unite with progressively larger lymphatic vessels that pass through the apex together with the blood vessels.

Numerous authors, using both histological and functional methods, have described extensive anastomoses between lymph vessels of the dental pulp, the PDL, and alveolar bone.[91–94] The structural identification of lymph vessels[86, 91] complements the functional studies, demonstrating that substances placed in the pulp chamber can be found in regional lymph nodes. The open endothelial margins and incomplete basal lamina permit the entry of large molecules and even microorganisms into the lymphatics. The fact that materials placed in the dental pulp can migrate to lymph nodes[95] indicates the possibility of the spread of microorganisms or their products from the pulp.[96] The anastomoses of pulpal, periodontal, and alveolar lymphatics may be important routes for the spread of pulpal inflammation into adjacent tissues.

The architecture of the initial lymphatics, the so-called lymph capillaries, in different tissues varies considerably. Although in general they are wider than the blood capillaries, their number is much smaller. The wall of the initial lymphatics consists of a single layer of thin endothelial cells, often with overlapping margins. Open gaps between the cells, up to 2–5 μm, are frequent in many tissues. The initial lymphatics dilate and the number of open junctions increases during edema formation. It has been proposed that interstitial fluid pressure is the main determinant of lymph flow, and that lymph flow increases consistently with the tissue pressure.[97] In the low-compliant pulp, this would imply that the increased local tissue pressure, as measured during pulpitis, induces increased lymph flow that will normalize the pressure and also drain off microorganisms and their byproducts, promoting healing of the pulp.

An enhanced lymph flow during inflammation has also been suggested by Feiglin and Reade.[98] They deposited radioactive microspheres in rat pulps and found more microspheres in the submandibular lymph nodes in those rats whose pulps had been exposed for 5 days in comparison with those with acute pulp exposure.[98] The relationship of teeth to the cardiovascular and lymphatic systems is intimate and absolute. Clinicians should remember this when performing dental procedures, because placement of materials on dentin or the pulp may result in widespread distribution of that material or medicament.

Interstitial Fluid

The rigid encasement allows the tissue-limited possibilities to expand and keeps the extracellular fluid volume, that is, blood plasma and interstitial fluid, relatively constant. The extracellular fluid volume in the dental pulp is normally relatively high of ~63%.[99] Due to the low compliance, a small increase in the pulpal volume, caused by an increase in blood or interstitial fluid, will raise the hydrostatic pressure inside the tooth. This is shown to happen normally by any increase in blood flow and thus blood volume.[100] However, as long as there are no noxious stimuli that increase the vessel permeability, any change in tissue pressure caused by blood volume changes will be transitory, because interstitial fluid is absorbed back to the blood vessels, and no harm will happen to the pulp (Figure 2-15).[80]

The hydrostatic pressure in this fluid is the interstitial fluid pressure, or the so-called tissue pressure (Figure 2-16). The interstitial fluid is similar to blood plasma, except for a lower concentration of plasma proteins (albumin and globulin). In a healthy dental pulp, plasma proteins do not permeate through the capillary wall and the concentration in the interstitial fluid is normally low. However, a study in rat incisors, surprisingly high protein concentration in the pulp interstitial fluid during physiological conditions was reported.[99]

The main function of the interstitial fluid is to act as a transport medium for nutrients and waste products between cells and capillary blood. Accordingly, the interstitial fluid acts as a middleman between cells and blood, or as an extension of the plasma. Every cell must have nutrition plus the means to rid itself of waste products. These metabolic requirements are taken care by the blood and the interstitial fluid. Blood is brought to the tissues by the smallest blood vessels, the capillaries. Substances are transported between the blood and the interstitial fluid mainly by diffusion through the capillary wall (Figure 2-16). These capillaries are so widely distributed that no cells are >50–100 μm from the blood vessels. Due to this short distance, the exchange of substances between blood and interstitial fluid may take place rapidly by simple diffusion. Diffusion depends on the concentration differences. Accordingly, increased blood flow creates higher concentration difference between plasma and interstitial fluid and faster exchange of substances.

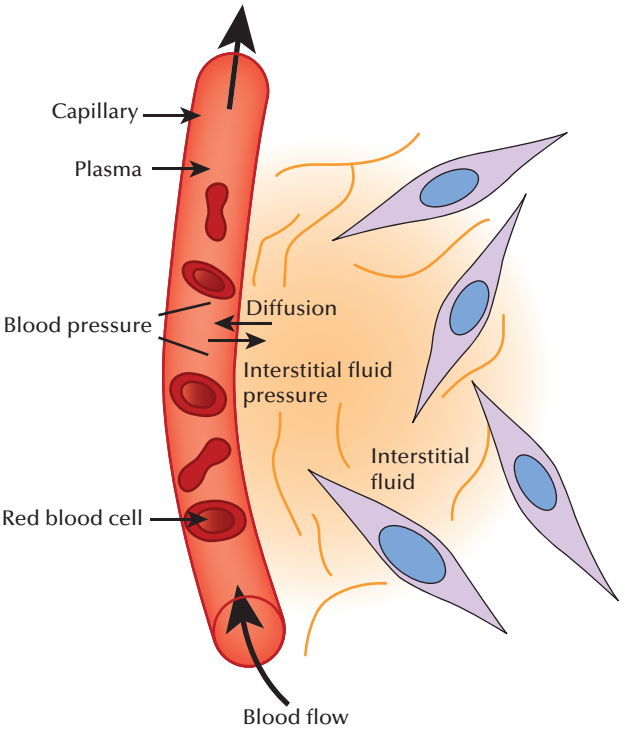


FIGURE 2-16 Schematic illustration of dental pulp tissue. The interstitial fluid is the fluid that surrounds cells and the hydrostatic pressure in this fluid is tissue pressure. (Courtesy of Dr. Karin J. Heyeraas, Bergen, Norway.)

Nerves in the Pulp

Classification of Nerves

The dental pulp has an abundant supply of both sensory and sympathetic nerves. The majority of these nerves are sensory. The trigeminal ganglion supplies sensory innervation to the pulp via the maxillary and mandibular nerves. Sympathetic nerves are less numerous with its source from the superior cervical ganglion. Postganglionic sympathetic nerves travel with the internal carotid nerve, reach the trigeminal ganglion, and supply teeth and supporting structures via the maxillary and the inferior alveolar nerve.[101–103] A small portion of sympathetic nerve fibers enter the dental pulp surrounding blood vessels. The proportion of sympathetic fibers in the dental pulp has been shown to be relatively small.[104, 105] Sympathetic fibers in fully developed teeth have been reported to make up no more than ~10% of the nerve fibers.[106] Parasympathetic fibers have been suggested to exist in the pulp.[107, 108] Anatomical and electrophysiological studies have classified nerves according to their diameter and conduction velocity (Table 2-1). The pulp contains two types of sensory nerve fibers: A fibers that are myelinated and C fibers that are unmyelinated. A fibers include both A-β and A-δ with A-δ amounting to ~90% of the total A fibers.[109] For more details, see Chapter 7.

Neuropeptides

Nerve fibers release biologically active peptides, known as neuropeptides that influence neural activity and

TABLE 2-1 Classification of Nerves in the Pulp

Type of Fiber	Function	Diameter (μm)	Conduction Velocity (m/sec)
A β	Pressure, Touch	5–12	30–70
A δ	Pain, Temperature, Touch	2–5	12–30
C	Pain	0.4–1.2	0.5–2
Sympathetic	Postganglionic sympathetic	0.3–1.3	0.7–2.3

functioning.[110–112] Neuropeptides are synthesized on ribosomes in the neuron cell body, processed through the endoplasmic reticulum and Golgi complex, transported in vesicles by axoplasmatic flow to the nerve terminals (Figure 2-17), and released from the peripheral terminals of mainly A-δ and C fibers.[113]

There are numerous neuropeptides in the dental pulp that are commonly classified as sensory, sympathetic, or parasympathetic neuropeptides according to the origin of nerve fibers. Sensory neuropeptides reported in the dental pulp are CGRP, SP, and neurokinin A (NKA). CGRP, a 37-amino acid peptide, is a member of the calcitonin family of peptides. It is synthesized in the trigeminal ganglion and transported to the dental pulp via maxillary and mandibular branches. CGRP is a potent vasodilator and can function in the transmission of pain.[114,115] In the dental pulp, CGRP seems to be mainly responsible for the increase in blood flow and interstitial fluid pressure during tooth stimulation.[116] SP belongs to the tachykinin neuropeptide family, a 11 amino acid peptide, closely related to NKA. Similar to CGRP, SP is also synthesized in the trigeminal ganglion and often co-localized with CGRP. The SP is found in small-diameter, predominantly unmyelinated sensory axons.[117,118] It is a vasodilator, increases vessel permeability and other aspects of inflammatory process, and is important in pain perception. The CGRP with SP has a synergistic effect on edema formation during inflammatory reactions.[119] When injected in the blood stream in experimental studies, CGRP, SP, and NKA produce vasodilation in the dental pulp.[120] Activation of nerve fibers by electrical stimulation, or electrical impulses on the tooth, produces long lasting vasodilation in the pulp.[77,121,122] Sensory denervation performed by axotomy of the inferior alveolar nerve decreases pulpal blood flow and interstitial fluid pressure indicating a basal release of vasoactive sensory neuropeptides.[116]

NPY, a neuropeptide released from sympathetic nerve terminals, is a 36 amino acid peptide that acts as a neurotransmitter and neuromodulator.[123,124] It is synthesized in the sympathetic ganglion and co-released with norepinephrine via anterograde axonal transport from the peripheral terminals.[125–127] It has been reported that NPY can also be found in sensory neurons and nerves when there has been neuronal injury such as axotomy.[128,129] Release of NPY causes vasoconstriction.[126] NPY has also been reported to be a modulator of the immune function in the dental pulp and surrounding structures.[130] Vasoactive intestinal peptide (VIP), a peptide containing 28 amino acid peptide, has been shown to be released from parasympathetic nerves.[131]

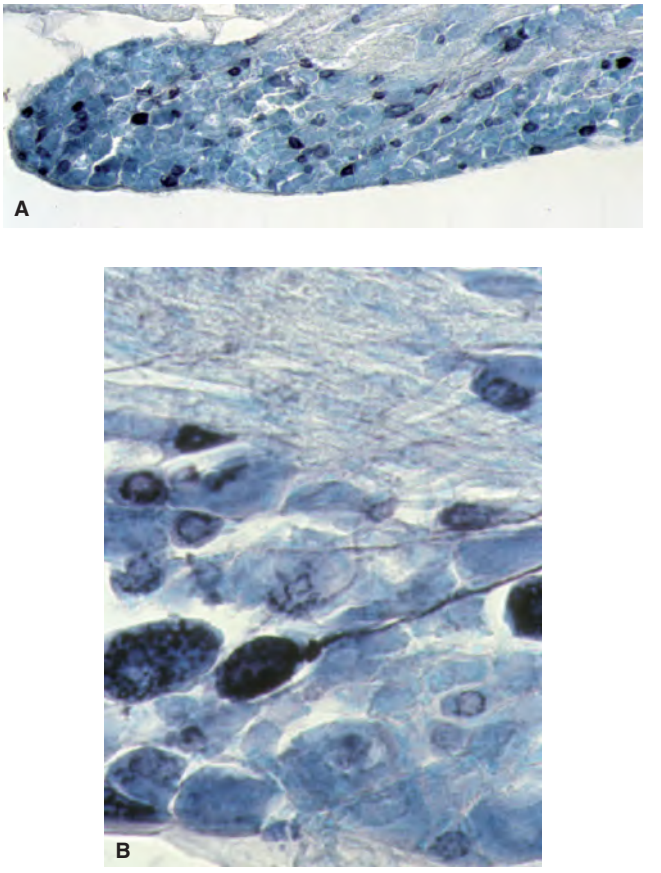


FIGURE 2-17 Immunohistochemical staining of the trigeminal ganglion. Calcitonin gene-related peptide-immunoreactive (CGRP-IR) neurons in the trigeminal ganglion. ***A.*** Low-magnification view of darkly stained neurons that contain the neuropeptide CGRP. ***B.*** Higher magnification view of neurons with axons transporting CGRP to the peripheral nerve terminals. Neurons display different sizes; small, medium, and large. Note, some neurons are darkly stained and some lightly stained. (Courtesy of Dr. Sivakami R. Haug, Bergen, Norway.)

Distribution

Immunohistochemical staining methods have made it possible to identify the location and distribution of peptidergic nerves in the dental pulp. The figures from immunohistochemistry, shown in this chapter, are from experimental animals unless otherwise stated. Due to the staining procedures, animals are first perfused to remove red blood cells and then post-fixed, decalcified, sectioned, and stained with specific antigen-antibody (immunohistochemical) reactions.

Sensory nerves fibers containing CGRP or CGRP-immunoreactive (-IR) nerve fibers (Figure 2-18) enter the

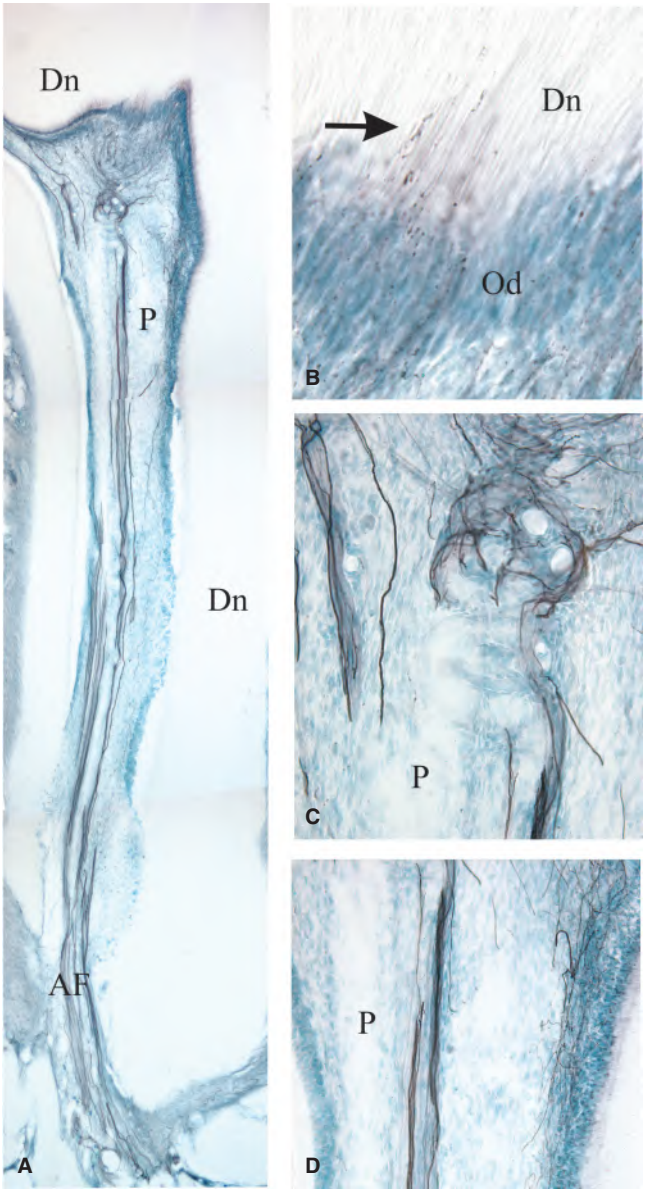


FIGURE 2-18 Immunohistochemical staining of calcitonin gene-related peptide-immunoreactive (CGRP-IR) nerve fibers in the rat dental pulp. ***A.*** Low magnification view of darkly stained nerve fibers entering the apical foramen and traversing the long axis of the tooth as thick bundles. Coronally, nerve fibers branch extensively, penetrate the odontoblastic layer and innervate the inner part of dentin. ***B.*** Thin CGRP-IR nerve fibers penetrate the cuspal odontoblast layer and enter dentin (arrow). ***C.*** In the mid-coronal region, thick bundles of fibers are seen surrounding the blood vessels. ***D.*** Thick band of fibers surrounding the blood vessels are in the root pulp. (AF: apical foramen, Dn: dentin, Od: odontoblast layer, P: pulp). (Courtesy of Dr. Sivakami R. Haug, Bergen, Norway.)

pulp via the apical foramen in bundles either surrounding the blood vessels or individually, and ramify into a network of fine fibers in the coronal pulp.[132–134] Coronally, individual fibers penetrate the odontoblast layer and predentin to terminate in the inner 100 μm of the dentinal tubules. Nerve fibers have been found in >50% of tubules in inner cuspal dentin.[53,109,135,136] Radicular pulp fibers run parallel to the long axis of the tooth with little arborization and limited penetration of the odontoblast layer.

CGRP is the most abundant neuropeptide in the dental pulp (Figure 2-18). It is three to four times more abundant than SP.[132] SP is commonly found in C fibers with distribution patterns similar to CGRP-IR nerve fibers.[117,118,132] NKA-IR nerve fibers have distribution pattern similar to SP-IR nerve fibers.[137] CGRP, SP, and NKA neuropeptides have been shown to coexist in the same nerve fibers in the dental pulp. Neuropeptide containing nerve fibers are found in close contact with immune cells suggesting important neuroimmune interactions (Figure 2-19).

Sympathetic nerve fibers containing NPY or NPY-IR fibers are thin with varicosities. In a normal healthy dental pulp, thin varicose fibers enter the dental pulp via the apical foramen in close approximation to blood vessels. In the root pulp, NPY containing fibers exhibit a network arrangement surrounding blood vessels. These NPY containing fibers terminate in the floor or the mid-coronal part of the pulp chamber (Figure 2-20). They do not extend to the pulp horn region nor penetrate the odontoblast layer. NPY containing fibers are more in number in the radicular than in the coronal pulp.[108,132,138–140] VIP-IR nerve fibers in the dental pulp are associated with blood vessels,[141] or occur as free and interlacing nerves in the central pulp and the subodontoblastic plexus.[141,142]

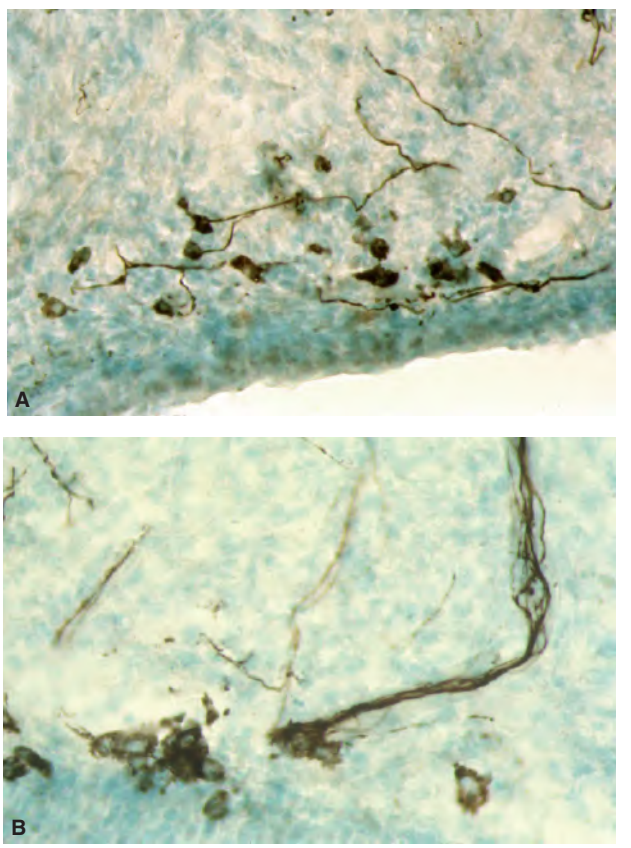


FIGURE 2-19 Immunohistochemical staining of the dental pulp for calcitonin gene-related peptide (CGRP) showing CGRP-immunoreactive nerve fibers in close proximity to darkly stained immune cells on the floor of the pulp chamber. (Courtesy of Drs. Sivakami R. Haug and Karin J. Heyeraas, Bergen, Norway.)

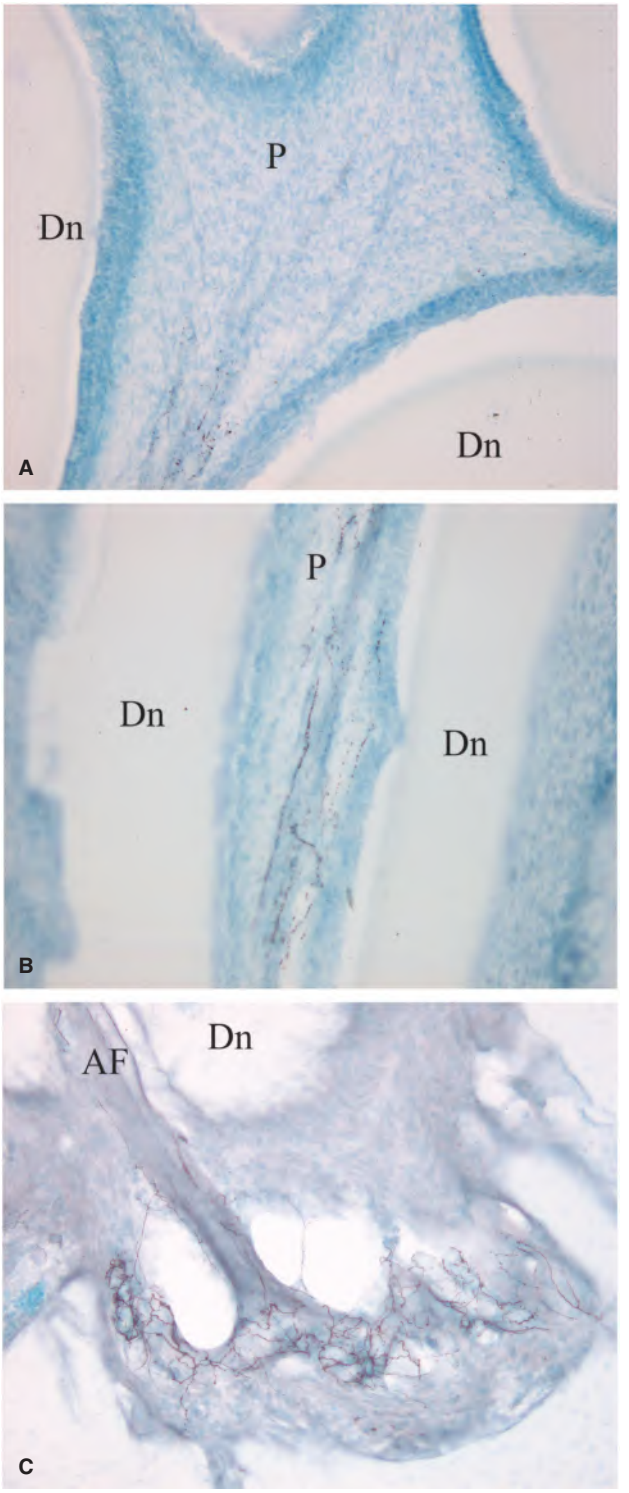


FIGURE 2-20 Immunohistochemical staining of neuropeptide Y-immunoreactive (NPY-IR) nerve fibers in the rat dental pulp. ***A.*** Thin, varicose, darkly stained NPY-IR nerve fibers in the coronal pulp. These fibers do not enter the pulp horn region nor penetrate the odontoblast layer. They are located on the floor of the pulp chamber where larger blood vessels are located. ***B.*** NPY-IR fibers in the root pulp showing a network arrangement around blood vessels. ***C.*** Apical foramen with NPY-IR fibers entering the dental pulp as thin, varicose fibers closely associated with blood vessels. (AF: apical foramen, Dn: dentin, P: pulp). (Courtesy of Dr. Sivakami R. Haug, Bergen, Norway.)

Neurogenic Inflammation

Sensory neuropeptides have been shown to cause neurogenic inflammation. A classical demonstration of neuropeptide activity is the flare component of the "triple response" in the human skin: wheal (edema formation), local reddening (increased blood flow), and flare.[143] The flare component of the triple reflex is a consequence of the sensory nerve-mediated axon reflex release of SP and CGRP causing increase in blood flow (vasodilation) and vessel permeability.

During inflammation, neuropeptide containing nerve fibers exhibit changes in number, density, and architecture. This observation was first made by Byers et al. where sensory nerves fibers containing neuropeptides CGRP and SP showed sprouting during pulpal injury.[144–147] In experimental pulp exposure studies, sprouting occurs as early as one day after pulp exposure and stays high until healing or pulp necrosis occurs.[133,146] Nerve fibers also change in quantity and architecture when inflamed (Figure 2-21). Sympathetic fibers containing NPY, unlike sensory nerve fibers, do not sprout immediately after injury in experimental studies but a few weeks later[139,148,149] (Figure 2-22). In clinical situations, neuropeptide levels are increased in dental pulp diagnosed as irreversible pulpitis.[150,151]

External root resorption originating from the periodontium, however, does not display any nerve sprouting in the dental pulp or PDL, unlike caries and experimental pulp exposure.[152] A complete root resportion from the periodontium to the dental pulp creates very little damage to the dental pulp (Figure 2-23). This has been concluded to be mainly due to the lack of infection when a root resorption originates from the periodontium.[152]

Sectioning of the inferior alveolar nerve removes almost all sensory neuropeptide containing nerves in the dental pulp.[153] Removal of the superior cervical ganglion, eliminates almost all NPY containing nerve fibers in the dental pulp.[154] Electrical stimulation of teeth causes release of neuropeptides, creating a local neurogenic inflammation such as increased blood flow, transendothelial migration of inflammatory cells. When nerve fibers have been removed from the pulp, via axotomy of inferior alveolar nerve, no recruitment of immune cells was observed, thus confirming the importance of nerve fibers for neurogenic inflammation.[155,156] When sympathectomy was performed, granulocytes were not recruited after electrical stimulation.[157] It has been reported that while sensory neuropeptides have a pro-inflammatory role in the dental pulp, sympathetic neuropeptide, NPY, may have an anti-inflammatory role on the immune system.[130,150,157–159]

Sensory neuropeptides increase vascular permeability during inflammation, and stimulate the production of other inflammatory cytokines such as interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNFα) (Figure 2-24). Sympathetic nerves inhibit the production of proinflammatory cytokines, while stimulating the production of anti-inflammatory cytokines.[130,160] Teeth lacking sympathetic nerves also tend to undergo

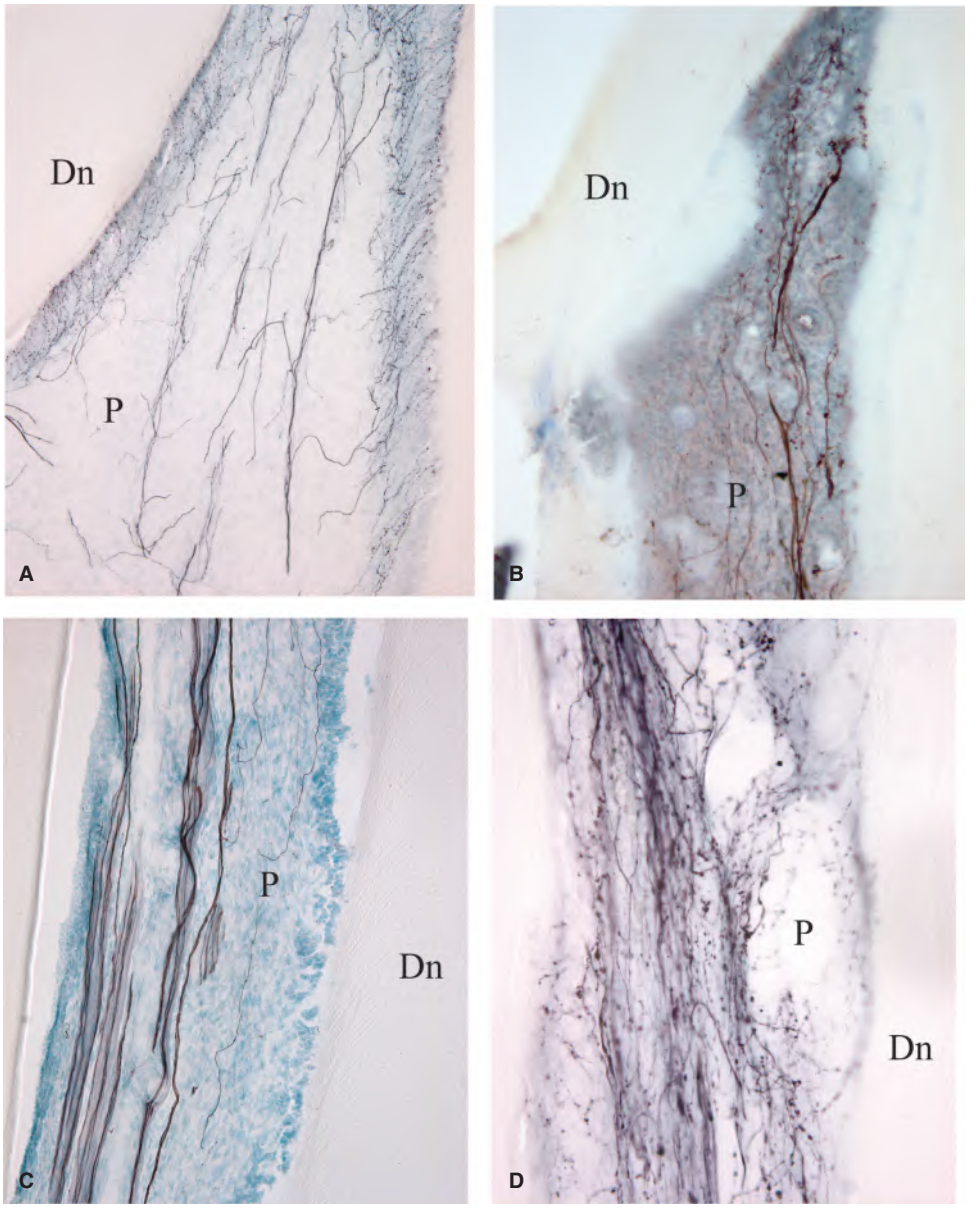


FIGURE 2-21 Calcitonin gene-related peptide-immunoreactive (CGRP-IR) nerve fibers in the normal dental pulp (A and C) and inflamed dental pulp (B and D). ***A & C.*** Normal architecture of nerve fibers in the root, coronal and pulp horn region. ***B & D.*** An inflamed dental pulp after 20-day pulp exposure injury shows extensive spouting of CGRP-IR nerve fibers. Nerve fibers containing CGRP neuropeptide are numerous, and with abnormal architecture. (Dn: dentin, P: pulp). (Courtesy of Dr. Sivakami R. Haug, Bergen, Norway.)

necrosis more frequently indicating the anti-inflammatory role of the sympathetic nervous system.[138]

Sympathetic nerves have been shown to be important in the recruitment of granulocytes during electrical stimulation of teeth and experimental tooth movement.[157,161] Sympathetic imbalance results in the recruitment of plasma cells in an intact tooth, suggesting an immune dysfunction.[162] Sympathetic nerves have also been shown to have an inhibitory effect on osteoclast resorption activity and on the cytokine IL-1α.[139,160,161] NPY, co-released in the sympathetic nerve terminal, has recently been reported to modulate immune function in a wide range of inflammatory conditions.[130]

Receptors

Neuropeptides cannot cross the cell membrane and, therefore, their action is dependent on the existence of specific receptors (Table 2-2). Without the existence of a particular receptor, release of neurotransmitters, or neuropeptides, will have no effect. Therefore, identifying the location and distribution of these receptors is important for understanding the functionality of a neuropeptide in a given location. NK1 receptors, specific for SP, are present in the blood vessels in the root pulp and smaller vessels along the odontoblast layer.[163,164] Fibroblasts express NK1 receptors during mechanical orthodontic stress.[161] NPY receptors are located in blood vessels, nerve fibers, fibroblasts, and immune cells.[165,166] Antagonists

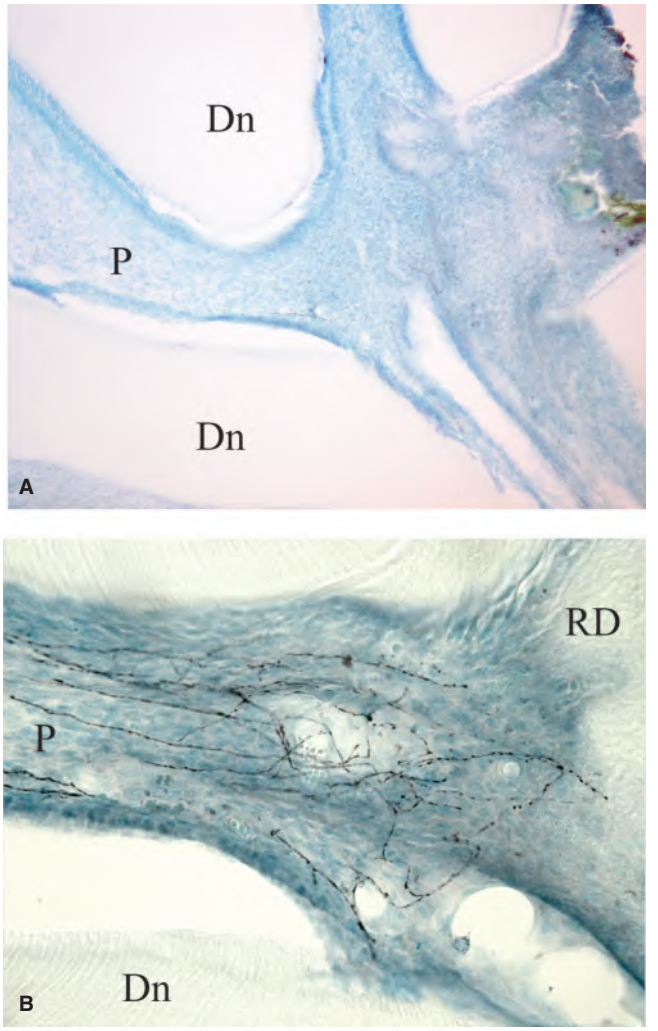


FIGURE 2-22 Neuropeptide Y-immunoreactive (NPY-IR) fibers in the inflamed dental pulp. ***A.*** After 6 days of pulp exposure injury, molar pulp shows no sprouting of nerve fibers. ***B.*** After 20 days of pulp exposure, there is extensive reparative dentin formation and increased number of NPY-IR nerve fibers penetrating the region of the reparative dentin.[140] (Dn: dentin, P: pulp, RD: reparative dentin). (Courtesy of Drs. Sivakami R. Haug and Karin J. Heyeraas, Bergen, Norway.)

to these receptors are now being widely studied as therapeutic agents against inflammatory diseases, or diseases involving overexpression of neuropeptides.

Extracellular Matrix

Extracellular matrix (ECM), or ground substance, is a structureless mass making up the bulk of the pulp tissue. It consists primarily of complexes of proteins, carbohydrates, and water. These complexes are composed of combinations of glycosaminoglycans, namely hyaluronic acid, chondroitin sulfate, and other glycoproteins.

Proteoglycans are a large group of extracellular and cell surface-associated molecules that consist of a protein core, to which glycosaminoglycan chains are attached, which binds them to water. Hyaluronic acid, a large glycosaminoglycan, forms a viscous hydrated gel. Proteins, glycoproteins, and

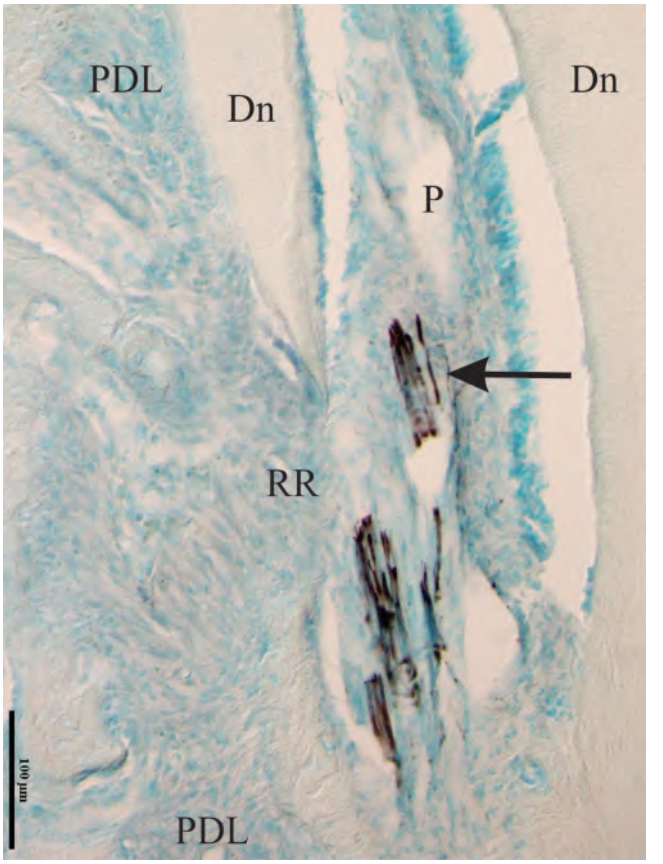


FIGURE 2-23 Immunohistochemical staining of PGP 9.5 immunoreactive nerve fibers in a tooth with experimental root resorption. Arrows pointing towards region of root resorption (RR) where whole dentin is resorbed. There is no sprouting of nerve fibers in the dental pulp or PDL.[153] (Dn: dentin, P: pulp, PDL: periodontal ligament). (Courtesy of Dr. Sivakami R. Haug, Bergen, Norway.)

proteoglycans of the ECM function in cell–matrix adhesion and signaling; regulate diffusion of nutrients, waste products, and soluble signaling molecules.

Cytokines, growth factors, and other inflammatory mediators are released by fibroblasts, endothelial cells, and immune cells in the dental pulp. Normal dental pulp contains cytokines such as IL-1α and TNFα[160]; however, during inflammation, orthodontic tooth movement, and other stimulation, IL-1α, IL-1β, IL-8, TNFα, prostaglandin E2, platelet-derived growth factor, insulin-like growth factor-1, transforming growth factor-b (TGFb1), vascular endothelial growth factor, basic fibroblast growth factor (FGF-2), hepatocyte growth factor, and keratinocyte growth factor levels are increased.[167–173] These molecules mainly act locally and play important roles during development, inflammation, and wound healing.

Fibers

Collagen is the major organic component in the dental pulp. The morphology of collagen fibers, a principal constituent in the pulp, has been described at the level of both light and electron microscopy.[174] Collagen is synthesized and secreted

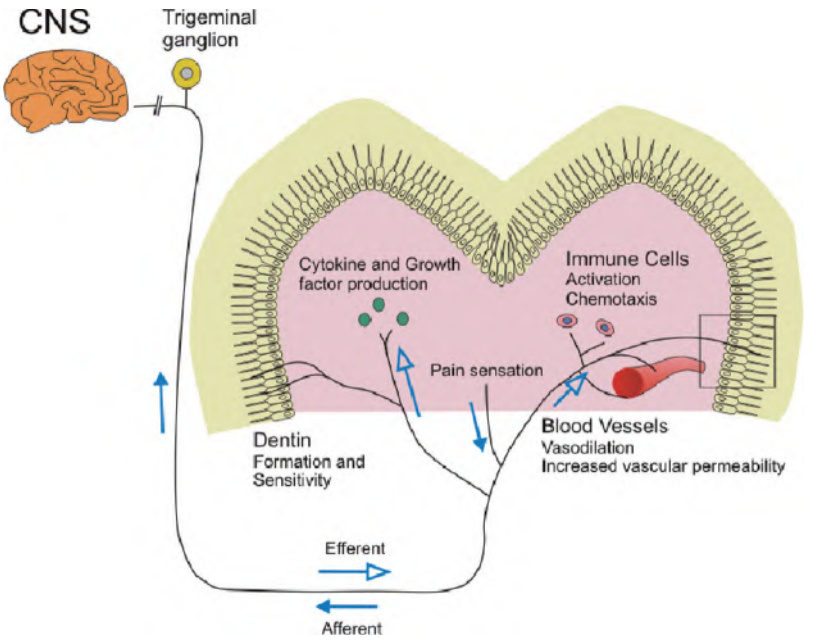


FIGURE 2-24 Schematic illustration summarizing some of the effects of neuropeptides (calcitonin gene-related protein, CGRP; substance P, SP) released from sensory nerves. Sensory nerves conduct afferent sensory information (e.g., pain sensations/dentin sensitivity) to the central nervous system. In addition, they have efferent functions, such as vasodilation and increased vessel permeability, due to liberation of neuropeptides at the nerve endings. It also affects various aspects of immune function such as activation and chemotaxis of immune cells, and release of cytokine and growth factors from various cellular structures in the dental pulp. Sensory nerves have also been shown to be involved in the formation of dentin. (Courtesy of Dr. Sivakami R. Haug, Bergen, Norway.)

TABLE 2-2 Neuropeptides and Their Origins Including Some of Their Main Actions and Specific Receptors

Neuropeptides	Released from	Actions	Receptors
Substance P	Sensory	Vasodilation, Increase vascular permeability	NK1
Neurokinin A	Sensory	Increase vascular permeability	NK2
Calcitonin Gene-Related Peptide	Sensory	Vasodilation	CGRP1 and CGRP2
Neuropeptide Y	Sympathetic	Vasoconstriction, pain modulation, immune function	NPY Y1-6
Vasoactive Intestinal Peptide	Parasympathetic	Vasodilation, immune function	VIP 1 & 2

by both odontoblasts and fibroblasts. However, the type of collagen secreted by odontoblasts to subsequently mineralize differs from the collagen produced by pulpal fibroblasts that normally does not calcify. They also differ not only in the basic structure but also in the degree of cross-linking and in slight variation in hydroxylysine content.

Tropocollagen is an immature collagen fiber that remains thin and stains black with silver nitrate, described as argyrophilic or reticular fibrils under light microscopy. If tropocollagen molecules aggregate into larger fibers, they no longer stain with silver and are then termed "collagen fibers." If several collagen fibers aggregate (cross-link) and grow denser, they are termed "collagen bundles." Collagen generally becomes more coarse (i.e., develops more bundles) as the patient ages. Collagen fibers are inelastic but have great tensile strength, giving its consistency and strength to the tissue.

Collagen type I and III make up the bulk of the tissue collagen. Collagen type IV is found in basement membranes. In the pulp, type III collagen appears as fine-branched filaments, whose distribution is similar to reticular fibers. Fibronectin is insoluble and the fibrils form part of the ECM. It acts as a mediator for cell–cell and cell–matrix adhesion. In the odontoblast layer, fibronectin is the fibrous structure between odontoblasts and probably corresponds to von Korff fibers, as they appear as corkscrew fibers passing from the pulp into predentin parallel to the long axis of the odontoblasts.[175] In the dental pulp, fibronectin forms a reticular network of fibrils, with an increased encasement around blood vessels. Integrins are transmembrane receptors that link intracellular actin network with ECM.

Cellular Structures

Odontoblasts

Odontoblast, the principal cell of the dentin-forming layer, is the first cell type encountered as the pulp is approached from the dentin (Figures 2-25, 2-26, and 2-27). These cells arise from peripheral mesenchymal cells of the dental papilla during tooth development and differentiate by acquiring the characteristic morphology of glycoprotein synthesis and secretion.[176] Glycoprotein forms the predentin matrix that is rendered mineralizable by the odontoblast, a unique

FIGURE 2-25 Pseudostratified appearance of odontoblasts. Dark horizontal line (arrow) delineates the cell body from the odontoblastic processes and predentin (once termed "pulpodentinal membrane"). Ultrastructural examination has shown this to be a terminal cell web that forms a support and attachment area between adjoining cells. (Reproduced with permission from Walton R, Leonard L, Sharawy M, Gangarosa L. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1979;48:545-7.)

cell producing a unique tissue, dentin. Synthesis and secretory activities render the odontoblast highly polarized, with synthesis occurring in the cell body and secretion from the odontoblastic process. The cell body contains organelles that represent different stages of secretion of collagen, glycoproteins, and calcium salts.[177] Matrix secretion precedes mineralization, with these two events separated in time and space by the predentin. The initial mineral seeding of predentin at the dentinoenamel junction is by the formation of "matrix vesicles, similar to bone mineralization."[178,179] Classic studies by Weinstock and Leblond,[180,181] using an autoradiographic technique, have demonstrated the functional sequence of matrix production and secretion.

In histological sections viewed under a light microscope, odontoblasts appear to vary from tall, pseudostratified columnar cells in the coronal pulp, to a single row of cuboidal cells in the radicular pulp to a flattened, almost squamous

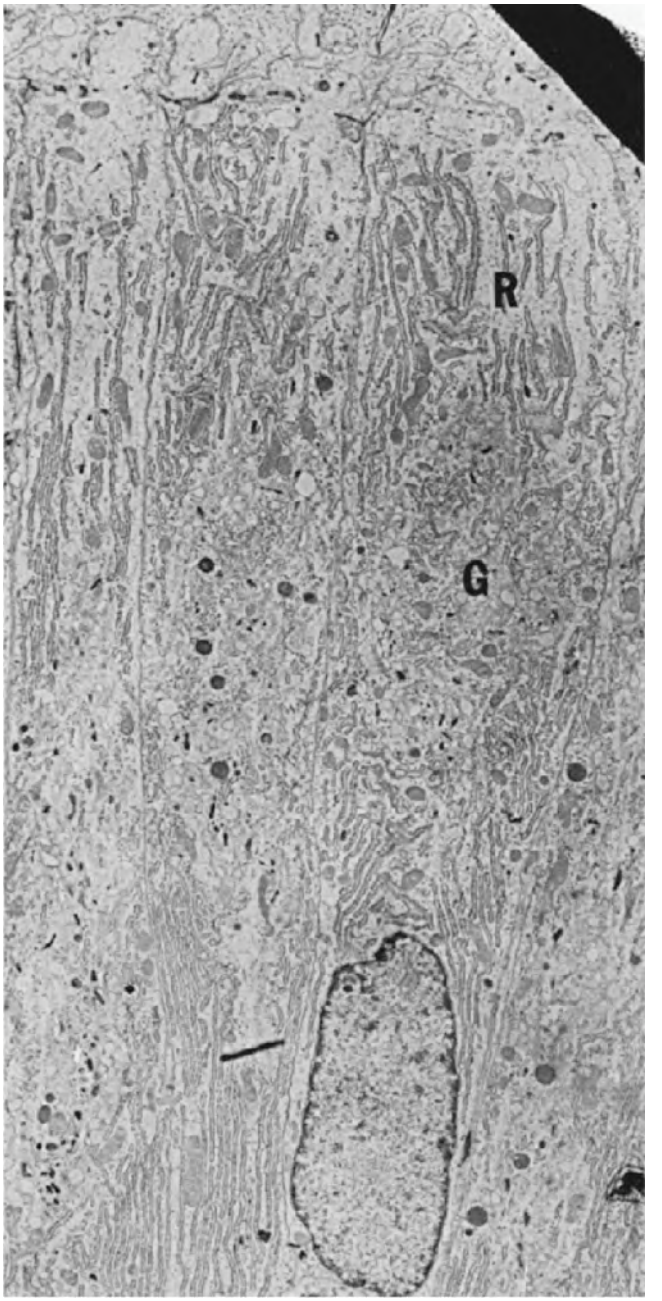


FIGURE 2-26 Odontoblasts, as viewed under the electron microscope, show organelles essential for protein synthesis; a plump nucleus filled with euchromatin, cytoplasm rich in rough endoplasmic reticulum (R), and a well-developed Golgi apparatus (G). (Courtesy of Dr. Dale R. Eisenmann, Chicago, IL, U.S.A.)

shape near the apex.[182,183] These squamous-shaped cells often form an irregular, atubular dentin. The large nucleus is located in the base of the cell, giving it a pear-shaped appearance.[184] From an exquisite scanning electron microscopic study, investigators have demonstrated that odontoblast cell bodies "appear tightly packed in the pulp horn and successively pear-shaped, spindle-shaped, club-shaped, or globular from the crown to the apex."[61]

During dentin formation in the crown, the odontoblasts are pushed inward to form the periphery of a pulp chamber,

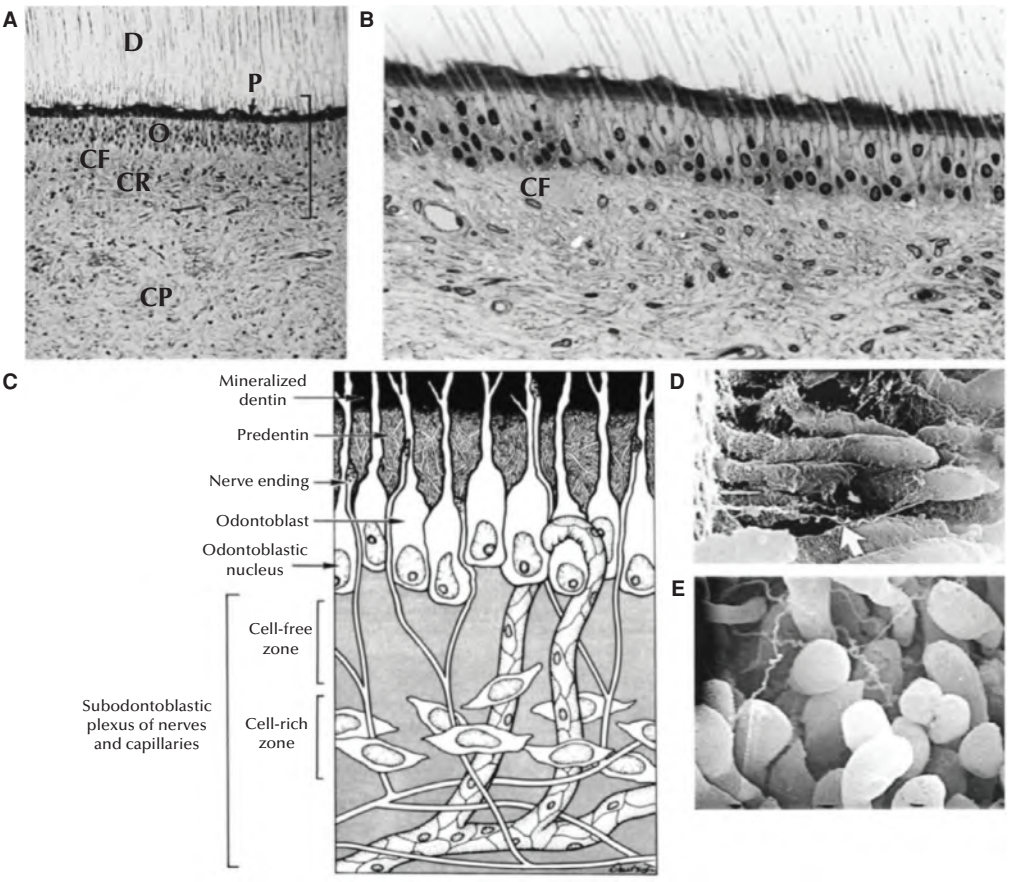


FIGURE 2-27 ***A.*** Medium-power photomicrograph of a human pulp specimen showing dentin (D), predentin (P), odontoblast layer (O), cell-free zone (CF), cell-rich zone (CR), and central pulp (CP). ***B.*** Region similar to the area bracketed in A. CF zone contains large numbers of small nerves and capillaries, not visible at this magnification. Underlying CR does not have high concentration of cells but contains more cells than the central pulp. (Courtesy of Drs. Dennis F. Weber and Michael K. Gaynor, Chicago, IL, U.S.A.) ***C.*** Diagram of peripheral pulp and its principal elements. ***D.*** Scanning electron micrograph (SEM) of the dentin–pulp junction. Note corkscrew fibers between odontoblasts (arrow). Reproduced with permission from Jean A, Kerebel JB, Kerebel LM. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1986;61:392-8. ***E.*** SEM of the pulpal surface of the odontoblast layer. Thread-like structures are probably terminal raveling of nerves. (Courtesy of Drs. Robert R. White and Melvin Goldman, Boston, MA, U.S.A.)

a circumference that is increasingly smaller than the original circumference at the dentinoenamel junction. This explains why the cells are packed and palisaded into a pseudostratified appearance of coronal odontoblasts. Conversely, because the space is not so compressed in the radicular pulp, the odontoblasts maintain a columnar, cuboidal, or (in the apical region) squamous shape. Also, the resulting cell and tubule density are much higher in the pulp chamber than in the root pulp.[185] This increased tubule density in the chamber may explain the greater sensitivity and permeability of the dentin of the crown.

The cell body manufactures the matrix material; the material is transported to and secreted from the odontoblastic process. Classically, the odontoblastic process has been described as extending from the cell body to the dentinoenamel junction, a distance of 2–3 mm. This concept was based on the observations of many light microscopists using a variety of special procedures and stains.[41] When dentin was examined by electron microscopy, the odontoblastic process was determined to be limited to the inner third of dentin, with the outer two thirds of the tubule devoid of processes or of nerves, but filled with extracellular fluid.[59,186–188] Some investigations indicate that odontoblastic processes may indeed extend to the dentinoenamel junction.[189] However, tubular structures in dentinal tubules are not necessarily odontoblastic processes.[190,191] Unequivocal identification can be done only by identifying a trilaminar plasma membrane around the putative process, using transmission electron microscopy.[192,193] The extent of the odontoblastic process remains controversial.[62] Therefore, modern interpretations of pulpal injury following conservative cavity preparation may not be attributable to amputation of odontoblastic processes but to desiccation, heat, and osmotic effects. Furthermore, dentin sensitivity may not be related to direct stimulation of odontoblastic processes or nerves in the peripheral dentin since the tubules may be devoid of such structures in the periphery of dentin.

After initial dentin formation, the odontoblast, via its process, can still modify the dentin structure by producing

peritubular dentin. This is a hypermineralized cuff with little organic matrix within the tubule, decreasing the diameter of the tubule.[194–196] When stimulated, the odontoblast can accelerate peritubular dentin formation to the point of complete occlusion of the tubule.[197–199] When tubule occlusions extend over a large area, it is referred to as sclerotic dentin, commonly found in teeth with cervical erosion. Alternatively, irritated odontoblasts can secrete collagen, amorphous material, or large crystals into the tubule lumen; these occlusions decrease dentin permeability to irritating substances.[197–199] Although these secretions have been described as a defensive reaction by the odontoblast to protect itself and the underlying pulp, this "protection" has never been proved.

Fibroblast

Fibroblasts originate from the mesenchymal tissue and are the predominant cells of the connective tissue. It is the most abundant cell in the dental pulp. Fibroblasts are elongated with little cytoplasm and nucleus-containing condensed chromatin. They are responsible for the formation and maintenance of the fibrous components and ground substances of the connective tissue. Fibroblasts can synthesize and secrete a wide variety of extracellular molecules that include fibrous elements of ECM such as collagen, elastin, proteoglycans, glycoproteins, cytokines, growth factors, and proteinases. They are also involved in the remodeling of connective tissue through the degradation of collagen and other ECM molecules and their replacement by newly synthesized molecules.

Fibroblasts release proteolytic enzymes such as the matrix metalloproteinase (MMP) family. MMPs are secreted as inactive precursors and cleave to become active. Extracellular degradation often occurs in inflammatory lesions. Inhibitors to MMPs, called tissue inhibitors of metalloproteinases, are also secreted by fibroblasts to regulate extracellular degradation. Intracellular degradation is for the normal physiological turnover and remodeling of collagenous connective tissue.

Immune Cells

A normal dental pulp has a large number and a variety of indigenous immune cells that play a crucial role to maintain homeostasis.[200, 201] Dendritic cells expressing Class II major histocompatibility complex (MHC) molecule are considered to participate in immunosurveillance. These cells have distinctive cytoplasmic extensions, have a high surface area for capturing antigens, while lacking intracellular lysosomes and phagocytic vacuoles.[200] MHC class II antigen presenting dendritic cells are predominantly located in the paraodontoblastic area and along the pulp–dentin border and in the central portion of the pulp (Figure 2-28). Macrophages also express MHC molecules, and these cells are present in the central portion of the dental pulp. In the healthy dental pulp, resident macrophages act as scavengers, eliminating dead odontoblasts and apoptotic bodies.[202,203] Class II MHC-expressing cells increase in number shortly after tooth eruption and continue to increase even after the completion of tooth formation.[204]

Other immune cells in the dental pulp are T lymphocytes that are largely present in the normal dental pulp with both cytotoxic and helper subtypes.[200, 205] Granulocytes are not uncommon in the healthy dental pulp. However, granulocyte numbers are much lower in the dental pulp than gingiva.[161] B lymphocytes have not been clearly demonstrated in the normal dental pulp. Plasma cells are not common in the dental pulp. It is however increased after sympathectomy.[162]

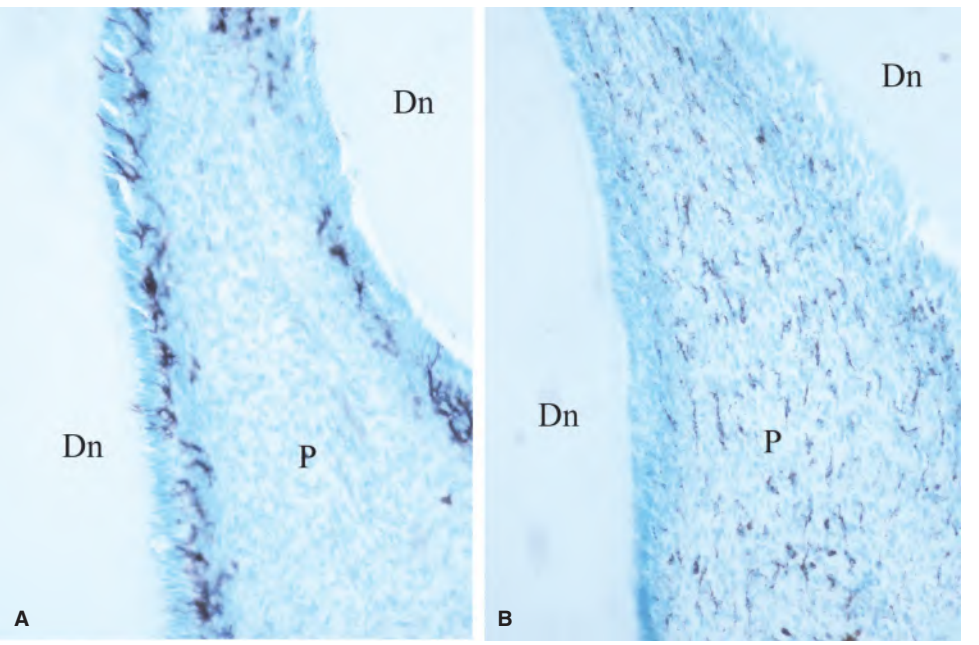


FIGURE 2-28 Immunohistochemical image from healthy coronal dental pulp with pulp horn region. Immune cells are from two different cell lines. ***A.*** MHC class II antigen presenting cells located within the odontoblast layer. ***B.*** Resident macrophages in the dental pulp proper. (Courtesy of Dr. Sivakami R. Haug, Bergen, Norway.)

Eosinophils are not common in a healthy dental pulp. Mast cells have not been shown to exist in the uninflamed dental pulp.[201,206–209] During an inflammatory process, all immune cell numbers are increased.[210,211] Inflammation of the dental pulp is discussed in another chapter.

Stem Cells

Dental pulp stem cells (DPSC) are highly proliferative, have the capacity for self-renewal, and have the ability to differentiate in response to injury. These cells were shown to produce sporadic and densely calcified nodules.[212,213] These cells were previously described as undifferentiated mesenchymal cells and are possibly the source of replacement odontoblasts that produce reparative dentin.[212] In addition to DPSC, stem cells are also present in the apical papilla of developing teeth.[212] For more details, see Chapter 29.

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Chapter 3

Microbial and Nonmicrobial Etiologies of Endodontic Diseases

José F. Siqueira, Jr, Isabela N. Rôças, Domenico Ricucci

Apical periodontitis is an inflammatory disease of microbial etiology, primarily caused by infection of the root canal system.[1] This disease may be associated with untreated (primary apical periodontitis) or treated teeth (posttreatment apical periodontitis) and can be asymptomatic (or chronic) or symptomatic (or acute). Chemical and physical factors can induce periradicular inflammation, but a large body of evidence indicates that infection is essential to the progression and perpetuation of the different forms of apical periodontitis.[2–5] Endodontic infection develops in root canals devoid of host defenses, either as a consequence of pulp necrosis or pulp removal for treatment.

Bacteria are the main microorganisms involved in endodontic infections. Other microorganisms such as fungi, archaea, and viruses have been occasionally found in association with endodontic infections,[6–10] but bacteria are virtually always present and are regarded as the major microorganisms implicated in the pathogenesis of apical periodontitis. In the root canal, most bacteria are usually found organized in biofilms adhering to the canal walls.[11–14] As a consequence, apical periodontitis has been included in the roll of biofilm-related oral diseases (Figure 3-1).[15,16]

Endodontics is ultimately concerned with prevention and treatment of apical periodontitis. Because it is an infectious disease, the rationale for endodontic treatment is to prevent or eradicate infection of the root canal system and the periradicular tissues. The cardinal principle of any healthcare profession is the thorough understanding of disease etiology and pathogenesis that provides a framework for effective treatment. In this context, understanding the microbiologic and non-microbiologic aspects of apical periodontitis is essential for an endodontic practice of excellence and founded on solid scientific bases.

APICAL PERIODONTITIS IS AN INFECTIOUS DISEASE

The Dutch amateur microscope builder Antony van Leeuwenhoek (1632–1723) was probably the first one to report the occurrence of microorganisms (called by him as "animalcules") in the root canals of a decayed tooth.[17] However, the role of Leeuwenhoek's "animalcules" in disease causation was largely unknown at that time. The microbial etiology of apical periodontitis was only suggested ~200 years later, when

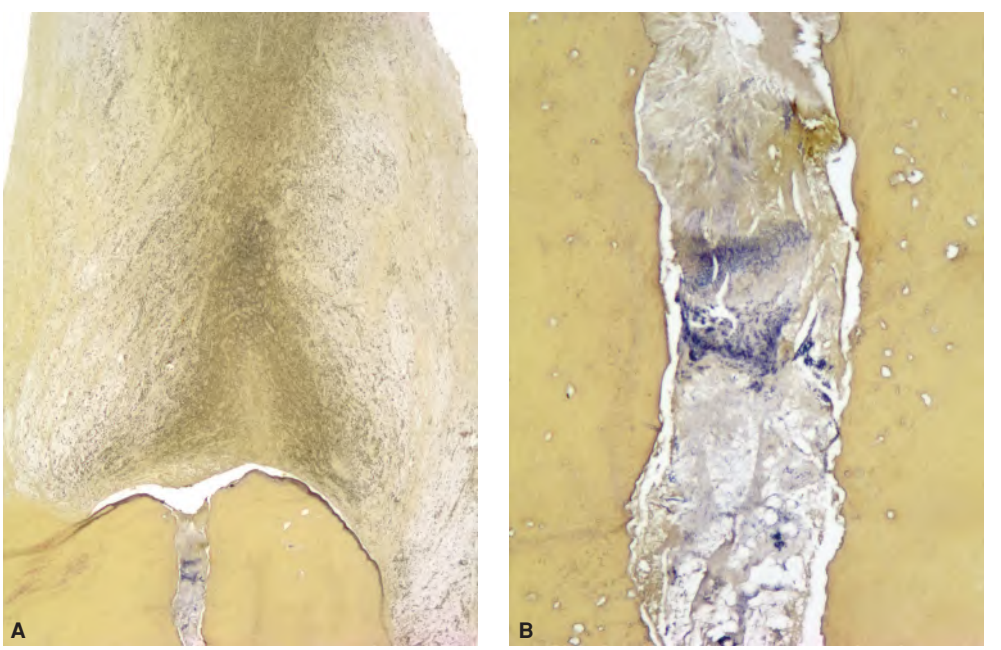

FIGURE 3-1 ***A***. Maxillary canine extracted with a large periapical lesion attached. Longitudinal section cut at the center of the foramen (Taylor's modified Brown & Brenn, original magnification ×16). ***B***. Detail of the apical canal showing its lumen clogged with necrotic debris and bacteria arranged in biofilm structures (original magnification ×100).

Willoughby Dayton Miller, an American dentist, working at the laboratory of Robert Koch in Berlin, Germany, reported the association between bacteria and apical periodontitis after the analysis of samples collected from root canals.[18] Miller observed that the bacterial diversity (as inferred by optical microscopy) was clearly different in the coronal, middle, and apical parts of the root canal. Spirochetes were found in high prevalence in acute apical abscesses, and an etiologic role was suspected for these bacteria. Miller also reported that most of the bacteria seen under microscopy could not be grown in culture. Those were conceivably anaerobic bacteria, which were only successfully cultivated ~50 to 100 years later with advanced technologies. It is important to point out that even today a large number of bacterial species living in diverse environments still remain to be cultivated.[19,20]

Miller reported association between bacteria and endodontic disease, but the cause-and-effect relationship was revealed only ~70 years later by Kakehashi et al.[2] These authors exposed the pulps of conventional and germ-free rats to their own oral cavities and observed by histology that although pulp necrosis and apical periodontitis lesions developed in all conventional rats, the pulps of germ-free rats not only remained vital but also repaired themselves by deposition of a dentin-like tissue that isolated them again from the oral cavity.

Other classic studies confirmed the important role of bacteria in the etiology of apical periodontitis. Sundqvist[3] used advanced anaerobic culturing techniques to evaluate the bacteriological conditions of pulps that became necrotic after trauma to the teeth. Bacteria were found only in the root canals of teeth with apical periodontitis. Most of them were obligate anaerobes, which corresponded to >90% of the isolates. In a study in monkeys, Möller et al.[4] demonstrated that only devitalized pulps that were infected induced apical periodontitis lesions, whereas devitalized and noninfected pulps did not cause significant pathologic changes in the periradicular tissues. These studies also revealed that the necrotic pulp per se is unable to induce and maintain an apical periodontitis lesion in the absence of infection.

Bacteria in root canals are primarily arranged in biofilm structures. Nair[11] was apparently the first one to observe intracanal bacterial organizations adhered to the root canal walls, resembling biofilms. Several other morphologic studies found similar structures,[12,13,21] but it was not until the study of Ricucci and Siqueira[14] that the high prevalence of bacterial biofilms and their association with primary and posttreatment apical periodontitis were consistently revealed.

PATHWAYS OF ENDODONTIC INFECTION

The pulpodentin complex is normally well isolated from oral bacteria by an intact hard tissue encasement and by a healthy periodontium. Loss of enamel or cementum integrity results in exposure of the pulpodentin complex to the oral environment and consequently to microorganisms present in saliva, caries lesions, or dental plaque formed on the exposed area. Conditions that may expose the pulpodentin complex include caries, trauma-induced fractures and cracks, leaking restorations, scaling and root planning procedures, attrition, and abrasion. Microorganisms from subgingival biofilms associated with periodontal disease may have access to the pulp via dentinal tubules at the cervical region of the tooth and lateral and apical foramina. (For more details, see Chapter 36.) Microorganisms may also have access to the root canal any time during or after root canal treatment.

Caries is by far the most common source of bacterial exposure to the pulp and is especially threatening when the lesion reaches the vicinity of the pulp (Figure 3-2). Whenever dentin is exposed, the pulp is put at the risk of infection as a consequence of the permeability of normal dentin dictated by its tubular structure.[22] The smallest diameter of the dentinal tubules is observed at the periphery, near the enamel, or cementum (mean, 0.9 μm),[23] and it is still compatible with the diameter of most oral bacteria, which usually ranges from 0.2 to 0.7 μm. However, frank invasion of the vital pulp by bacteria does not usually occur until a small width of dentin remains between the caries lesion and the pulp, and dentin permeability is significantly increased (Figure 3-3). However, bacterial invasion of dentinal tubules occurs more rapidly in nonvital teeth,[24] which helps explain the bacterial invasion of traumatized teeth that developed pulp necrosis. In vital teeth, the outward movement of dentinal fluid, the tubular contents, dentinal sclerosis, tertiary dentin, smear layer, intratubular deposition of fibrinogen, and host defense molecules also counterattack bacterial invasion.[25–28]

In contrast, direct exposure of the pulp to the oral environment is the most obvious route of endodontic infection as oral bacteria may now gain frank access to the tissue. Even a minuscule exposure can be critical. The most common causes of pulp exposure are caries, crown fracture due to trauma, and iatrogenic restorative procedures. Almost invariably, the exposed pulp will undergo inflammation and necrosis and subsequently becomes infected (Figure 3-4). The time elapsed between pulp exposure and infection of the entire canal is unpredictable, but it is usually a slow process.[29]

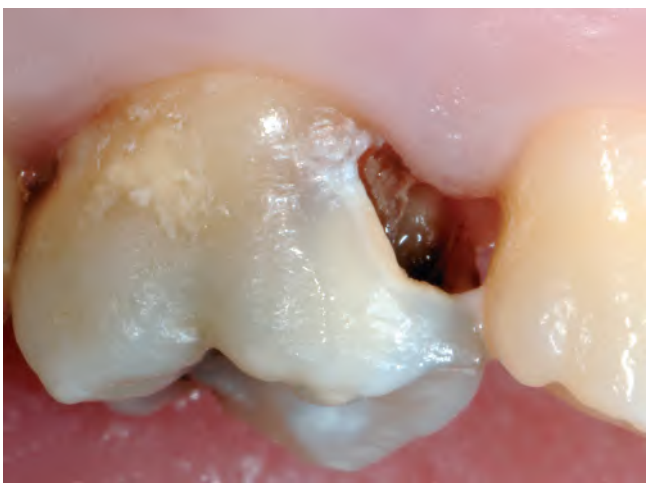

FIGURE 3-2 Maxillary first molar of a 20-year-old woman with a deep mesial caries lesion. The patient complained of sensitivity to cold and sweet stimuli.

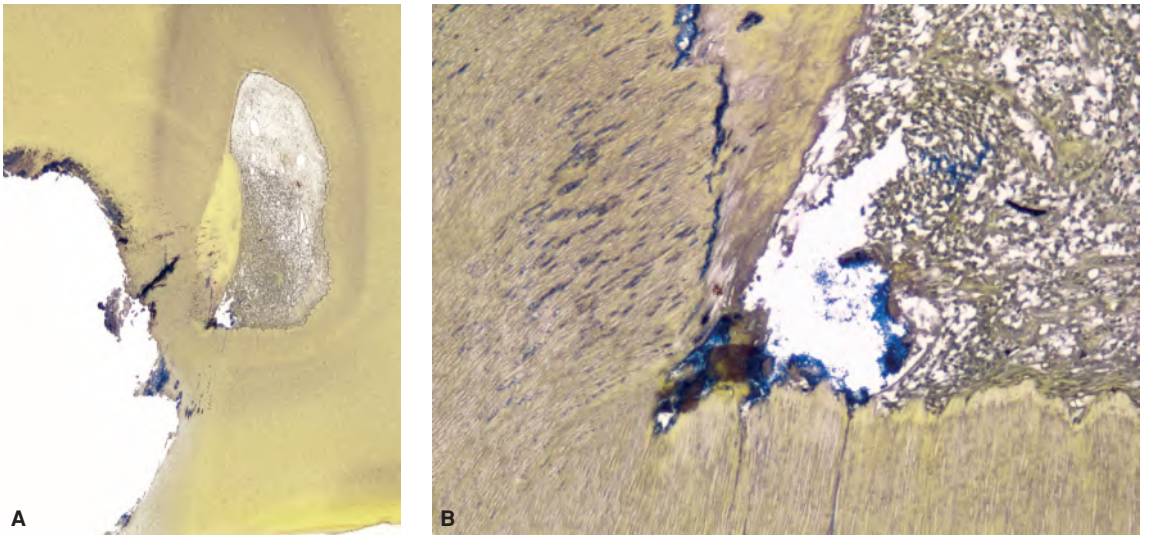

FIGURE 3-3 Symptomatic maxillary third molar with deep mesial caries. ***A***. The thin residual dentin layer is heavily infiltrated by bacteria owing to necrosis and infection localized in the subjacent pulp horn. Histologic serial sections could not demonstrate a perforation of the pulp chamber (Taylor's modified Brown & Brenn, original magnification ×16). ***B***. Detail from the pulp horn showing bacterial colonization (original magnification ×100).

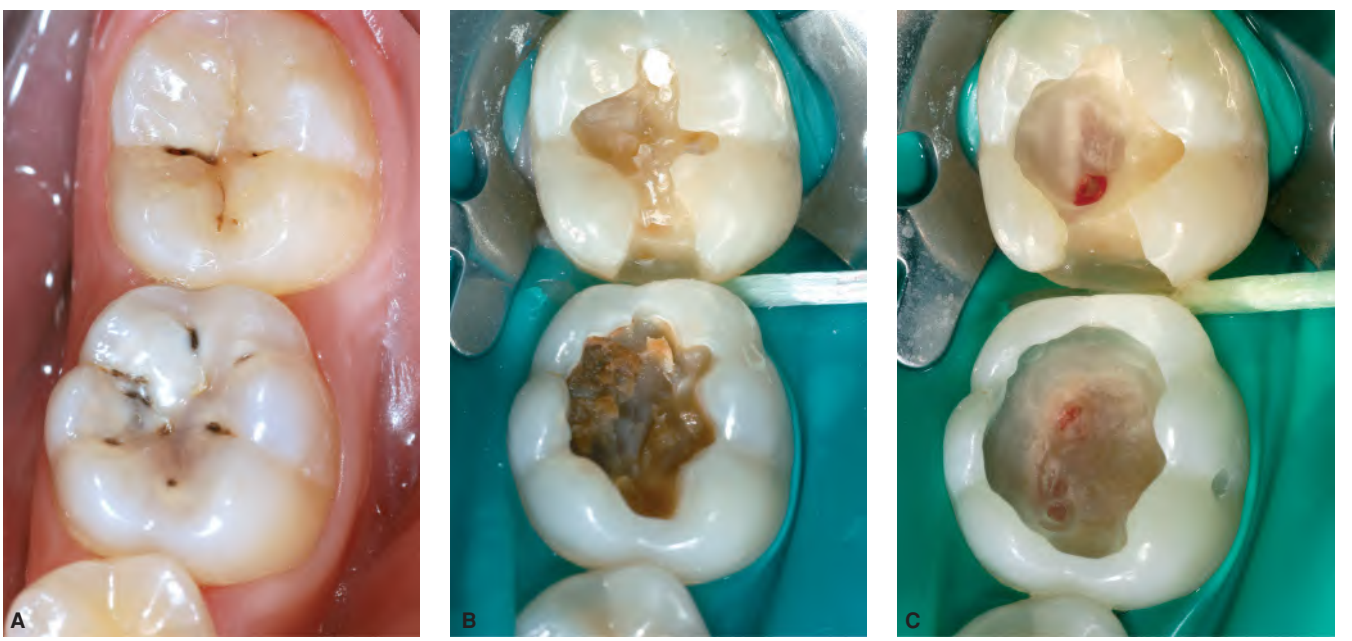

FIGURE 3-4 ***A***. A 17-year-old girl with carious lesions involving mandibular first and second molars. Apparently there are only small occlusal cavities in the enamel. ***B***. After removal of the occlusal enamel, a large amount of decayed dentin appears. ***C***. Careful excavation revealed perforations in the pulp horn areas.

Microorganisms in subgingival plaque biofilms associated with periodontal disease may reach the pulp by the same pathways intracanal microorganisms reach the periodontium. However, it has been shown that although degenerative and inflammatory changes of different degrees may occur in the pulp of teeth with associated periodontal disease, pulpal necrosis usually develops only when the periodontal biofilm reaches the main apical foramen, leading to irreversible damage to the main blood vessels that enter this foramen to irrigate the pulp (Figure 3-5).[30] Therefore, periodontal disease is usually a source of bacteria infecting the pulp once the disease reaches the root apex and causes pulp necrosis.

Although it has been suggested that microorganisms could reach the pulp via bloodstream in a phenomenon called anachoresis,[31–33] there is no solid evidence for it. A study showed that bacteria could not be recovered from unfilled root canals when the bloodstream was experimentally infected, unless the root canals were overinstrumented during the period of bacteremia, resulting blood seepage into the canal.[34]

In the past, anachoresis was used to explain why traumatized teeth, which were apparently intact, could have their root canals infected.[31] How did bacteria gain access to the canal? First, it is salient to point out that endodontic infection only develops after the pulp becomes necrotic or removed. Trauma, including concussion, subluxation, and various forms of tooth displacement, may cause rupture of the neurovascular supply at the root apex, a major internal bleeding and ischemia, leading to complete pulp necrosis. Because regeneration of the necrotic pulp is highly unlikely in mature teeth,[35] the pulp tissue is now devoid of a defense apparatus against microbial invasion. The origin of infecting microorganisms in these cases is the oral cavity, specifically via cracks in the enamel and dentin.[36–38] Macro- and microcracks are expected in traumatized teeth, as well as in teeth with caries and large restorations, and do not necessarily end at the enamel–dentin junction, but extend deep into the dentin and even reach the pulp (Figure 3-6).[36,38]

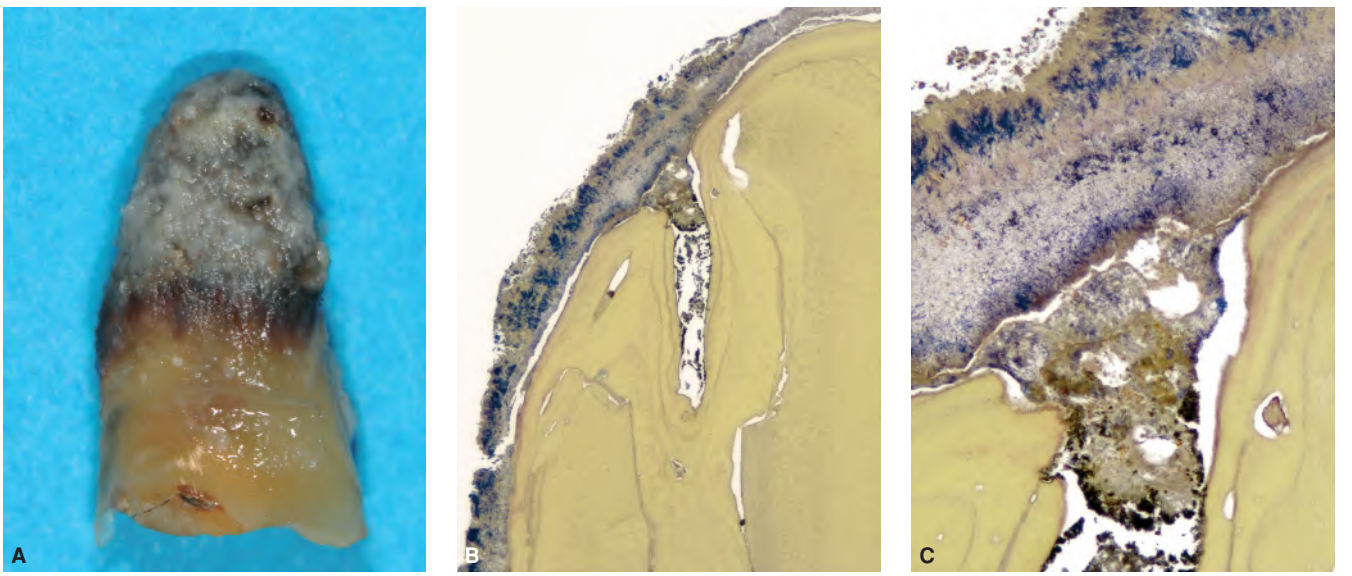

FIGURE 3-5 ***A***. Palatal root from a rizectomized maxillary first molar. Note that the apical half of the root is totally covered by calculus. ***B***. Section cut through the apical foramen emerging laterally to the root apex. A bacterial biofilm has engulfed the apical foramen, cutting the circulation and invading the apical root canal retrogradely (Taylor's modified Brown & Brenn, original magnification ×16). ***C***. Higher magnification of the foraminal area (original magnification ×100).

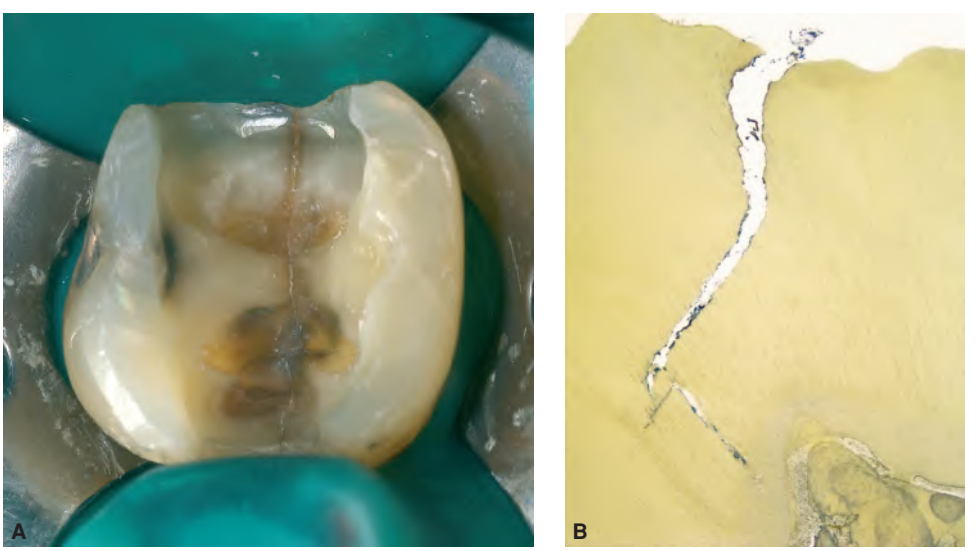

FIGURE 3-6 Mandibular molar with an extensive MOD amalgam restoration in a 55-year old man. The patient complained of pain upon chewing. ***A***. After anesthesia and removal of the old restoration, a mesio-distal crack crossing the entire cavity floor was evident. The tooth was extracted since the crack extended to the distal aspect of the root. ***B***. Section cut on a buccolingual plane. The crack appears close to the pulp chamber and is clogged with bacteria. Note the large pulp stone occupying the subjacent pulp horn (Taylor's modified Brown & Brenn, original magnification ×16).

Although the endodontic treatment is directed to prevention or control of the endodontic infection, there is a risk of secondary infection because the pulp is removed and the root canal is exposed and manipulated by the clinician. The source of microorganisms for a secondary infection includes a breach in the aseptic chain (during treatment), coronal leakage, fracture of the tooth or restorative material (between appointments and after treatment).[39]

PATTERNS OF MICROBIAL COLONIZATION OF THE ROOT CANAL SYSTEM

Spatial Distribution of Bacteria in Infected Canals

Understanding how microorganisms colonize the root canal system is of crucial therapeutic importance. With this knowledge in mind, researchers and clinicians can envision and develop enhanced strategies to eliminate infection from the entire system and improve outcome.

Morphologic studies of teeth with necrotic pulps and primary apical periodontitis lesions have demonstrated that although planktonic bacterial cells suspended in a fluid phase and enmeshed in the necrotic pulp tissue can be observed in the main root canal (Figure 3-7), the majority of bacteria colonizing the root canal system are organized in sessile biofilm communities adhering to the canal walls (Figure 3-8).[11–15] Bacterial cells from the bottom of the biofilm structures are often seen penetrating the subjacent dentinal tubules to varying depths (Figure 3-9). Dentinal tubule infection can occur in about 70% to 80% of the teeth associated with apical periodontitis lesions.[40,41] A shallow penetration is more common, but bacterial cells can be observed reaching ~300 µm in some teeth.[12] Infection can also spread to lateral canals, apical ramifications, isthmuses, and other areas in the root canal system (Figures 3-10 and 3-11).[42,43]

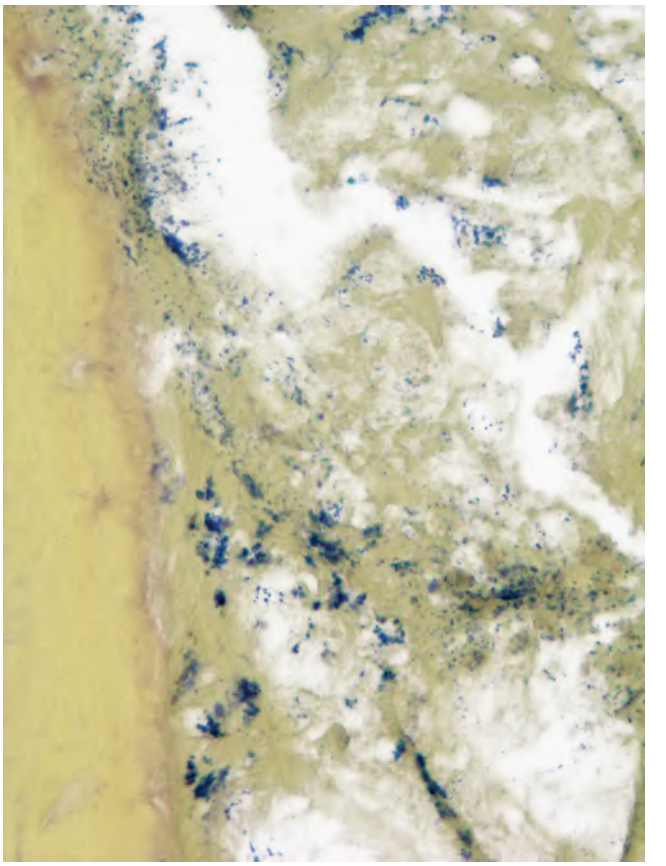

FIGURE 3-7 Necrotic root canal. Bacteria enmeshed in the necrotic pulp tissue, apparently in the planktonic state (Taylor's modified Brown & Brenn, original magnification ×400).

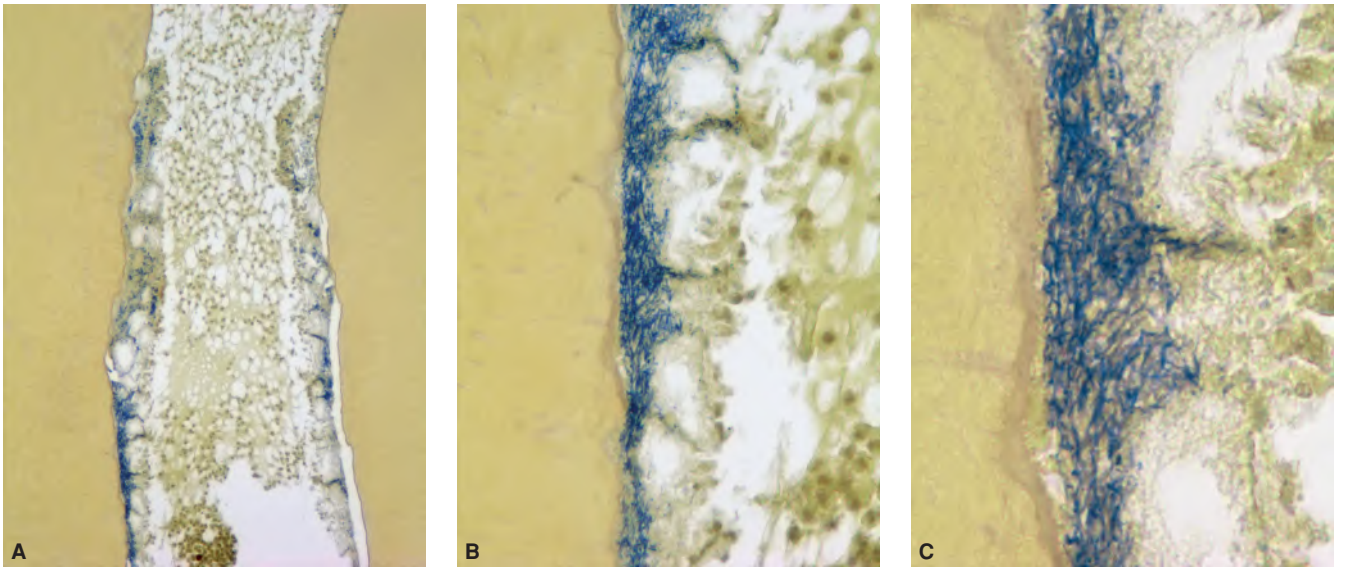

FIGURE 3-8 ***A***. Necrotic root canal. Biofilms adhering to the canal walls (Taylor's modified Brown & Brenn, original magnification ×100). ***B & C***. Progressive magnifications of the area of the left wall indicated by the arrow in A (original magnification ×400 and ×1000).

Bacteria found as planktonic cells in the main root canal are conceivably easy to eliminate by instruments and irrigants during preparation. Nevertheless, bacterial arrangements in biofilms, especially those located in difficult-to-reach areas, are definitely more difficult to access and eliminate.[42,44]

COMMUNITY-BASED PATHOGENESIS

Bacterial community refers to a unified assemblage of microcolonies or populations that coexist and interact at a given habitat. These populations perform functions that contribute to the physiology and pathogenicity of the overall community. Highly structured and spatially organized communities may exhibit properties that are greater than the sum of the component populations. Therefore, the interplay of the different parts composing the community will ultimately determine its properties. The community-forming ability can be regarded as essential for microbial survival in virtually all environments. The vast majority of microorganisms in nature invariably grow and function as members of metabolically integrated communities—the so-called biofilms (see below).[45,46]

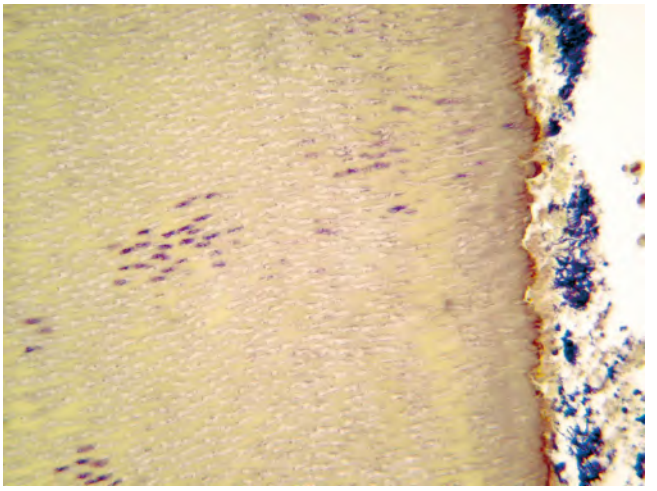

FIGURE 3-9 Bacteria penetrating dentinal tubules to a considerable depth from the bottom of the biofilm covering the canal wall (Taylor's modified Brown & Brenn, original magnification ×100).

The ability of forming organized biofilm communities also influences pathogenicity of involved microorganisms. The concept of the community as the unit of pathogenicity is based on the principle that teamwork is what eventually counts. The community behavior and the outcome of the host/bacterial community interaction will depend on the types and numbers of species present in the community biofilm and the network of resulting associations among them.[47]

Community-profiling molecular studies have provided insights into the endodontic bacterial community structure, offering new perspectives on the etiology and pathogenesis of apical periodontitis.[48] These studies moved the focus from the "single-pathogen," which was very popular in classic medical microbiology, to the "community-as-pathogen" concept that has arisen to explain the pathogenesis of polymicrobial infections, such as apical periodontitis. The fact that the bacterial community structure (composition and abundance of members) in endodontic infections varies from individual to individual indicates that apical periodontitis has a heterogeneous etiology and is the result of the

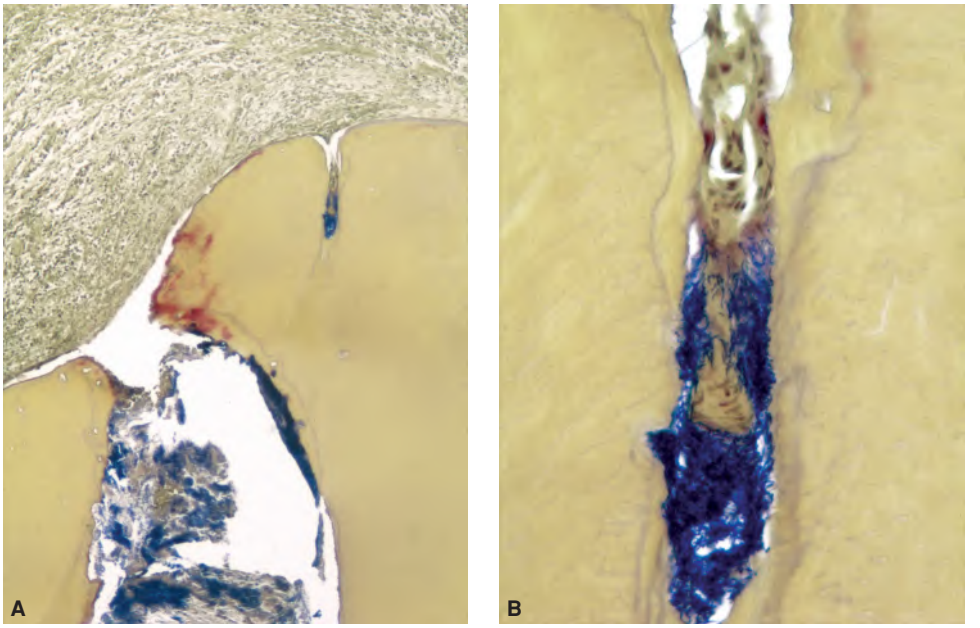

FIGURE 3-10 ***A***. Root canal with longstanding pulp necrosis. The main apical foramen is located laterally to the root apex. The canal is filled with a bacterial biofilm. The exit of a tiny apical ramification (arrow) can be seen at the geometrical top of the root (Taylor's modified Brown & Brenn, original magnification ×50). ***B***. High power view of the ramification reveals its lumen filled with bacterial biofilm, faced with inflammatory cells apically (original magnification ×400).

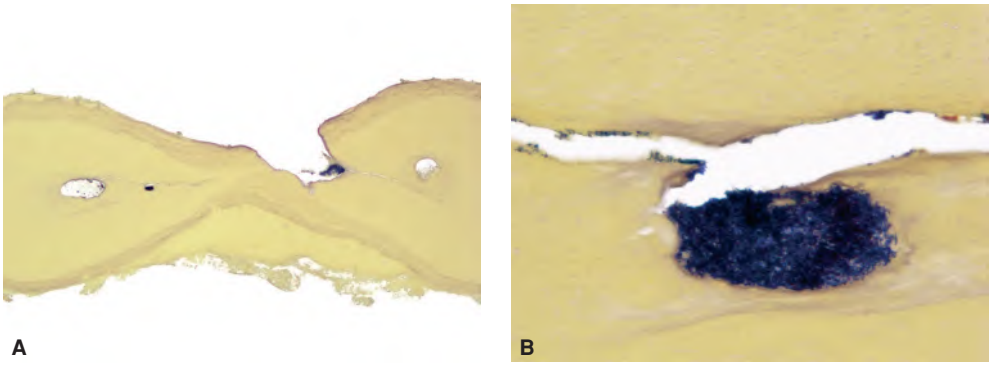

FIGURE 3-11 ***A***. Mesial root of a mandibular first molar with necrotic pulp and a large apical periodontitis lesion. Transversal section cut at about 2 mm from the apex. Two main canals can be seen. A narrow and long isthmus is departing from both canals (Taylor's modified Brown & Brenn, original magnification ×16). ***B***. High power view of the area of the isthmus indicated by the arrow in A. A dilation of the isthmus appears completely clogged by a thick bacterial biofilm. Biofilms can also be appreciated on the walls of the isthmus (original magnification ×400).

collective pathogenicity of the bacterial species composing and interacting in the endodontic biofilm.

Biofilm Infections

Biofilm can be defined as a sessile multicellular microbial community characterized by cells that are firmly attached to a hard or soft surface and enmeshed in a self-produced matrix of extracellular polymeric substance (EPS), usually polysaccharide. However, it can contain proteins and nucleic acids (Figure 3-8).[45,49,50] Bacterial cells in biofilms form microcolonies (~15% by volume) embedded and nonrandomly distributed in the EPS matrix (~85% by volume) and separated by water channels.[50–53] Dental biofilms can reach up to 300 or more cell layers in thickness.[53] 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 plays a role in bacterial adhesion to the surfaces, serves as a scaffold that determines the biofilm structure; retains nutrients, water, and essential enzymes; and provides protection against exogenous threats, such as antimicrobials and host defense cells and molecules.[54,55] The water channels permit circulation of fluid that carries nutrients, waste products of bacterial metabolism, and signal molecules involved in bacterial intercommunication systems.[56]

The biofilm community lifestyle affords a number of advantages to colonizing bacteria, including establishment of a broad habitat range for growth, increased metabolic diversity and efficiency, enhanced possibilities for genetic exchanges and bacterial intercommunications as well as protection from external threats.[57] Biofilm organizations can also result in enhanced and collective pathogenicity. In multispecies communities, a broad spectrum of relationships may arise between the component species, and in some circumstances interactions may result in synergistic pathogenic effects.

Bacteria in biofilms exhibit increased resistance to antimicrobial agents when compared to their counterparts growing in planktonic state. For instance, the antibiotic concentration required to kill bacteria in a biofilm can be ~100 to 1000 times higher than that needed to kill the same species in planktonic state.[58] The possible reasons for increased biofilm resistance include restricted penetration of antimicrobial agents in the biofilm, the altered growth rate of biofilm bacteria and the presence of a subpopulation of specialized survivor cells with altered phenotypes (so-called "persisters").[49,59]

Apical Periodontitis as a Biofilm-associated Disease

Although bacterial organization in biofilms had been revealed by isolated morphological studies, the prevalence of biofilms and their association with diverse presentations of apical periodontitis were only recently disclosed in a study by Ricucci and Siqueira.[14] Histobacteriological analysis of the apical root canal system of teeth with primary or posttreatment apical periodontitis yielded some important findings:

a. Bacteria were present in all root canals but biofilms occurred in ~80% of the root canals of teeth with primary or posttreatment apical periodontitis.
b. Biofilms had varied morphologies in terms of thickness, morphotypes, and bacterial cells/extracellular matrix ratio.
c. Biofilms were seen not only in the main root canal, but also in apical ramifications, lateral canals, and isthmuses (Figures 3-10 and 3-11).
d. Biofilms were more frequent in root canals of teeth with large apical periodontitis lesions as shown in radiographs and lesions histopathologically diagnosed as cysts. Large lesions (most of them were cysts) 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.
e. Biofilms adhered to the outer root surfaces (extraradicular biofilms) were present in only 6% of the teeth and usually associated with intraradicular biofilms and clinical symptoms (Figure 3-12).

FIGURE 3-12 Mesiobuccal root of a maxillary second molar with longstanding pulp necrosis. The section, cut at the center of the foramen, discloses a bacterial biofilm extending from the root canal walls onto the external apical surface. Note the large area of resorption (Taylor's modified Brown & Brenn, original magnification ×16).

f. Flocs, which are different forms of biofilms (planktonic large colonies), were seen in some teeth in the main canal lumen.

Apical periodontitis seems to fulfill most of the established criteria used to determine whether a given infectious disease can be classified as a disease caused by biofilm communities.[14,60,61] Nonetheless, because biofilms are detected in the majority but not in all cases of apical periodontitis, it remains to be determined if biofilm formation is required for apical periodontitis development. For time being, it seems prudent to state that apical periodontitis is a disease strongly associated with bacterial biofilms.[62]

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 based on the time infecting microorganisms gained entry into the root canal system: *primary infection,* caused by microorganisms that invade and colonize the necrotic pulp tissue; *secondary infection,* caused by microorganisms not present in the primary infection, but introduced in the root canal at some time during or after professional intervention; and *persistent infection,* caused by microorganisms that were members of a primary infection and in some way resisted intracanal antimicrobial procedures and were able to endure periods of nutrient deprivation in treated canals.

Primary infections are the cause of primary apical periodontitis. Persistent infections and less frequently secondary infections are the main cause of posttreatment apical periodontitis. *Extraradicular infection* in turn is characterized by microbial invasion of the inflamed periradicular tissues and is a sequel to the intraradicular infection. Extraradicular infections occur in abscessed cases and have been suggested as a possible cause of posttreatment apical periodontitis.

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. Because all the studies of the endodontic microbiota are cross-sectional in nature, data are restricted to prevalence of species and associated with disease; consequently, causation cannot be proven. Thus, any species that has a pathogenic potential (as inferred by animal studies or participation in other diseases) and have been found in high prevalence in endodontic infections are regarded as candidate or putative endodontic pathogens.

ETIOLOGY OF PRIMARY APICAL PERIODONTITIS

Primary Intraradicular Infection

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.[3,63–66] Molecular studies have disclosed a mean of 10 to 20 species/phylotypes per infected canal.[67–71] Canals of teeth with sinus tracts may exhibit a number of species close to the top of this range.[70] The more complex the infection, the larger the apical periodontitis lesion; in other words, the size of periradicular bone destruction is proportional to the number of bacterial species (diversity) and cells (density) in the root canal.[3,70,72]

Bacterial species/phylotypes detected in primary infections fall into 9 of the 13 phyla that have oral representatives, namely *Firmicutes*, *Bacteroidetes*, *Fusobacteria*, *Actinobacteria*, *Proteobacteria*, *Spirochaetes*, *Synergistetes*, TM7, and SR1.[64,67,70,73–75] However, there may be representatives of at least nine other phyla in endodontic infections as revealed by pyrosequencing.[76–81] Figure 3-13 depicts a phylogenetic tree with the species/phylotypes more commonly detected in endodontic infections. The following is an overview of the major bacterial groups and species regarded as candidate endodontic pathogens.

Gram-Negative Bacterial Species

Fusobacterium species, especially *Fusobacterium nucleatum*, are common members of the endodontic microbiota in primary infections, including acute apical abscesses.[79,82–90] PCR-based microbial typing approaches have revealed that different clonal types of *F. nucleatum* can be isolated from the same infected canal.[91]

Black-pigmented gram-negative anaerobic rods include species formerly known as *Bacteroides melaninogenicus*. These bacteria have been reclassified into two genera—the saccharolytic species were transferred to the genus *Prevotella* and the asaccharolytic species to the genus *Porphyromonas*.[92,93]

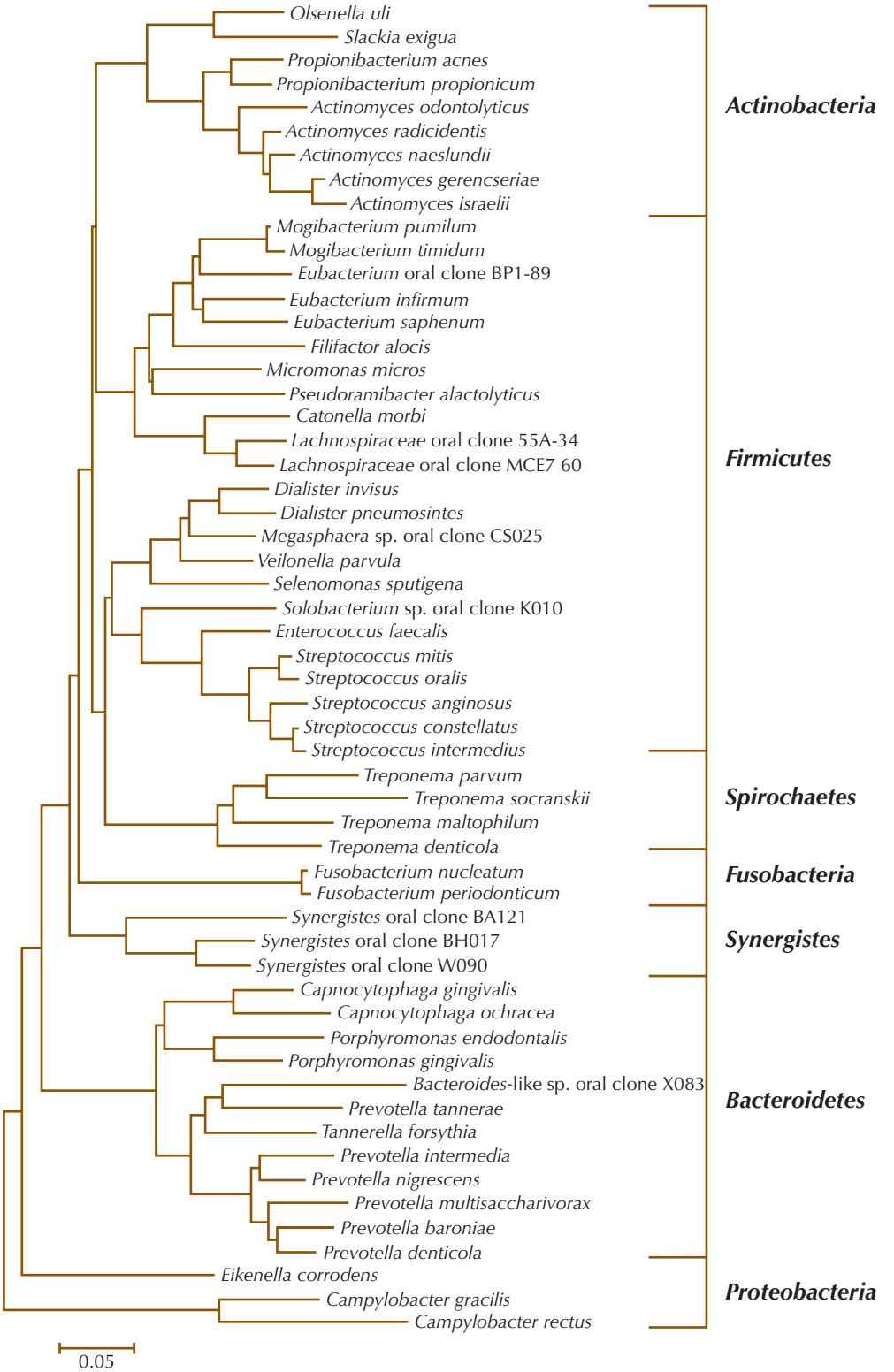

FIGURE 3-13 Phylogenetic tree of the most prevalent bacterial species and phylotypes found in endodontic infections. Note that the major endodontic pathogens fall into seven major phyla. Other phyla have been identified, but they do not contain species that are frequently detected.

Some bile-sensitive nonpigmented *Bacteroides* species were also transferred to the genus *Prevotella*.[94] *Prevotella* species frequently detected in primary endodontic infections include *Prevotella intermedia*, *Prevotella nigrescens*, *Prevotella multissacharivorax*, *Prevotella baroniae*, and *Prevotella denticola*.[74,87,95–106] *Prevotella tannerae*, which has also been found in endodontic infections,[107,108] has been transferred to the new genus *Alloprevotella*.[109] Of the *Porphyromonas* species, only *Porphyromonas endodontalis* and *Porphyromonas gingivalis* have been consistently found in endodontic infections

and they seem to play an important role in the etiology of different forms of apical periodontitis lesions, including acute apical abscesses.[90,95,96,104,110–114]

Dialister species are asaccharolytic obligate anaerobic gram-negative coccobacilli that represent another example of bacteria that have been consistently detected in endodontic infections only after the advent of molecular microbiology techniques. *Dialister invisus* and *Dialister pneumosintes* are among the most frequently detected species in asymptomatic and symptomatic primary endodontic infections in several molecular studies.[67,71,73–75,89,115–119]

Spirochetes are highly motile spiral-shaped Gram-negative bacteria that have been linked to several oral diseases.[120–122] For many years, spirochetes have been observed in samples from endodontic infections by microscopy, but they have never been reliably identified to the species level.[11,18,123] Culture-independent molecular microbiology methods allowed speciation of spirochetes in endodontic infections and revealed that all of the hitherto named oral treponemes can be found in primary endodontic infections.[124–137] The most prevalent treponemes in infections of endodontic origin are *Treponema denticola* and *Treponema socranskii*.[124,125,129,132,136,137] The species *Treponema parvum, Treponema maltophilum,* and *Treponema lecithinolyticum* have been moderately prevalent.[125,126,131,132,137]

Tannerella forsythia (formerly *Bacteroides forsythus*) is a recognized periodontal pathogen and another example of species that has been detected in endodontic infections only by molecular methods, usually in relatively high prevalence.[70,71,84,86,112,133,138–141]

Gram-Positive Bacterial Species

Gram-positive anaerobic rods have also been found as common members of the microbiota associated with primary infections. Of these, *Pseudoramibacter alactolyticus* has been detected by culture-dependent and culture-independent studies in frequencies as high as the most prevalent Gram-negative species,[71,82,142] including the very apical part of the root canal.[143,144] *Filifactor alocis* is an obligate anaerobic rod that had been only occasionally isolated from root canal infections by culture,[82] but molecular studies detected this species in several cases of primary endodontic infections.[145–147] Other Gram-positive rods, including *Actinomyces* species, *Propionibacterium propionicum*, *Olsenella* species, have also been reported to occur in relatively high prevalence in infected root canals.[67,70,82,86,148–153]

Some Gram-positive cocci are frequently present in primary endodontic infections. *Parvimonas micra* (formerly *Peptostreptococcus micros*) have been among the most commonly detected species in several studies, including high prevalence in symptomatic infections.[70,82,83,89,102,103,154,155] Members of the *Streptococcus anginosus* group have been reported to be the most prevalent streptococci, but other species of this group can also be often recovered/detected.[82,89,90,149] *Enterococcus faecalis*, which has been closely found in association with root-filled teeth, is not so frequent in primary infections.[149,156]

Many other Gram-positive and Gram-negative bacterial species have been detected in endodontic infections, but in lower prevalence than the ones discussed here. For a comprehensive review of the bacteria already detected in endodontic infections, the reader is referred to a review article by Siqueira and Rôças.[157]

As-Yet-Uncultivated Phylotypes

Molecular studies have demonstrated that as-yet-uncultivated bacteria comprise ~40% to 60% of the species-level taxa found in primary infections.[64,67,71,74,158] Uncultivated phylotypes from several genera have been identified, including *Dialister*, *Treponema, Prevotella*, *Solobacterium*, *Olsenella*, *Fusobacterium*, *Eubacterium*, *Megasphaera*, *Veillonella*, and *Selenomonas* as well as phylotypes related to the family *Lachnospiraceae* or the *Synergistetes* and TM7 phyla.[67,73–75,115,137,159–161] One of the most prevalent as-yet-uncultivated phylotypes found in endodontic infections is *Bacteroidaceae* sp. HOT-272 (synonym, *Bacteroidetes* oral clone X083).[70,106] Some members of the *Synergistetes* phylum that had been previously regarded as uncultivated and detected in endodontic infections in relatively high prevalence[75,115,162,163] have been recently cultivated, characterized, and formally named as *Pyramidobacter piscolens*[164] and *Fretibacterium fastidiosum*.[165]

The Complex Etiology of Symptoms

Symptomatic apical periodontitis and acute apical abscesses are the typical examples of endodontic infections causing severe symptoms. In these cases, infection is located within the canal system, but it has also advanced to the periradicular tissues. In acute apical abscesses, an extraradicular infection is established and results in purulent inflammation. Clinically, the disease leads to pain and/or swelling and has the potential to diffuse to sinuses and other fascial spaces of the head and neck to form a cellulitis or other complications. For more details, see Chapter 30.

A matter of great interest in endodontic microbiology is to find an explanation as to why only some infected teeth develop acute symptoms and complications. The desire to find a single or at least a group of major species that is associated with acute symptoms is an ever recurrent topic in the field of endodontic microbiology. In his milestone study, Miller[18] was the first one to suggest the involvement of a specific group of bacteria with endodontic symptoms—spirochetes were associated with acute abscesses. However, Sundqvist[3] was the first one to consistently report on the association of a given species with endodontic symptoms—dark-pigmented bacteria (formerly *B. melaninogenicus*) was found only in teeth with acute symptoms. Further culture and molecular studies reported on the association with symptoms of several species, the most frequent being *Porphyromonas* and *Prevotella* species, *F. nucleatum, P. micra,* and *S. anginosus* group.[3,79,96,149,166–169] Nevertheless, these findings have not been consistently confirmed by other studies, because basically the same species can also be found in the root canals

of teeth with asymptomatic apical periodontitis in similar prevalences.[68,70,84,86,95,101,112,141]

Animal studies have given some support to the potential role of some endodontic pathogens in causing abscesses. The ability to cause purulent infections in animal models strengthens the possible causative role of certain species clinically associated with symptomatic disease. Many candidate endodontic pathogens, alone or in combinations, have been shown to cause abscesses in animals.[170–174] One of the main problems with the animal model is that only cultivable bacteria have been tested, either in single culture or in combinations of two or a few species. Thus, any speculation about extrapolation of these results to a clinical condition where there is a network of interacting species (10 or more), including as-yet-uncultivated phylotypes, may lead to oversimplification.

Based on this discussion, one may assume that the etiology of acute endodontic infections is characterized by low specificity. The species that have been more frequently detected than others and either cause abscess in animals or are involved with diseases in other sites are suspected to play a role in making the community more virulent.[175,176] However, in addition to the presence of these pathogenic species, a multitude of other factors can be regarded as influential to the development of acute endodontic infections,[175] including (a) differences in the virulence ability among clonal types of the same species; (b) bacterial interactions resulting in collective pathogenicity; (c) overall and specific bacterial load; (d) environment-regulated expression of virulence factors; and (e) host resistance and disease modifiers.

Microbial Ecology in Endodontic Infections

The root canal system, containing necrotic pulp tissue, provides a space for bacterial colonization and affords bacteria a moist, warm, nutritious, and anaerobic environment that is by and large protected from the host defenses because of lack of active blood circulation in the necrotic pulp. In addition, the root canal walls are nonshedding surfaces conducive to persistent colonization and formation of complex biofilm communities. Although a large number of bacterial species (~100–200) can be found in the oral cavity of a particular individual,[177] only a limited assortment of these species (~10–20) is consistently selected for growth and survival within a root canal containing necrotic pulp tissue. This indicates that ecological determinants operate in the necrotic canal and dictate which species will succeed in colonizing this previously sterile environment. The major ecological factors that determine the composition of the root canal microbiota include oxygen tension, type, and amount of available nutrients, and bacterial interactions.

Oxygen Tension

Root canal infection is a dynamic process, and different bacterial species apparently dominate at different stages.[178] Shifts in the composition of the microbiota are largely due to changes in the environmental conditions, particularly in regard to oxygen tension and nutrient availability. In the very initial phases of the pulpal infectious process, facultative bacteria predominate.[179] 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. At this point, an anaerobic milieu develops; highly conducive to 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.

Type and Amount of Nutrients

Bacteria colonizing the necrotic root canal utilizes as the main source of nutrients (1) the necrotic pulp tissue itself; (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) end-products of the metabolism of other bacteria. Because the largest amount of nutrients is supposedly available in the main canal that is the largest area of the whole system, most of the infecting bacteria are expected to concentrate in this region.

Shifts in the composition of the microbiota colonizing the root canal system can also be dependent on 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.[180] The necrotic pulp tissue is a finite source of nutrients to bacteria. However, once inflammation is established at the periradicular tissues, a sustainable source of nutrients to bacteria in the apical root canal is maintained in the form of proteins and glycoproteins present in the exudate that seep into the canal. At this point, proteolytic and asaccharolytic bacteria start to dominate. 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 and/or amino acids in their metabolism.

Bacterial Interactions

Multispecies biofilms are the major form of bacterial organization in infected root canals. In these structures, 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. 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. They include the following:

a. interbacterial nutritional interactions (food chains/webs, and concerted action to break down complex substrates);
b. local environment modification;

c. collective protection against external threats;
d. intrageneric and intergeneric coaggregations;
e. cell–cell signaling (quorum-sensing systems); and
f. horizontal gene transfer.

For instance, interbacterial nutritional interactions are of special interest as they represent important ecological determinants leading to a high metabolic diversity and efficiency of the whole community. Nutritional interactions are mainly represented by food chain/webs, including utilization of metabolic end-products from one species by another, and bacterial cooperation for the breakdown of complex host-derived substrates.

In addition, one species can provide growth conditions favorable to another, for example, by reducing the oxygen tension in the environment and favoring the establishment of anaerobes (environment modification) or by releasing some proteinases or antibiotic-inactivating enzymes that can provide protection to the entire community from host defenses and antibiotics, respectively.

Many species adhere directly to host surfaces, while other species adhere to other bacteria already attached to the surface. The latter is called coaggregation and is a highly specific phenomenon with regard to the partners involved.[181] Coaggregation can favor colonization of host surfaces and also facilitate metabolic interactions between the partners. Coaggregation has been demonstrated for several pairs of endodontic bacteria.[182]

Negative interactions in turn act as feedback mechanisms that limit population densities. Examples include competition (for nutrients and space) and amensalism (when one species produces a substance that inhibits another species).

Other Microorganisms in Endodontic Infections

Fungi

Fungi are chemoorganotroph eukaryotic microorganisms that may be found in two basic forms: molds and yeasts. They constitute a small part of the oral microbiota, with members of the *Candida* genus as the most common fungal species. Fungi have been only occasionally detected in primary intraradicular infections.[85,183–185] This suggests that bacteria colonizing the root canal may inhibit the establishment of fungi. However, a molecular study[186] reported the occurrence of *C. albicans* in 21% of samples from primarily infected canals. Fungi are more frequently detected in canals of teeth with posttreatment apical periodontitis (see below).

Archaea

Archaea comprise a highly diverse group of prokaryotes distinct from bacteria. Members of the Archaea domain have been traditionally recognized as extremophiles, but some species have also been found to survive in nonextreme environments, such as the human body.[187] Some studies have failed to detect archaea in samples from primary endodontic infections,[188,189] others detected a *Methanobrevibacter oralis*-like phylotype in some primarily infected canals.[7,158] A study evaluating the exoproteome of endodontic infections detected one protein of archaeal origin linked to methane production.[190] Because archaea have not been consistently detected in infected root canals, their role, if any, in the ecology of the endodontic bacterial community and pathogenesis of apical periodontitis remains to be elucidated.

Virus

Viruses are particles structurally composed of a nucleic acid molecule (DNA or RNA) and a protein coat that require viable host cells to infect and replicate its genome. Thus, viruses cannot thrive in a root canal containing necrotic pulp tissue. The presence of viruses in the root canal has been reported only in teeth with vital pulps. For instance, the human immunodeficiency virus (HIV) has been detected in vital pulps of HIV-seropositive patients[191] and some herpesviruses have been identified in both noninflamed and inflamed vital pulps.[192]

On the other hand, viruses have been detected in apical periodontitis lesions, where living host cells abound. Herpesviruses, especially human cytomegalovirus (HCMV), Epstein-Barr virus (EBV), and Human Herpesvirus-8 (HHV-8), have been the most commonly detected viruses in apical periodontitis specimens. Suggestive evidence of herpesvirus infection has been observed in symptomatic apical periodontitis lesions,[193,194] abscesses,[10,114,195] and large lesions.[194,196] Papillomavirus has also been found in abscesses.[10]

It has been hypothesized that herpesviruses may 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 that might give rise to an overgrowth of pathogenic bacteria in the very apical part of the root canal.[9] The arrival of herpesviruses at the periradicular tissues seems to be related to the influx of virus-infected inflammatory cells in response to bacterial infection in the apical canal. Reactivation of herpesviruses by tissue injury induced by bacteria may 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. Moreover, herpesvirus-infected inflammatory cells are stimulated to release pro-inflammatory cytokines.[197,198]

The mere presence of virus in clinical samples does not necessarily imply a role in the pathogenesis of apical periodontitis. Several herpesviruses can be chronically present in the body infecting leukocytes and as these cells are attracted and accumulate in inflamed areas, these viruses will also be present. The role of herpesviruses in the pathogenesis of apical periodontitis, if any, has still to be clarified.

BACTERIAL CAUSES OF FLARE-UPS

Flare-up is "an acute exacerbation of an asymptomatic pulpal and/or periapical pathosis after the initiation or continuation of root canal treatment."[199] It is a true emergence,

characterized by severe pain and/or swelling that develops a few hours or the next day following professional intervention.[200] The periradicular tissues are acutely inflamed and the condition is diagnosed as an acute apical periodontitis or acute apical abscess secondary to treatment. Flare-ups are caused either by bacteria or iatrogenic approaches (e.g., overinstrumentation, apical extrusion of irrigants) or both.[200–204] There are numerous studies evaluating the influence of diverse factors in the incidence of flare-ups. The two most significant risk factors seem to be presence of pre-operative pain and intervention in the infected root canals (necrotic pulps and retreatment cases).[205–209] The prevalence of flare-ups ranges from 2.5% to 16%.[210]

Because of the low incidence and the unexpected/unpredictable nature of flare-ups, no study has so far consistently evaluated the bacterial species associated with this condition. It has been suggested that combinations of *F. nucleatum* and *Porphyromonas* and *Prevotella* species may lead to exacerbations.[211]

Flare-ups are caused by bacteria in the following situations: (a) apical extrusion of infected debris, (b) incomplete root canal instrumentation, and (c) secondary intraradicular infections. For more details, see Chapter 30.

Apical Extrusion of Infected Debris

Apical extrusion of infected debris during chemomechanical preparation is allegedly the major cause of postoperative pain.[201,203,204] In teeth with asymptomatic apical periodontitis, a balance is established between bacterial aggression from the infected canal and the host defenses at the periradicular tissues. If during chemomechanical preparation bacteria are forced to the periradicular tissues, the host will face a situation that is now challenged by a larger number of irrigants than before. Consequently, there will be a transient disruption in the balance between aggression and defense, in such a way that an acute inflammatory response is mounted in an attempt to re-establish the equilibrium.

Exacerbation of inflammation in response to extruded bacteria depends on their numbers and virulence. Thus, quantitative and qualitative factors are decisive in causing interappointment pain associated with apical extrusion of debris. The quantitative factor comprises the number of bacterial cells extruded, while the qualitative factor encompasses the virulence of the extruded bacteria. Virtually all instrumentation techniques promote apical extrusion of debris.[212–214] However, techniques significantly diverge as to the amount of extruded debris. Some techniques extrude less than the others. Such differences in the amount of extruded debris may be crucial for the development of postoperative pain. Techniques that extrude more debris conceivably increase the risk for exacerbations.

In the event of overinstrumentation during preparation of infected canals, the risk of flare-ups is increased. In addition to the mechanical injury to the periradicular tissues, overinstrumentation usually promotes extrusion of a large amount of infected debris.

Retreatment of teeth with posttreatment disease has been found to be a risk factor for postoperative pain.[206,209,215] During removal of the root canal filling material and further instrumentation, filling remnants and infected debris tend to be pushed ahead of the instruments and forced into the periradicular tissues, exacerbating inflammation and causing pain.[209,215] Solvents used during filling removal are also cytotoxic and may contribute to exacerbation of the periradicular inflammation.[216]

Incomplete Root Canal Preparation

Ideally, chemomechanical procedures should be planned to be completed in a single visit. The microbiota associated with endodontic infections are usually established as a multispecies community, and abrupt changes imposed to this consortium by instruments and antimicrobial irrigants have the potential to drastically affect the ecology of the microbiota. However, complete preparation is not always possible. The clinician should be aware that incomplete chemomechanical preparation can disrupt the community balance by eliminating some inhibitory species, leaving behind other previously inhibited species that can then overgrow.[217] If overgrown species are virulent and reach sufficient numbers, damage to the periradicular tissues can be intensified and result in exacerbation of inflammation.

Another form of environmental change induced by endodontic intervention refers to the entry of oxygen into the root canal. It has been suggested that this would alter the oxidation–reduction potential (Eh) in the root canal and, as a consequence, acute exacerbation might occur because of overgrowth of facultative bacteria.[218] However, this mechanism remains speculative and there is no scientific evidence indicating that it is valid.

Secondary Intraradicular Infections

Secondary infections are a contamination problem and can be a cause of flare-ups when it develops following a breach in the aseptic chain or coronal leakage. If the microorganisms that gained access to the root canal are successful in surviving and colonizing this new environment, a secondary infection is established and may be one of the causes of postoperative pain, providing the newly established species are virulent and reach sufficient numbers to induce acute periradicular inflammation.

ETIOLOGY OF POSTTREATMENT APICAL PERIODONTITIS

Microbial Factors

Persistent and Secondary Endodontic Infections

Persistent and secondary infections are two different conditions—the former is caused by bacteria that were in primary infections that persisted after treatment, while the latter is caused by bacteria introduced in the canal during

or after treatment. For the most part, these infections are clinically indistinguishable. Exceptions include infectious complications (e.g., acute 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. Persistent and secondary infections are responsible for several clinical problems, including persistent exudation, persistent symptoms, interappointment flare-ups, and failure of the endodontic treatment, characterized by a posttreatment apical periodontitis lesion (Figure 3-14).

Although there is evidence in the literature that extraradicular infection or nonmicrobial factors may be involved,[219,220] intraradicular infections (persistent or secondary) are the major causes of endodontic treatment failure (Figure 3-15). This statement 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 filling.[221–223] Second, studies have detected microorganisms in the canals of virtually all teeth with posttreatment apical periodontitis.[224–231]

Studies have been conducted to identify the species persisting after treatment that have the potential to influence the treatment outcome, and the species present in teeth with posttreatment apical periodontitis that may be the cause of this disease and consequently the cause of failure.

Bacteria Persisting After Treatment Procedures

In some cases, even diligent antimicrobial treatment may fail to properly control the root canal infection. This is because persisting bacteria can be located in areas that are inaccessible to instruments, irrigants, and medications. Inherent resistance to treatment procedures or substances might also be a cause of persistence, but it is difficult to assume that bacteria may resist to both mechanical debridement and the strong antimicrobial effects of sodium hypochlorite. Thus, bacteria usually persist because they are not reached by antimicrobial procedures. Following antimicrobial endodontic treatment, the intracanal bacterial diversity and density are substantially reduced. In cases where persisting bacteria are detected, one to five bacterial species are usually found per canal, with counts usually varying from 10^2 to 10^5 cells per sample.[64,66,222,232,233]

No single species has been significantly found to persist after treatment procedures. Gram-negative bacteria that are common members of primary infections, are usually eliminated. Exceptions include some anaerobic rods, such as *F. nucleatum, Prevotella* species, and *Campylobacter rectus,* which are among the species found in postinstrumentation or postmedication samples.[64,65,222,232,234,235] Most studies have demonstrated that persisting species are generally Gram-positive bacteria, including streptococci, *P. micra, Propionibacterium* species, *P. alactolyticus, Actinomyces* species, lactobacilli, *E. faecalis,* and *Olsenella uli* [64,189,222,232–244]. As-yet-uncultivated bacteria have also been detected in posttreatment samples.[64,163,245]

Persisting bacteria will not always induce or maintain an infectious process. Some apical periodontitis lesions can heal even in cases where bacteria were detected in the canal at the time of filling.[221,222] There are some possible explanations for this finding[246]:

- Residual bacteria died after filling, because of the toxic effects of the filling material, access denied to nutrients, or disruption of bacterial ecology;
- Residual bacteria were present in quantities and virulence subcritical to sustaining periradicular inflammation;
- Residual bacteria remained in locations where access to the periradicular tissues was denied.

Bacteria resisting the effects of intracanal procedures and present in the canal at the time of filling can influence the outcome of the endodontic treatment when[246]

- they managed to withstand periods of nutrient scarcity, scavenging for low traces of nutrients or assuming a state of low metabolic activity, to prosper again when the nutrient source is reestablished;
- they endured 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;
- they reached critical counts necessary to inflict damage to the host;
- they had unrestrained access to the periradicular tissues through apical/lateral foramina or iatrogenic root perforations;
- they were sufficiently virulent and antigenic to directly or indirectly induce damage to the periradicular tissues.

Microbiota in Root Canal-Treated Teeth

The root canal microbiota of teeth with posttreatment apical periodontitis exhibits decreased diversity in comparison to primary infections. Canals apparently well treated harbor one to five species, but the number of species in canals with inadequate treatment can reach up to 10 to 20 species that is very similar to untreated canals.[226–230,247] Bacterial density in root canal-treated teeth ranges from 10^3 to 10^7 cells per canal.[63,236,248,249]

E. faecalis has been the most frequently detected species in association with posttreatment disease, with prevalence values reaching up to 90%.[156,226–229,248–251] Because *E. faecalis* is not so common in primary infections[156] and has been commonly recovered from teeth treated at multiple visits and/or left open for drainage,[252] the possibility exists that this species is a component of secondary intraradicular infection.

E. faecalis has some characteristics that help explain its high prevalence in retreatment cases. Its ability to invade dentinal tubules can enable it to escape the action of endodontic instruments and irrigants used during chemomechanical preparation.[253,254] Its ability to form biofilms on

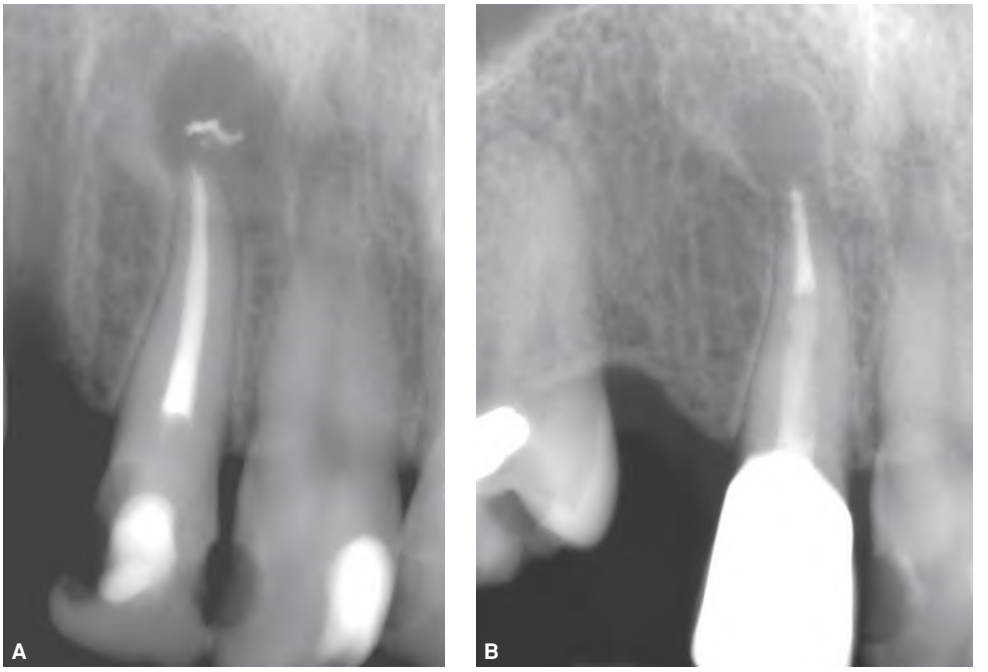

FIGURE 3-14 Lateral incisor with necrotic pulp and a large radiolucency. ***A***. Post-obturation radiograph. ***B***. Five-year recall. The lesion has slightly decreased in size. However, the residual radiolucency after a long observation period indicates root canal treatment failure.

the canal walls may be important for resistance to intracanal antimicrobial procedures.[255] *E. faecalis* shows resistance to calcium hydroxide,[256] possibly related to a functioning proton pump that, in an alkaline environment, drives protons into the cell to acidify the cytoplasm and allow survival.[257] Unlike most anaerobic bacteria frequently found in primary infections, *E. faecalis* may colonize the root canal in single infection.[228] Finally, this species can enter a so-called viable but non-cultivable (VBNC) state[258] that is a survival mechanism adopted by several bacteria when exposed to unfavorable environmental conditions.[259] This may be the case in treated canals. By the way, *E. faecalis* has been shown to survive in conditions of nutrient scarcity and then flourish again when nutrients are reintroduced.[260]

Its high prevalence and attributes do not suffice to prove a causative role for *E. faecalis* in posttreatment apical periodontitis. Its status as the main pathogen in failed cases has been questioned because of the following findings:

- *E. faecalis* is not detected in all studies evaluating the microbiota of root canal treated teeth with posttreatment disease.[159,261]
- If present, *E. faecalis* is rarely one of the most dominant species in the community associated with posttreatment disease.[230,247,249,262]
- *E. faecalis* is as prevalent in root canal treated teeth with lesion as it is in treated teeth with no lesion.[251,263]

Many other species have been detected in root canal-treated teeth with apical periodontitis, including *Streptococcus* species, *P. alactolyticus, Propionibacterium* species, *F. alocis, Dialister* species, *T. forsythia, P. micra, Prevotella* species, and *T. denticola.*[75,227–230,247,249,250,262,264,265] Uncultivated phylotypes can also be found and may correspond to 55% of the taxa.[230] Occurrence of uncultivated phylotypes may help explain the occurrence of negative cultures in studies evaluating the microbiota of teeth with posttreatment disease.

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.[183,184,227–229,236,250,261] *Candida albicans* is 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[266–268] and resistance to calcium hydroxide.[269,270]

EXTRARADICULAR INFECTIONS

In response to root canal infection, the host mounts an inflammatory response at the periradicular tissues to curb the advance of infection to the bone and other areas of the body. In some specific circumstances, bacteria egressing from the canal can overcome this defense barrier and establish an extraradicular infection. The acute apical abscess is the best and most common example of extraradicular infection. Other forms of extraradicular infection have been proposed as one of the possible etiologies of posttreatment apical periodontitis.[271–275]

An extraradicular infection can develop in the following situations[276]:

- As a result of direct advance of bacteria into the periradicular tissues due to an extension of the intraradicular infectious process. Sometimes, it can occur in the

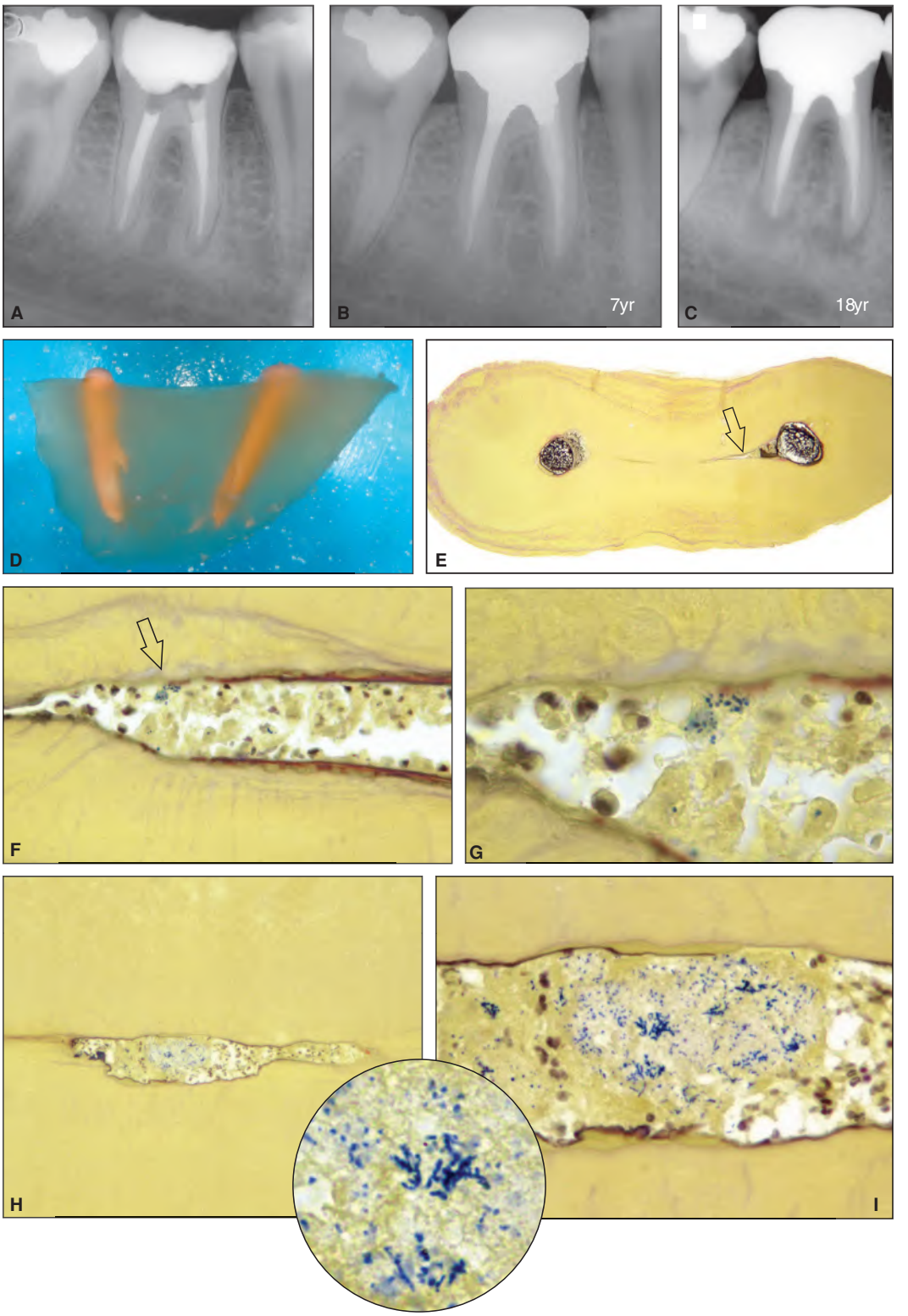

FIGURE 3-15 Mandibular molar with necrotic pulp. ***A***. Post-obturation radiograph. ***B***. Seven-year follow-up. Normal periapical conditions can be appreciated. ***C***. Eighteen-year follow-up. Radiolucencies are now present on both roots. Endodontic surgery was performed. ***D***. Apex of the mesial root after demineralization and immersion in clearing agent. ***E***. Crosscut section taken approximately at the point indicated by the line in D. An isthmus is present (Taylor's modified Brown & Brenn, original magnification ×16). ***F***. Portion of the isthmus indicated by the arrow in E. Inflammatory cells in different state of conservation. A small bacterial aggregation can be noted (original magnification ×100). ***G***. High power view (original magnification ×400). ***H*** and ***I***. Section cut 1 mm coronally to that in E. Progressive magnifications of the expansion of the isthmus halfway between the two mesial canals show necrotic tissue colonized by bacteria and surrounded by inflammatory cells (original magnification ×100 and ×400. Inset, ×1000).

form of a biofilm advancing along the outer root surface. Alternatively, advancing bacteria may penetrate the lumen of a pocket cyst that is in direct communication with the apical foramen, resulting in an infected cyst (Figure 3-16).

- As a result of bacterial persistence in the apical periodontitis lesion after remission of an acute apical abscess. In these cases, an actively draining sinus tract can be observed in a condition now recognized as a chronic abscess.
- As a sequel to apical extrusion of infected debris during root canal instrumentation (particularly after overinstrumentation) (Figure 3-17). Bacteria embedded in dentinal chips can be physically protected from the host defense cells and persist in the periradicular tissues to sustain periradicular inflammation.
- As a result of bacteria in the very apical part of the canal that are left outside after inflammatory apical root resorption associated with apical periodontitis.

Conceivably, the extraradicular infection can be dependent on or independent of the intraradicular infection.[272] Independent extraradicular infections are those no longer fostered by the intraradicular infection and can persist even after successful eradication of the latter. So far, it has been suggested that the main bacterial species implicated in independent extraradicular infections are *Actinomyces* species and *Propionibacterium propionicum,* in a pathologic entity named *apical actinomycosis.*[275,277–279] These bacteria may form cohesive colonies that are collectively resistant to phagocytosis.[280] However, existence of apical actinomycosis as a self-sustained

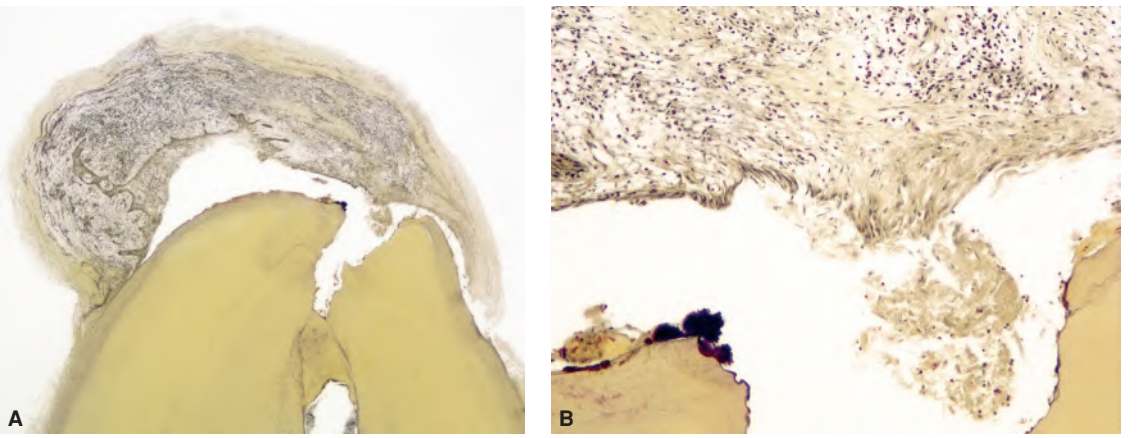

FIGURE 3-16 ***A***. The lesion remaining attached to this maxillary canine at extraction exhibits the characteristics of a "pocket cyst" (Taylor's modified Brown & Brenn, original magnification ×16). ***B***. Magnification of the foramen reveals that a bacterial biofilm is forming on the external surface of the root. This condition may sustain infection of the cyst cavity (original magnification ×100).

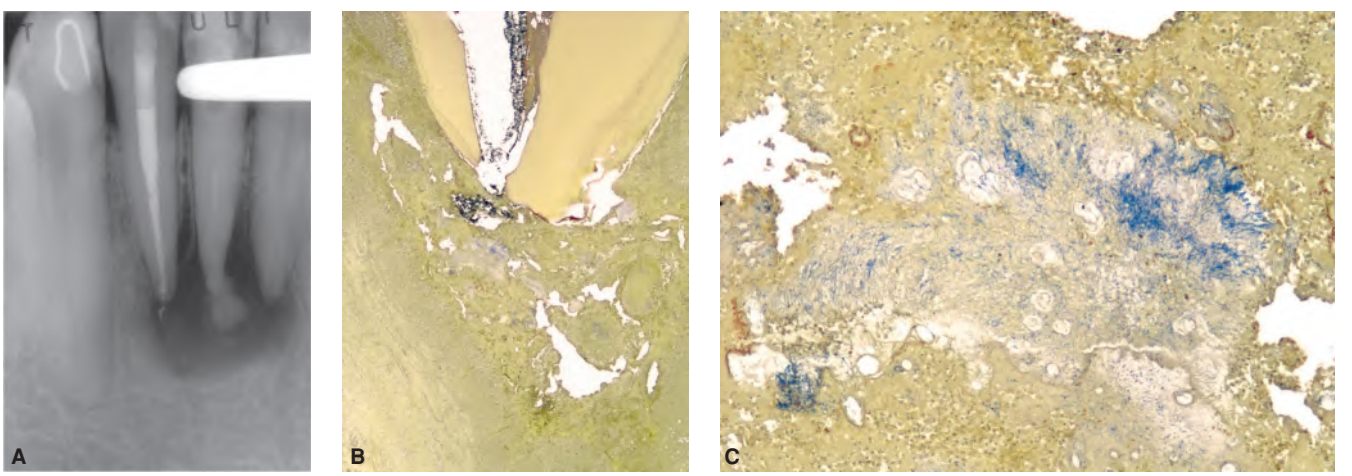

FIGURE 3-17 Mandibular incisor with necrotic pulp. ***A***. Radiograph taken after medication with calcium hydroxide revealed a considerable amount of the medicament inadvertently forced into the periradicular tissues. This may be an indication of overinstrumentation. Despite several sessions of instrumentation and intracanal medication, signs of persisting infection were constantly present. Permanent canal obturation was done and followed by perirapical surgery. ***B***. Longitudinal section cut through the foramen (Taylor's modified Brown & Brenn, original magnification ×16). ***C***. Magnification of the area of the lesion indicated by the arrow in B. A large bacterial colony with the characteristics of actinomycosis can be recognized (original magnification ×100). It is highly likely that this condition was caused by apical extrusion of infected debris as a consequence of overinstrumentation.

pathologic entity, no longer sustained by the intraradicular infection, and its involvement as an exclusive cause of treatment failure remains to be proven.[43,281]

It is still controversial whether extraradicular bacteria can establish themselves in an asymptomatic (chronic) apical periodontitis lesions for very long beyond initial tissue invasion.[282] Culture-dependent[219,283,284] or culture-independent molecular microbiology studies,[285–289] have reported the extraradicular occurrence of a complex microbiota associated with apical periodontitis lesions that did not respond favorably to the root canal treatment. Apart from a discussion about whether or not contamination can be effectively precluded during surgical sampling of periradicular lesions, these studies did not evaluate the bacteriologic conditions of the apical part of the root canal. This makes it difficult to ascertain whether those extraradicular infections were dependent on or independent of an intraradicular infection. Findings from a study evaluating both the resected root ends and the apical periodontitis lesions from treated teeth indicated that the large majority of cases of extraradicular infections are associated with concomitant infection of the apical root canal.[290]

NON-MICROBIAL FACTORS

As discussed above, numerous studies have demonstrated that the majority of teeth with posttreatment apical periodontitis are associated with intraradicular or extraradicular infections.[14,224,226,227,230,231,249,264,291] Because some studies failed to observe a possible microbial cause for this disease, it was suggested that endogenous or exogenous non-microbial factors may cause some lesions not to heal.[220] Endogenous causes purportedly include cholesterol crystals and true cysts, while exogenous causes encompass foreign body reactions caused by extruded endodontic filling materials[231,292,293] or cellulose-containing particles from paper points or food.[294,295]

Exogenous Factors

Some authors suggested that a foreign body reaction to the cellulose component of paper points, cotton wool, and some food material of vegetable origin may be the cause of persisting periradicular inflammation.[294,295] Cellulose is a stable polysaccharide of plant cell walls that is not degraded by the host defense cells. Once introduced into the tissues, cellulose may remain therein for long periods and elicit a foreign body reaction. Paper points or particles thereof can be dislodged or pushed into the periradicular tissues, inducing a foreign body giant cell response or sustaining an already existing inflammatory lesion. Cellulose-containing food particles may be impacted in the canals of teeth with grossly damaged crowns that have been left open for drainage, and be inadvertently pushed into the periradicular tissues during root canal treatment. It is salient to point out that in all these circumstances, it is virtually impossible to rule out the occurrence of concomitant infection (see Chapter 6), making it difficult to establish the cause of persisting inflammation. In the event of overfilling, the components of some filling materials may elicit a foreign-body reaction that has been claimed to sustain periradicular inflammation.[231,292]

Endogenous Factors

It has been suggested that cholesterol crystals can evoke a foreign body reaction and lead to persisting inflammation.[296] In areas of chronic inflammation associated with cysts and granulomas, cholesterol crystals can precipitate and accumulate as they are released from disintegrating host cells (see Chapter 6). They can also originate from circulating plasma lipids. If multinucleated giant cells are ineffective in removing these crystals, they continue to accumulate and may maintain chronic inflammation. Again, there is no consistent evidence indicating that cholesterol crystals are the cause of posttreatment apical periodontitis in the absence of concomitant infection.

"True" cysts that contain cavities completely enclosed by epithelial lining and not communicating with root canal, have been suggested to be self-sustaining entities that may not heal after root canal treatment.[297] However, it has never been proven that all apical cysts can be sustained without concomitant infection. Actually, some cysts are expected to heal after the cause of epithelial proliferation, that is, the root canal infection, is properly controlled.[298–300] The cyst epithelium loses the source of cytokines and growth factors that stimulate proliferation and serve as survival factors, leading to apoptosis of epithelial cells and resolution of the cyst.[300]

Pocket cysts are different from true cysts in the sense that the epithelial-lined cavity is open to the root canals. As a consequence of this direct contact between the lumina of the canal and the cyst, pocket cysts are more prone to be infected (Figure 3-16). Within the cyst cavity, bacteria egressing from the root canal are combated by host defense molecules and polymorphonuclear neutrophils that transmigrate through the epithelium into the cyst lumen. Because of the physicochemical conditions within the cyst cavity, the host defense mechanisms may not be effective in eliminating bacteria. Thus, persisting bacteria and their products within the cyst lumen may sustain periradicular inflammation in treated root canals.

Association of cholesterol crystals or true cysts with posttreatment apical periodontitis has not been observed in a study evaluating several cases of endodontic treatment failure.[231] In most studies that suggested a role for non-microbial agents, the microbiological conditions of the apical root canal was not assessed. Therefore, there is no consistent evidence regarding the role of cholesterol crystals as the sole cause of posttreatment apical periodontitis.

It is important to point out that endodontic infections and apical periodontitis are effectively treated by nonsurgical endodontic treatment with a very high success rate and the few failed cases can be managed by retreatment of periradicular surgery and converted to success in 80–90% of the cases. Therefore, endodontic treatment is an important and predictable approach to save teeth and promote oral health.

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Chapter 4

Inflammation and Immunological Responses

Ashraf F. Fouad, George T.-J. Huang

Endodontic pathosis is primarily caused by infectious agents that mediate a series of inflammatory and immunological responses in the dental pulp and periapical tissues. This chapter describes some of the fundamental immune reactions that take place in these tissues, as well as other connective tissues, and outlines the basic mechanisms involved in disease initiation and progression.

The science of immunology is constantly changing and remarkable discoveries are constantly being made about the unique molecules, cells, tissues, and pathways that take place in response to external and autogenic stimulation. While an attempt has been made to address most of the critical and well-understood immunological data, it is virtually impossible to fully cover this topic in a comprehensive manner in this chapter. Many immunological reactions have not been adequately emphasized in the endodontic literature, or may exceed the scope of this textbook. These include neoplastic changes, autoimmune reactions, and some congenital and post-natal abnormalities in gene expression. Indeed, the reason why neoplastic changes are not common in the dental pulp may be of fundamental importance in understanding the pathogenesis of neoplasia in general, and may have relevance in the growing field of regenerative endodontics, particularly in gene therapy. However, given the paucity of data in some of these areas, and the speculative nature of the data that is available, this material may not be suitable for publication in clinical textbooks at this time. Therefore, these topics will not be addressed in this chapter, and the reader is referred to specialized textbooks or journals for more information on these topics.

The reactions and mechanisms that will be covered here are those that primarily deal with the inflammatory response, and that have been described to some degree in pulpal and periapical tissues or are widely recognized in other tissues and presumed to be in operation in endodontic tissues. In fact, wherever relevant, specific literature that have addressed these questions will be cited in the text, to bring the basic concepts closer to the mind of the reader, who is assumed to be primarily a clinician or a researcher in the endodontic field.

The material described in this chapter will be presented under discrete topics, for example, the individual cells and molecular mediators encountered in immunological reactions. However, the reader should always be cognizant of two fundamental concepts in immunology. The first is that there are many interrelationships between the pathways and mechanisms that will be presented, and much overlap among the functions of the cells is to be described. This creates a large amount of redundancy that assures a competent and robust response. Indeed, it is commonly stated that the main purpose of treatment of various disease processes is to remove the source of irritation and provide a suitable environment for the body to cure itself, through the effective healing mechanisms. The second concept is that there is a substantial degree of variability among different individuals in their response to a particular irritant. While some of this difference in response is quite extreme, and explained by the various hypersensitivity reactions, most of the variation is more subtle, and is mediated by systemic conditions and diseases, or individual genetic polymorphism of different patients. The concept of genetic polymorphism commonly refers to differences in expression of certain inflammatory mediators, cellular activation, or other immunological reactions. This variation may indeed render certain individuals more prone to a more dramatic expression of the disease process or delay in healing mechanisms. Genetic polymorphism may be explained by minor, perhaps a single nucleotides variation in the genetic make-up of particular genes. Thus, it is commonly referred as a single nucleotide polymorphism or SNP. It has been described with respect to the expression of a number of proteins, such as interleukin (IL) 1, and may explain why some inflammatory reactions to seemingly similar irritants, undergo different clinical presentations and response to treatment in different patients.

CELLS OF THE IMMUNE SYSTEM

The immune system represents a complex system of cells, tissues, organs, as well as molecular mediators that act in synchrony to assure the maintenance of health and the defense against disease. Cells interact with microbial irritants as well as with each other via a large number of molecular mediators and cell surface receptors that together result in the various phenotypic reactions. The various host reactions will be discussed in the context of a description of the main cells of the immune response, as these cells play a critical role in mobilizing the immune response. It is appropriate initially to present the ontogeny of immune cells from hematopoietic stem cells (Figure 4-1).[1–3] A description of the mature inflammatory cells and their activities is given below, starting with the innate immune system.

Innate Immune System

The innate immune system is composed of a number of natural barriers, as well as cellular and molecular elements that represent the initial nonspecific reactions of the immune response. The barriers are epithelial or generally ectodermal

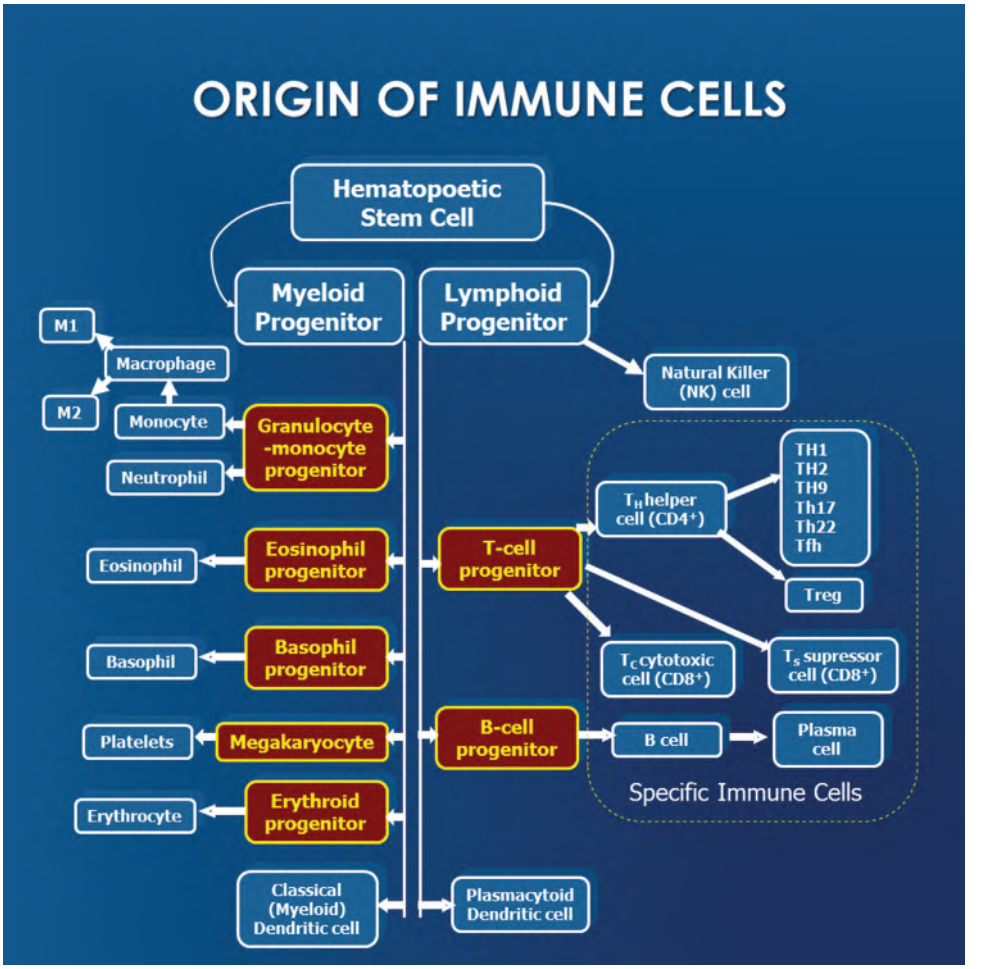

FIGURE 4-1 Hematopoiesis of principal immune cells. Note that dendritic cells may develop from both myeloid and lymphoid progenitor cells. Cells of the specific immune system develop memory clones for future interactions with the similar antigens.

TABLE 4-1 Normal Blood White Cell Counts[3]

	Mean count per microliter	Normal range
Total leukocytes	7400	4500–11,000
Neutrophils	4400	1800–7700
Eosinophils	200	0–450
Basophils	40	0–200
Lymphocytes	2500	1000–4800
Monocytes	300	0–800

or ectomesenchymal in origin. Examples include the skin, oral mucosa, junctional, and sulcular epithelium, as well as intact enamel and dentin. These barriers prevent the dissemination of microorganisms and their byproducts into the subjacent connective tissue that would result in disease. Dental caries, fractures of teeth, or congenital anomalies that expose the dental pulp create a break in this barrier, resulting in inflammation of the pulp, and ultimate necrosis. Once the pulp is necrotic and the pulp space is occupied by microorganisms, there is no natural barrier to the dissemination of these irritants into the periapical region and/or systemically through the apical, lateral, or accessory foramina. In fact, junctional complexes between odontoblasts in the vital pulp have been shown to play a role in preventing the leakage of microbial irritants into the pulp.[4,5]

Phagocytosis is another important component of innate immunity. Similarly, the presence of certain non specific proteins such as, lysozyme or complement, or pattern-recognition receptors, such as Toll-like receptors (TLRs) (see later), on the surface of immune cells are also constituents of the innate immune system. Inflammation is associated with a number of vascular changes such as vasodilatation, increased capillary permeability, and extravasation of blood cells and inflammatory mediators, together with reduced pain threshold. Many of these non specific changes are caused by neurogenic elements, such as neuropeptides. These concepts will be addressed later in the chapter. Therefore, innate immunity does not involve sensitization or priming, has a uniform response to all irritants that are detected, lacks memory, and aims to rapidly eliminate irritants and degenerated host tissues.

Cells of Innate Immunity

Neutrophils Neutorophils, also known as polymorphonuclear leukocytes or PMNs, are the most numerous white blood cells (Table 4-1), representing ~54%–63% of peripheral blood leukocytes. They have a short half-life, generally functioning

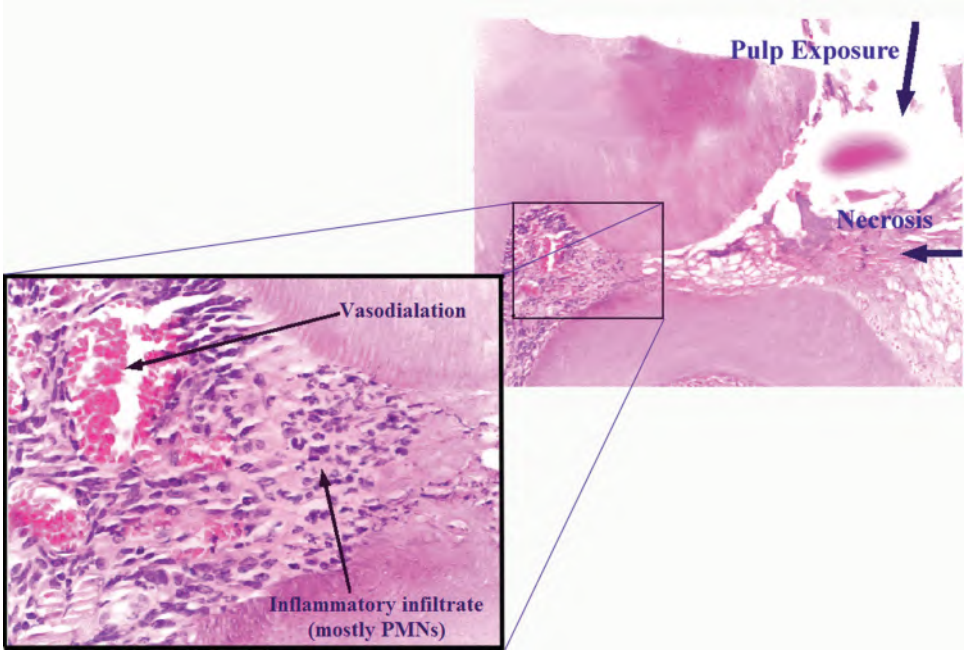

FIGURE 4-2 Experimental pulp exposure in a rodent molar showing pulp necrosis, acute and chronic inflammation, and vasodilatation.

for 1–2 days, and if not recruited to a site of inflammation, they undergo apoptosis. PMNs represent the first defense line against microbial irritants, being mobilized in what is traditionally known as acute inflammation (Figure 4-2). PMNs utilize a large number of defense mechanisms to eliminate invading pathogens and inactivate their toxins. These include the production of lysosomal enzymes, cytokines, oxygen-derived free radicals, as well as phagocytosis.

Within PMNs, defense proteins are contained within two types of granules that do not stain acidic or basic, that differentiate these cells from basophils and eosinophils. The first granule is azurophilic (large) granuales (lysosomes) (~0.5 μm in diameter) that contain lysozyme, myeloperoxidase, defensins, acid hydrolases, and neutral hydrolases such as collagenases, elastase, cathepsin G, and other proteinases. The second type is specific (small) granules (~0.2 μm in diameter) that contain lactoferrin, lysozyme, collagenases, plasminogen activator, histaminase, alkaline phosphatase, and membrane-bound cytochrome b_{558}. Production of neutrophils is stimulated by granulocyte colony-stimulating factor (G-CSF). An adult human produces >1011 neutrophils every day.[3] The cell also has abundant glycogen stores, that enable the cell to utilize glycolysis to produce energy in anaerobic conditions,[6] such as during an abscess formation.

Monocytes/Macrophages Monocytes are circulating leukocytes that represent 4%–8% of blood leukocytes. Macrophages reside within connective tissues. Certain macrophages become especially adapted for immunological functions in certain tissues. These cells are collectively referred to as members of the mononuclear phagocyte system. Examples of these cells are Kupffer cells in the liver, microglia in the central nervous system, or alveolar macrophages in the lungs. It is generally accepted now that both macrophages and osteoclasts have a common lineage. Likewise, a number of other cells commonly seen in inflammatory reaction or granulomatous disease, such as foam cells, epithelioid cells, or giant cells, may have a common lineage.

Macrophages have a relatively long half-life in tissues, commonly measured in months. They are considered among the principal inflammatory cells, commonly mobilized in areas of chronic inflammation. They possess major histocompatibility complex (MHC)-II receptor, have antigen-presenting capabilities, and are involved in phagocytosis. They are also among the principal producers of pro-inflammatory cytokines such as IL-1, IL-6, TNF-α, and IL-12.

Macrophages may be activated by activated Th1 cells of the specific arm of the immune response, or nonspecifically, such as by natural killer (NK) cells (Figure 4-3). There are two types of macrophages: M1 and M2. M1 macrophages express surface receptors, known as pattern-recognition receptors (PRRs). These receptors identify pathogen-associated molecular patterns (PAMPs) such as lipopolysaccharide (LPS) of Gram-negative bacteria, or peptidoglycan (PG) and lipoteichoic acid (LTA) of Gram-positive bacteria. PRRs generally belong to the TLRs, or nucleotide binding oligomerization domain (NOD)-like receptors (see later), and have intracytoplasmic component (homologous with the IL-1 receptor molecule) that transmits the signal through transduction proteins like myeloid differentiation factor 88 (MyD88) and transcription factors such as nuclear factor kappa-B (NF-κB) and other mitogen-activation protein (MAP) kinases. Eventually, messenger RNA is translocated into the nucleus in order to produce certain pro-inflammatory

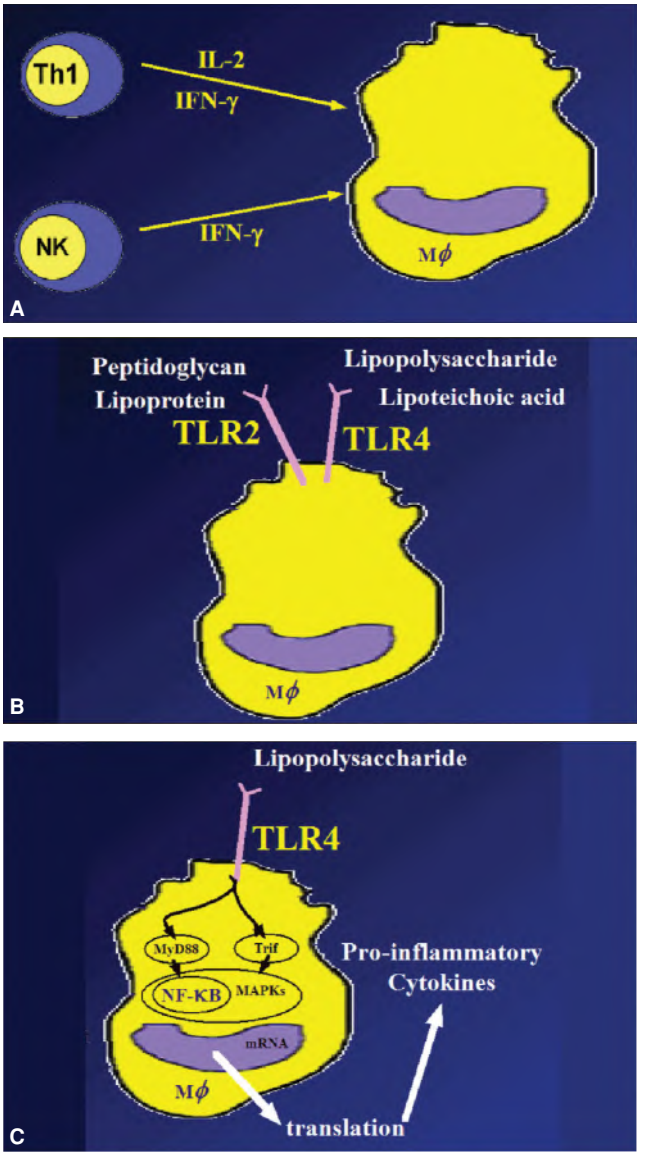

FIGURE 4-3 Macrophage activation and function. ***A.*** Cell is activated by cytokines from specific and non-specific immune cells. ***B.*** Cell expresses various pattern recognition receptors to microbial molecules. ***C.*** Signal transduction through myeloid differentiation factor 88 (MyD88) or other intermediaries, transcription through nuclear factor kappa-B (NF-κB) and mitogen-activated protein (MAP) kinases, that result in translation of mRNA and production of pro-inflammatory cytokines.

proteins, such as the cytokines, IL-1, IL-6, and TNF-alpha. The importance of these factors in inflammatory periapical lesions is demonstrated by the fact that all lesions (but not controls) were shown to have NF-κB.[7] More recently, periapical lesions were induced in MyD88-knock out mouse models and controls. Lesions in the knock out mice had much larger size, extensive neutrophils and monocyte counts, and tissue degradation, indicating a more extensive infection due to the down regulation of the inflammatory response.[8]

Molecules such as LPS are very potent biological toxins, capable of causing septic shock and killing the host. Therefore, in addition to the membrane-bound TLRs, a number of circulating molecules such as CD14 are available, that can bind to LPS in the blood rendering it less biologically active. This reaction is mediated by a plasma protein called LPS-binding protein (LBP). CD14 is also available in a membrane-bound form to the surface of macrophages and can bind LBP in this location; however, unlike TLRs, CD14 has no intracytoplasmic component, and thus is not capable of initiating the signal transduction pathway to initiate the production of cytokines. M2 macrophages are generally activated by IL-4 and IL-13, and are involved in tissue repair and control of the inflammatory response.[9] Recently, they have been shown to play a role in pulpal repair following vital pulp therapy.[10,11]

Phagocytosis The PMNs and macrophages are the main cells involved in phagocytosis. The surface of phagocytes can bind to the pathogen surface pattern molecules by lectin, through complement C3b or C5b, or by binding to the Fc portion of an opsonizing antibody through the FcγR receptors, which bind the carboxyl-terminal constant region of IgG molecules (Figure 4-4). These receptors include the high-affinity FcγRI receptor present in macrophages and PMNs, a low-affinity FcγRIIB that transduces inhibitory signals in B-cells and a low-affinity FcγRIIIA that mediates targeting activation of NK cells.[9] Studies in the endodontic literature have shown that healing following endodontic treatment was associated with genetic polymorphism in some alleles for the genes of FcγR receptors, such as FcγRIIA and FcγRIIIB,[12] but not FcγRIIIA.[13] After the initial binding of pathogen, which is significantly enhanced by opsonins, pseudopodia develop to surround and engulf the pathogenic organism, leading to the formation of a phagosome. The final stage of phagocytosis involves lysosomal enzymes that are released into the phagosome for degrading the microorganism or other foreign particles.

Microbial killing within the phagosome is effected through a series of oxidative reactions that require glycolysis, glycogenolysis, and the generation of reactive oxidative free radicals (Figure 4-5).[14] The fusion of the membranes of a phagosome and the lysosomal granules of the cells (such as the azurophilic granules of PMNs) results in an oxidative burst mediated by cytoplasmic- and membrane-bound oxidases. Through the action of NADPH oxidase, superoxide ions and hydroxyl radicals are generated. While these are sufficiently antimicrobial, lysosomes of PMNs contain myeloperoxidase, which through a halide intermediate can result in the generation of hypochlorous radicals (HOCl•). As is the case with exposure to sodium hypochlorite (NaOCl), HOCl• is a powerful oxidant and antimicrobial agent that kills bacteria by halogenation, or by protein or lipid peroxidation. Within the phagosome, other mechanisms of bacterial killing include lysozyme, which causes degradation of bacterial coat oligosaccharides, and defensins, which are peptides that kill bacteria by forming holes through their membranes.[14]

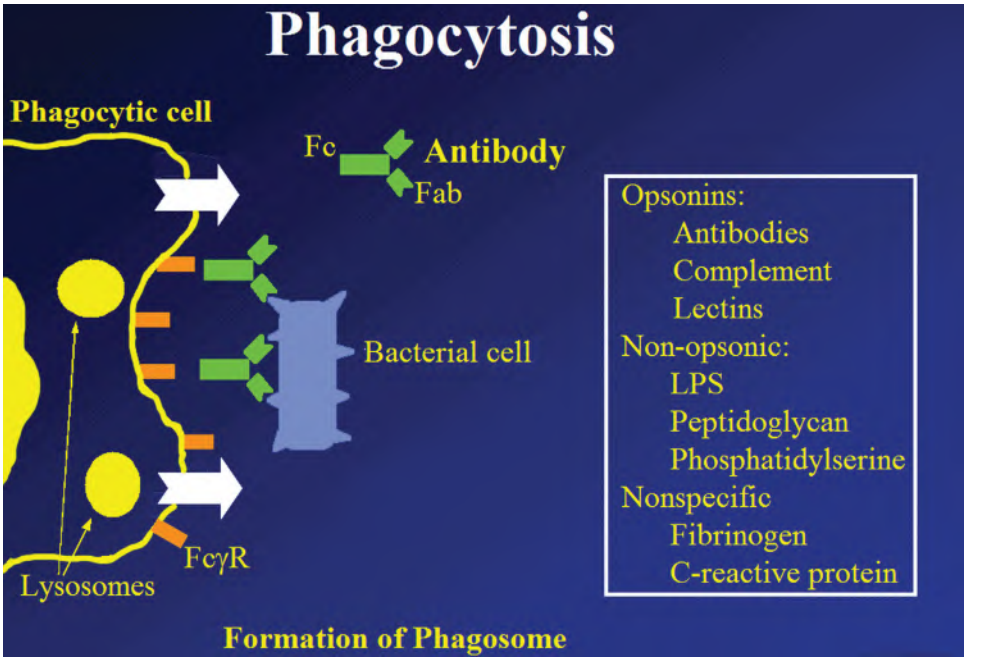

FIGURE 4-4 Phagocytic cell recognizes an opsonized bacterial cell, and starts to form a phagosome (white arrows) to engulf it. Eventually, lysosomal enzymes open into the phagosome to digest the material.

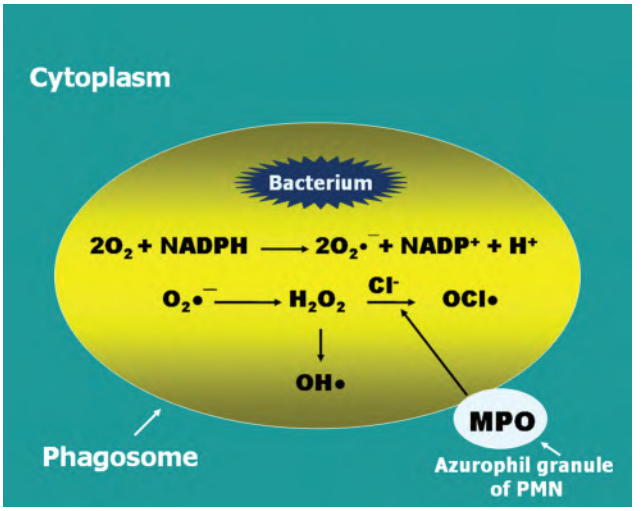

FIGURE 4-5 Oxidative burst reactions within a phagosome that result in destruction of a pathogen. MPO: myeloperoxidase; NADPH: reduced nicotinamide adenine dinucleotide phosphate; O2•⁻: Superoxide radical, OH•: hydroxyl radical; OCl•: hypochlorous radical.

While phagocytosis is quite effective in eliminating microbial irritants, when they are isolated or individualized, it frequently cannot deal with a microbial biofilm. A microbial biofilm has a large number of microorganisms embedded in a polysaccharide matrix. An example that is relevant to endodontics is a periapical infection with *Actinomyces* spp. that is generally thought to occur in the form of microbial colonies within the lesions. Inflammatory cells usually surround these colonies and attempt to eliminate them by forming phagosomes, but instead result in the release of oxygen radicals and lysosomal enzymes into the periapical tissues, causing tissue degradation.

Eosinophils Like neutrophils, eosinophils are segmented leukocytes, however, they stain red with hematoxylin and eosin stains (thus the name). They are much less prevalent than PMNs, constituting only ~1%–3% of circulating leukocytes. They are involved in allergic and some parasitic reactions. Their actions are primarily mediated by a low-affinity surface receptor for the Fc portion of the IgE molecule called FcεRII. Eosinophils express a number of inflammatory and healing mediators, most notably transforming growth factors (TGF) alpha and beta-1.[15–17] Eosinophils in human periapical cysts were shown to express TGF-α, and those in both cysts and granulomas also had TGF-β1, at the mRNA and protein levels.[18]

Basophils Basophils share some similarities in shape, size, and life-span with neutrophils and eosinophils; however, they are more intimately involved in the mediation of allergic reactions. In this regard, they share some features with connective tissue mast cells (Table 4-2).

TABLE 4-2 Comparison of Mast Cells and Basophils

	Mast Cells	Basophils
Size	10–15 μm	5–7 μm
Location	Connective tissue	Blood
Life span	Weeks or months	Days
Terminally differentiated	No	Yes
Granule content	Histamine, heparin, PAF	Histamine, heparin, PAF
High-affinity FcεRI	Yes	Yes

Mast Cells As noted in Table 4-1, mast cells reside in connective tissues for weeks to months. They are generally dormant with little activity, until they are stimulated by IgE activity in type 1 hypersensitivity reactions (see below). Complement activation also results in the production of C3a and C5a that are both described as anaphylatoxins as they initiate the acute responses effected by mast cell degranulation. Mast cell degranulation results in local (atopic) or systemic (anaphylactic) reactions. The primary outcomes of these reactions are vasodilation and broncho-constriction. Anaphylactic reactions can be life threatening, because of the resulting apnea and reduction in peripheral circulatory tone that dramatically reduces the blood pressure. These reactions are mediated by the release of granule contents, namely, histamine, arachidonic acid metabolites (especially prostaglandins: PGE2 and PGI1, and leukotrienes: LTB4 and LTD4), platelet activating factor (PAF), eosinophil chemotactic factor A, and a number of other enzymes that cause tissue damage.

Dendritic Cells Dendritic cells are the most important antigen presenting cells for activating naïve T-cells, thereby playing a critical role in linking innate and adaptive immunity.[3] Dendritic cells are stellate-shaped and reside in a variety of tissues including blood, lymph, epidermis, dermis, and secondary lymphoid organs.[19] They lack long-term adherence to immune cells or molecules and are not involved in phagocytosis. Like other antigen-presenting cells (APCs), namely macrophages, Langerhans cells (a subset of dendritic cells [DCs] present in the epidermis), B cells, and endothelial cells, they express MHC II. They have a variety of phenotypes, as determined by their morphology, surface markers, and the tissue where they reside. Circulating blood DCs have two different origins: classical (myeloid) and plasmacytoid. The former is characterized by the CD11c+ marker, while the latter lack CD11c, but have the CD123+ marker.[19] Classical CD11c+ DCs, such as macrophages, express a large number of TLRs, to both bacterial and viral molecules. In contrast, plasmacytoid DCs do not express TLR-2 and -4, and thus do not respond to bacterial molecules.[20]

They are also present in the dental pulp, where they are prevalent among the odontoblastic cell layer and around blood vessels,[21,22] in periodontal ligament[23] and in periapical lesions.[23,24] In mouse models, pulpal CD11b (the equivalent of human CD11c) DCs have been shown to migrate to regional lymph nodes in response to dentinal irritation, to present detected antigens to T-cells.[25]

NK Cells NK cells are lymphocytes that do not require activation like CD4 or CD8 lymphocytes. Therefore, they are members of the innate immune response. They represent ~4%–20% of circulating mononuclear cells. They produce interferon (IFN)-γ and are thus capable of activating macrophages to produce pro-inflammatory cytokines, without the need for activated T-lymphocytes. NK cells are particularly effective in killing host cells infected with viruses or other intracellular microorganisms.

Odontoblasts and Innate Immunity Odontoblasts are the first line of cells encountering foreign antigens after enamel and dentin breakdown, these resemble epithelial cells in this concept. Epithelial cells have been considered as an integrate part of innate immunity for being capable of producing pro-inflammatory cytokines and expressing antimicrobial peptides β-defensins.[26,27] Similarly, human odontoblasts have also been observed to express chemokines such as IL-8 and CCL20, chemokine receptor CCR6, as well as antimicrobial peptides β-defensins 1, 2, and 3.[28-31] Furthermore, odontoblasts express TLR including TLR2, TLR4, and virus recognizing TRLs 3, 7, 8, and 9,[30,32-34] indicating that odontoblasts plays a significant role in the pulp innate immunity.

Cells of Adaptive Immunity

The adaptive immunity involves the development of specific receptor molecules made by lymphocytes that recognize and bind to foreign or self-antigens. These specific receptor molecules on T cells are called T-cell antigen receptors (TCRs) and on B cells are B-cell antigen receptor (BCRs) or immunoglobulins. TCRs on T cells interact with antigens presented by MHC molecules along with other accessory molecules, whereas BCRs on B cells or as secreted form, generally known as antibodies, interact with antigens directly. The variable regions of both TCR and BCR are rearranged at the genomic level via V(D)J segment recombination. The estimated total diversity after this recombination for TCR is ~10^{18}, and for BCR is ~10^{14} that generates the repertoire of different individual T and B cell clones.[3,35]

Innate Lymphoid Cells Innate lymphoid cell (ILCs) not only play important roles in the development of lymphoid tissues and the initiation of inflammation in response to infection at the barrier surface, they also play complex roles throughout the duration of immune responses, participating in the transition from innate to adaptive immunity and contributing to chronic inflammation.[36] ILC family includes three groups, each predominantly express certain cytokines. ILC1s: IFN-γ; ILC2s: IL-5, IL-9, and IL-13; and ILC3s: IL-22 and/or IL-17. ILC2s are important in driving type 2 immune responses (Th2), while other members of the ILC family play roles in type 1 (Th1) and Th17 cell-mediated immunity and disease. Together they play roles in protective immunity against microbes, autoimmune disorders, and allergic diseases.[36]

T Cells T cells are found in normal pulp and constitute ~30% of the $CD45^+$ cells present in the pulp.[37,38] They play a pivotal role in adaptive immunity and their development goes through a rather intricate process. Originated from bone marrow hematopoietic stem cells, the precursor T cells migrate to the thymus to undergo maturation in which they express different cell surface markers (e.g., CD44, CD3, CD4, CD8, and CD25) at various stages and are screened via positive and negative selection processes. About 97% of T cells undergo apoptosis and only a small percentage of these cells are exported to the periphery

as mature T cells. During the positive selection, double-positive $CD4^+CD8^+$ T cells bearing TCRs that can interact with self-peptide: self-MHC on thymic epithelial cells are rescued from apoptosis. In the negative selection, these $CD4^+CD8^+$ T cells recognizing self-peptide: self-MHC too well are deleted via apoptosis. The positive selection ensures that mature T cells can recognize foreign antigen in the context of self MHC molecules that present the antigen, whereas the negative selection eliminates potentially self-reactive T cell clones. Before exiting the thymus, these cells become single-positive either $CD4^+$ or $CD8^+$ along with other surface markers such as CD25. These naive T cells then circulate back and forth between the lymphatic system and blood until they encounter foreign antigens presented by antigen presenting cells.

The interactions between TCR and antigen peptide: MHC and between co-stimulators CD28 and B7 activate signal transduction pathways in T cells leading to the synthesis of T cell growth factor IL-2 and its receptor and T cell clonal expansion/proliferation. Subsequently, T cells differentiate into armed effector T cells and some become memory cells. There are a number of T cell subpopulations categorized by their functions and some, can be distinguished by their cell surface markers, cytokine profiles, or transcriptional factors. Four main subpopulations are described below: T-helper cells, T-regulatory cells (Treg), T-suppressor, and T-cytotoxic (cytolytic) cells.

T-helper Cells (Th Cells) Naïve CD4 T cells upon antigen stimulation proliferate and differentiate into subsets of Th1, Th2, Th9, Th17, Th22, T-follicular helper cells and different subsets of regulatory T cells.[39] Th0, that subsequently commit into Th1 or Th2 cells. Classical (myeloid) DCs induce T-helper (Th) 1-type responses, and plasmacytoid DCs selectively induce Th2 responses. Each subset has distinct functions and cytokine profiles as depicted in Table 4-3. A Th1-specific T box transcription factor, T-bet, suppresses early Th2 cytokine production and induces Th1 T cell development via up-regulation of *IFN-γ* gene transcription and IL-12Rb2 chain expression. The differentiation of Th2 is dependent on the induction of transcription factors STAT6, c-maf, and GATA-3.[40] Th1 cells mainly produce IL-2 and IFN-γ; activate macrophages and induce B cells to produce opsonizing antibody. Th2 cells produce IL-4, -5, -10, and -13; activate B cells to make neutralizing antibody; have various effects on macrophages. Overall, Th1 and Th2 have mutually modulatory effect.[40]

Th9 Cells Th9 cells are a recently described new helper T-cell subset; the signature cytokine for Th9 cells is IL-9 (without IL-4).[41] Th9 cells are closely associated with Th2 cells, which co-express both IL-4 and IL-9 in the early phase of Th2 differentiation, and IL-4 is a key signal for Th9 induction.[41] Th9 cells play a role in allergic lung inflammation and certain autoimmune diseases.[41]

Th17 and Th22 Cells Th17 and Th22 cells are two emerging Th cell subsets that link the immune response to tissue inflammation; IL-17A, IL-17F, and IL-22 are their respective prototype cytokines.[39] Th17 cells are closely associated with Th1 in diseased tissues and may be directly derived from naïve CD4+ T cells.[42-44] Th17 produces IL-17 and its development can be induced by IL-23, which also stimulates Th1 production of IL-17-a cytokine that is linked to autoimmune and inflammatory diseases including rheumatoid arthritis and lupus. Th22 cells are characterized by particularly high IL-22 production. As a member of the IL-10 family, IL22 has 22.8% identity to IL-10.[39] Although both cytokines play roles in immune defense to extracellular bacteria, IL-17 augments inflammation, whereas IL-22 plays a role in recovery and regeneration.[39]

T follicular Helper Cells Generation of T follicular helper (Tfh) cells is similar to other CD4+ T cell lineages (e.g., Th1, Th2, Th17, and Treg cells) requiring signaling pathways. Tfh cells express a unique combination of effector molecules including high levels of the surface receptors ICOS, CD40 ligand (CD40L), OX40, PD-1, BTLA and CD84, the cytokine IL-21, the cytoplasmic adaptor protein

TABLE 4-3 Subpopulations of T Cells

Subtype	Main CD	Key cytokine profile	Transcriptional factors*	Main function
Th0	CD4			Challenged naive T cells
Th1	CD4	IFN-γ, IL-2	T-bet	Pro-inflammatory
Th2	CD4	IL-4, 5, 10, 13	GATA-3	Anti-inflammatory
Th9	CD4	IL-9	PU.1, IRF4	Allergy
Th17	CD4	IL-17	PORC2	Pro-inflammatory
Th22	CD4	IL-22	–	Promote healing
Tfh	CD4	IL-21	Bcl-6, c-Maf	Helper to B cells
Tr	CD4, CD25	IL-10	Foxp3	Anti-inflammatory
Tr-1	CD4, CD25	TGF, IL-10	–	Anti-inflammatory
Th3	CD4, CD25	TGF	–	Anti-inflammatory
Ts	CD8		–	Anti-inflammatory
Tc	CD8		–	Kill virus-infected cells

*Transcriptional factors are identified as important markers for the respective T cell subsets.

SLAM-associated protein (SAP), and the transcription factors Bcl-6 and c-Maf. These molecules play critical roles in promoting activation, differentiation, and survival of B cells and/or CD4+ T cells.[45]

T-regulatory Cells Treg cells have the cell surface markers CD4 and CD25 and therefore are generally named $CD4^+CD25^+$ Treg. They play an essential role in mediating immunosuppression and maintenance of tolerance. These T-cell clones that may react to self-antigens but are not deleted via negative selection in the thymus are considered to be suppressed by Treg cells in the periphery. Therefore, dysregulation of Treg function may elicit autoimmune diseases. There are subtypes of Treg cells based on their origin and the mechanisms of action and function.

Treg Cells or Natural Occurring (Tr) Natural occurring $CD4^+CD25^+$ Tr cells arise either in the thymus and reside in the blood and peripheral lymphoid tissues, or they can also arise in the periphery induced by antigen (Ag). Human Tr potently suppress the proliferation and effector functions in a cell contact-dependent manner or by acting on antigen presenting cells.[46,47] The transcriptional factor FoxP3 is important to their development and a marker. Human Tr can be further divided into two subsets with distinct functions based on differential expression of integrins. α4β7 integrin expressing Tr cells induce *de novo* differentiation of IL-10-producing Tr1 cells. α4β1 expressing Tr cells induce the differentiation of TGF-β-producing Th3 cells. The ratio of Th17 (pro-inflammatory) to Foxp3+ Treg (immunosuppressive) T-cells was found to increase during initial induction of periapical lesions, and decrease at later stages, indicating the importance that these two cell populations play in regulating periapical lesions.[48] Furthermore, the gene methylation (epigenetic changes that do not relate to the gene sequence) of the Foxp3 gene promoter were recently found to correlate with reduced activity in human periapical lesions.[49]

Tr1 Cells ($CD4^+$ Type-1 Treg) Tr1 cells arise in the periphery upon encountering antigen (Ag) presented by tolerogenic DCs, which requires IL-10. Tr1 cells produce high levels of IL-10 themselves, and mediate IL-10-dependent suppression of T cell response. In contrast, Tr cells do not produce IL-10. Defining Tre1 phenotype currently is solely based on cytokine production—IL-10, but not IL-4 and they express variable CD25. Tr1 cells regulate the response of naïve and memory T cells in vitro and in vivo and can suppress both Th1 and Th2 cell-mediated pathologies. Tr1-cell clones suppress the production of immunoglobulin by B cells. The major function of Tr1 cells is to control homeostasis of the response to foreign antigens in the periphery.[47]

Th3 Cells Although Th3 cells are named under T helper cells, they are grouped with Tr cells due to their close functionality to Tr-1 cells. They are $CD4^+$ T cells, which produce TGF-β. Similar to Tr1 cells, these T cells are generated in the periphery induced by oral antigen in the mesenteric lymph nodes. They express variable CD25 and produce TGF-β and varying amounts of IL-4 and IL-10. They also inhibit Th1- and Th2-mediated adaptive immune responses. It is speculated that Tr1 and Th3 cells derive from the same population and perhaps represent adaptive Tregs, as they have a similar phenotype and usually mediate their suppressive activities via the release of IL-10 and/or TGF-β (Figure 4-6).[50,51]

T Suppressor ($CD8^+$ T Suppressor Cells or Ts) There is a controversy regarding the recognition of $CD8^+$ T suppressor cells as a distinct subset of $CD8^+$ T cells. There is also a disputable hypothesis that $CD8^+$ Ts cells are merely cytotoxic T lymphocytes ($CD8^+$ CTLs) based on the finding that both $CD8^+$ Ts and CTL play roles in immunosuppressive activities and maintenance of tolerance. Evidence has suggested that $CD8^+$ Ts and $CD8^+$ CTLs represent distinct subpopulations of $CD8^+$ T cells. Human $CD8^+CD28^-$ T cells have also considered another subtype of Ts cells. These cells are MHC-class I-restricted $CD8^+CD28^-$ Ts cells that act on APCs in a contact-dependent manner, rendering them tolerogenic to Th cells. They inhibit the proliferation of Th by blocking the activation of APC and preventing the upregulation of co-stimulatory molecules required for efficient help. CD8+CD28– Tregs can block the upregulation of co-stimulatory molecules on APCs such as CD80, CD86, CD54, and CD58. Suppressor function could not be abrogated using neutralizing antibodies against IL-4, IL-10, TGF-β, and CTLA-4. In contrast to the suppression of co-stimulatory molecules, CD8+CD28+ Treg have been demonstrated to upregulate the inhibitory immunoglobulin-like transcript 3 (ILT3) and ILT4 receptors on DCs. Taken together, these results suggest that CD8+CD28– Tregs suppress immune responses by directly interacting with DCs and rendering these cells tolerogenic.[52]

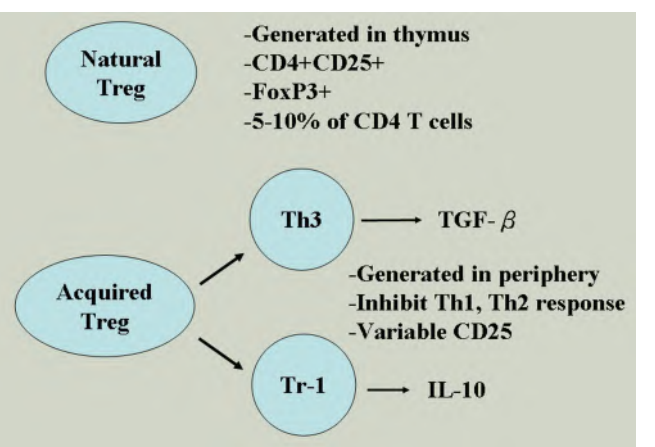

FIGURE 4-6 Regulatory T-lymphocytes. Redrawn with permission from van Ooosterhout and Bloksma, *Eur Respir J.*, 2005;26:918-32.

T Cytotoxic Cells (CD8⁺ T Cytotoxic Cells or Tc) A subset of T cells that kill target cells expressing MHC-associated peptide antigens are also known as cytolytic T lymphocytes (CTLs). The majority of Tc expresses CD8 and recognizes antigens degraded in the cytosol and expressed on the class I MHC molecules of the target cells. Tc develops from "pre-Tc" that lack cytolytic functions. Two separate kinds of signals are needed for pre-Tc to differentiate from functional Tc. One is specific recognition of antigen on a target cells and the other may be provided either by co-stimulators expressed on professional APCs or by cytokines produced by Th cells. Functional Tc acquires specific membrane-bound cytoplasmic granules including a membrane pore-forming protein called perforin or cytolysin and enzymes called granzymes. Tc also expresses FasL that can deliver apoptosis-inducing signals to target cells expressing Fas protein. The killing of target cells require cell contact and is antigen specific, that is, killing of the target cells bearing the same class I MHC-associated antigen that triggered the pre-Tc differentiation.[3,35]

B Cells B cells are rarely found in normal pulps.[37] Their role in adaptive immunity mainly lies in the production of antibodies that constitute the host humoral immune response. The rise and development of B cells take place in bone marrow where the progenitor B cells rearrange their immunoglobulin (Ig) genes and turn into immature B cells with assembled antigen receptors (BCRs) in the form of cell surface IgM. The commitment to B-cell lineage occurs prior to Ig gene rearrangement. This commitment is controlled by the expression of Pax5 transcriptional factor. The bone marrow stromal cells interact with progenitor B cells and provide signals and growth factors (e.g., IL-7) important for B-cell development.

The productive and sequential rearrangement of heavy- and light-chain gene dictates the survival of B cells-positive selection. Those cells that failed to rearrange their Ig gene segments to form functional heavy- and light-chain pairs will be lost. The V(D)J gene recombination occurs in both heavy- and light (κ and λ)-chains. A recombinase system, which is an enzymatic complex consisted 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 to the recombination process. This recombinase system is also used for TCR recombination.

Similar to T-cell selection, B cells also undergo negative selection. Developing B cells expressing BCRs that recognize self-cell-surface molecule are deleted via apoptosis. Immature B cells that bind soluble self-antigens are rendered unresponsive to the antigen-anergic. Only those B cells that do not interact with self-antigens mature normally and enter the peripheral lymphoid tissues bearing both IgM and IgD on their surface. Mature IgM/IgD co-expressing 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 Ig, such as IgG, IgE, and IgA besides IgM. In addition to isotype switching, activated B cells undergo somatic mutation in V region gene leading to affinity maturation of antibodies and alternative splicing of VDJ RNA to membrane or secreted Ig mRNA. A large quantity of antibody is secreted when B cells terminally differentiate into plasma cells.

B-regulatory Cells (Breg) Specific regulatory B cell subsets also exist that downregulate adaptive and innate immunity, inflammation, and autoimmunity.[53] These rare, but specific, subset of regulatory B cells produce IL-10, referred as B10 cells, are capable of downregulating immune responses and inflammatory disease. No phenotypic, transcription factor, or lineage markers are unique to B10 cells, and they are functionally defined and enumerated by their competence to express measurable IL-10.[53]

MHC and Antigen Presenting Cells

Multistep antigen-processing pathways within the APCs are involved in the generation of cell surface antigen: MHC. Distinct subcellular pathways take place to generate complexes of peptides with class I versus class II.

Class I MHC and CD8+ T Cells Class I MHC molecule consists of α (heavy) chain and β chain (β2 microglobulin that is located only extracellularly). Almost all cells express class I MHC that display peptide antigens presented to CD8+ TCRs. Endogenously synthesized foreign proteins (e.g., viral proteins) or products of mutated genes in tumor cells are degraded into peptides by proteosome in the cytosol and transported via Transporter associated with antigen processing (TAP) peptide transporter into the endoplasmic reticulum (ER) in which the peptides are bound onto assembled class I MHC. Subsequently, this peptide:MHC is transported onto cells surface. CD8+ T cells (mostly CTLs) recognize peptide antigen associated with class I MHC.[3,35]

The molecules involved in the interactions between peptide antigen: class I MHC and TCR include CD8, CD3/ζ, CD2, CD28 and LFA-1 (on T cells) and ICAM-1, LFA-3, and B7-1/B7-2 on APC (Figure 4-7).

Class II MHC and CD4+ T Cells Class II MHC molecules composed of two noncovalently associated polypeptide chains-α and β chains, both of which have peptide-binding, immunoglobulin-like, transmembrane, and cytoplasmic domains. Unlike MHC class I, only restricted group of cells normally express class II MHC. They are APCs: 1) DCs, 2) macrophages, 3) B cells, 4) vascular endothelial cells, and 5) epithelial cells. The former three are considered professional APCs. The latter two nonprofessional APCs are induced by IFN-γ to express class II MHC.

DCs and macrophages phagocytose antigens, while B cells utilize the membrane immunoglobulin to bind and internalize antigens. Other nonprofessional APCs endocytose antigens into cytoplasm for antigen processing. These

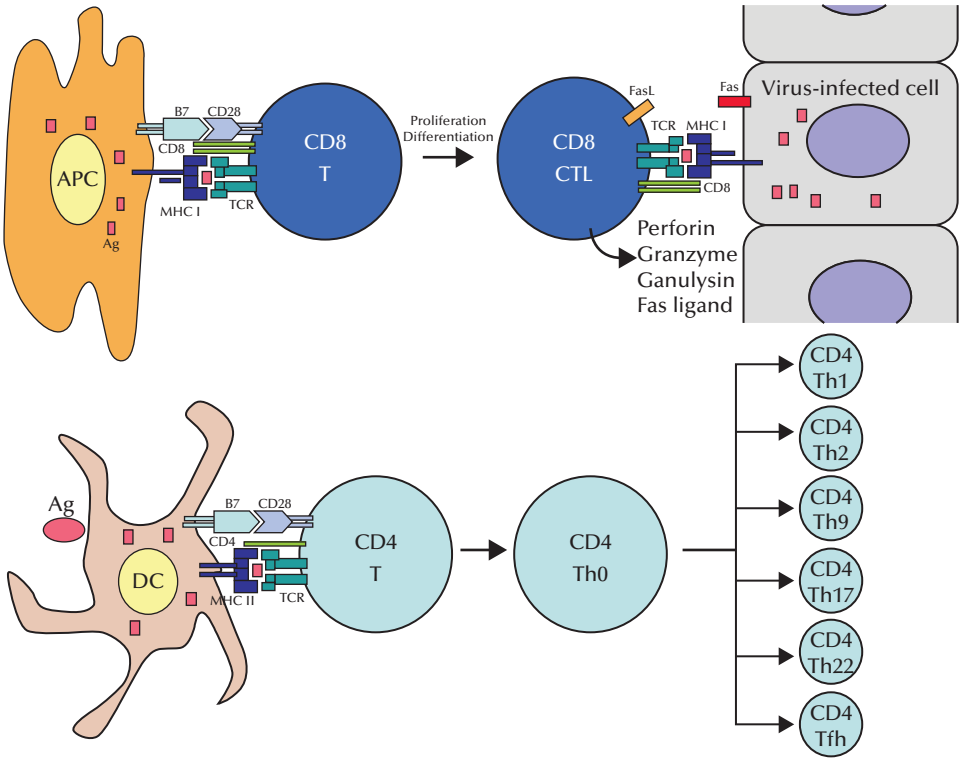

FIGURE 4-7 Cells and molecules involved in the interaction of APCs and CD4/CD8 T lymphocytes.

internalized antigens are localized in endosomes in which the antigens are degraded and then further processed in lysosomes. The processed antigens turn into small peptides and many of which are 10–30 amino acid long capable of binding onto the newly synthesized class II MHC molecules within intracellular vesicles before this antigen:class II MHC is transported to the surface of cells and presented to TCRs of CD4+ T cells (Figure 4-7).[3,35]

MOLECULAR MEDIATORS OF PRO-INFLAMMATORY RESPONSE

Chemokines

Chemokines are a group of ~50 chemoattractant cytokines. There are two main types of chemokines: (1) CC Chemokines: Macrophage/monocyte chemoattractant proteins (MCP-1-4); Macrophage Inflammatory Protein (MIP)-1α, MIP-1β, Eotaxin, RANTES; and (2) CXC Chemokines: IFN inducible protein 10 (IP-10); Monokine inducible IFN-γ (MIG); IL-8/CXCL8. These chemokines have currently been renamed to include CXCL1-16; XCL1, 2; CX3CL1; and CCL1-28. Table 4-4 contains different names of the commonly studied chemokines. The chemokines are primarily involved in trafficking/homing of cells of the innate immune system toward the site of an inflammatory response or during lymphoid organ development and angiogenesis.[6] They may also be involved in recruitment of stem cells to the site of tissue regeneration, such as with pulp regeneration.[54] For more details, see Chapter 29. IL-8 (CXCL8) is among the first recognized and most studied chemokines, which has also been most studied in endodontic pathosis.[28,55–58] There is some redundancy with respect to the cells that express chemokine receptors, and receptor specificity for the various chemokines (Table 4-5).

Adhesion Molecules

There are a variety of adhesion molecule classes that are expressed at the site of an inflammatory process. Adhesion molecules serve a critical role with respect to halting the inflammatory cells that are circulating in the capillaries at high speed, and which, through the function of chemokines, are directed to the site of inflammation. This process is achieved stepwise: the cells initially slow down, roll on the endothelial wall, and then start to bind to the endothelial cells. Eventually, through diapedesis they become extravasated and migrate to the area of inflammation. The slowing down of circulating leukocytes is mediated by Selectins, and then Integrins and Ig superfamily proteins mediate the binding of the cells to endothelial cells. The expression of these adhesion molecules is mediated by a number of inflammatory mediators, most notably IL-1 and TNF-α.

Platelet Activating Factor

PAF is a ubiquitous factor that is secreted by many different cells and plays a role in a number of immune mechanisms. It is secreted by platelets, basophils, monocytes/macrophages, PMNs, and endothelial cells. It is primarily involved in leukocyte adhesion to endothelial cells, production of prostaglandins and thromboxane, and in chemotaxis. In low doses,

TABLE 4-4 Common Chemokines, Their Receptors, and the Cells That They Target[3,6,59,60]

Chemokine	Other name	Cell for Chemotaxis	Receptor
CXCL8	IL-8	Neutrophils	CXCR1, CXCR2
CXCL9	Mig	T cells (T), NK cells (NK)	CXCR3-A, CXCR3-B
CXCL10	IP-10	T, NK	CXCR3-A, CXCR3-B
CXCL1	GROα	Neutrophils	CXCR1
CXCL2	GROβ	Neutrophils	CXCR2
CX_3CL1	Fraktalkine	T cell, NK cell, monocyte	CX_3CR1
CXCL12	SDF-1αβ	Mixed leukocyte; HIV co-receptor	CXCR4
CCL1	I-309	Monocyte, endothelial cells	CCR8
CCL2	MCP-1/ MCAF	T, NK, dendritic cells (DC), monocytes (Mono), basophils (Baso)	CCR2
CCL3	MIP-1α	T, NK, DC, Mono, eosinophils (Eosino)	CCR1, CCR5
CCL4	MIP-1β	T, NK, DC, Mono	CCR5
CCL5	RANTES	T, NK, DC, Mono, Eosino, Baso	CCR1, CCR3, CCR5
CCL7	MCP-3	T, NK, DC, Mono, Eosino, Baso	CCR1, CCR2, CCR3
CCL8	MCP-2	T, NK, DC, Mono, Baso	CCR3
CCL11	Eotaxin-1	T, DC, Eosino, Baso	CCR3
CCL13	MCP-4	T, NK, DC, Mono, Eosino, Baso	CCR2, CCR3
CCL20	MIP-3 α	Th17, DC	CCR6
CCL24	Eotaxin-2	T, DC, Eosino, Baso	CCR3
CCL26	Eotaxin-3	T	CCR3

*For a full list of all 50 chemokines see reference #3.

TABLE 4-5 Important Adhesion Molecules, Their Tissue Distribution and Ligands[3,35]

	Name	Tissue Distribution	Ligand
Selectins	P-selectin (CD62P)	Activated endothelium and platelets	Sialyl-Lewis X on glycoproteins; neutrophils, monocytes, T cells
Bind carbohydrates. Initiate leukocyte-endothelial interaction	E-selectin (CD62E)	Activated endothelium	Sialyl-Lewis X on glycoproteins; neutrophils, monocytes, T cells
	L-selectin (CD62L)	Neutrophils, monocytes, T cells, B cells	Sialyl-Lewis X on glycoproteins; neutrophils, monocytes, T cells
Integrins	$\alpha_L : \beta_2$ (LFA-1, CD11a/CD18)	Monocytes, T cells, macrophages, neutrophils, dendritic cells	ICAMs
Bind to cell-adhesion molecules and extracellular matrix. Strong adhesion	$\alpha_M : \beta_2$ (Mac-1, CD11b/CD18)	Neutrophils, monocytes, macrophages	ICAM-1, iC3b, fibrinogen
	$\alpha_X : \beta_2$ (CR4, p150.95, CD11c/CD18)	Dendritic cells, macrophages, neutrophils	iC3b
	$\alpha_5 : \beta_1$ (VLA-1, CD49d/CD29)	Monocytes, macrophages	Fibronectin
	VLA-4 (CD49aCd29)	Monocytes, T cells	VCAM-1 (CD106)
Immunoglobulin superfamily	ICAM-1 (CD54)	Activated endothelium	LFA-1, Mac-1
Various roles in cell adhesion. Ligand for integrins	ICAM-2 (CD102)	Resting endothelium, dendritic cells	LFA-1
	VCAM-1 (CD106)	Activated endothelium	VLA-4
	PECAM (CD31)	Activated leukocytes, endothelial cell-cell junctions	CD31

PAF may cause vasodilatation; however, in regular doses, it is involved in vasoconstriction.

Plasma Proteases

There are a large number of enzymatic changes that take place during an immune response, and that aim to remove antigenic or foreign material, kill microbial cells, control hemorrhage, as well as initiate and promote the healing process that takes place following the elimination of irritants. These enzymatic processes are closely regulated so that the response is proportionate to the irritation; however, they frequently result in side effects causing break down of host tissues that frequently accompanies an inflammatory response. The main pathways that are involved in these reactions will be described below. The principal mediators and interactions of these pathways are summarized in Figure 4-8.

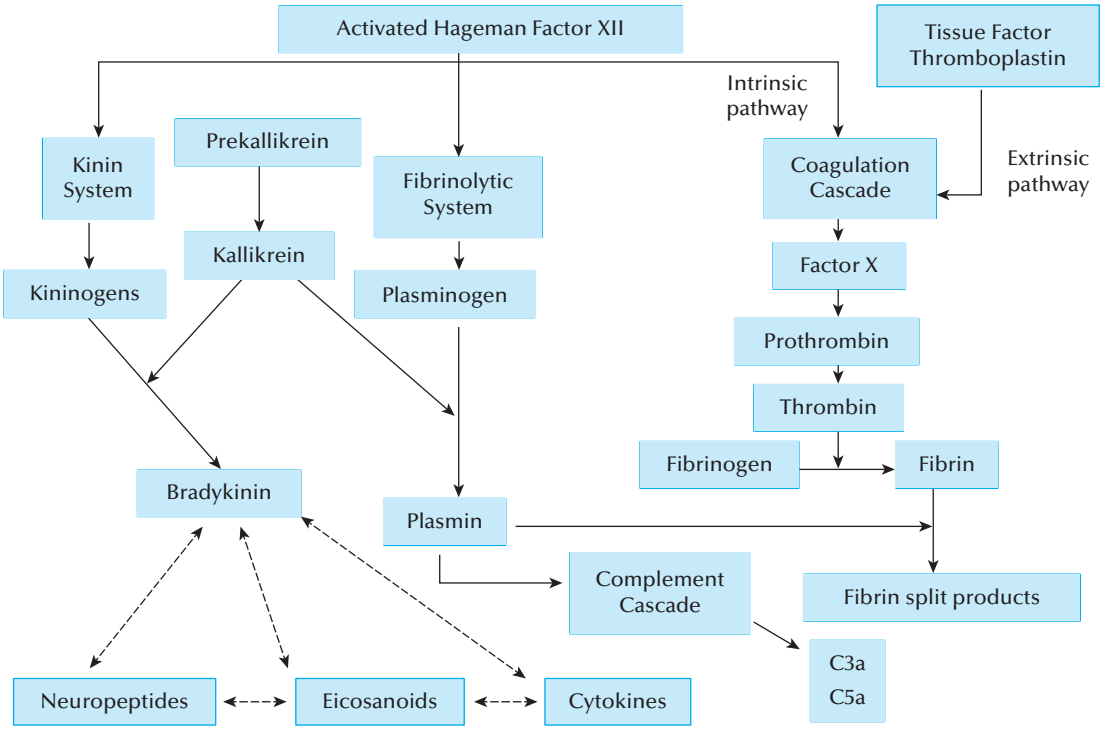

FIGURE 4-8 Diagram showing various pathways that are activated during an inflammatory response. Dotted lines indicate a synergistic or co-stimulatory function.

Kinin System: Bradykinin

As shown in Figure 4-8, the activated Hageman factor causes the formation of kinins from kininogen, a reaction that is mediated by kallikrein. The most important of the kinins is bradykinin. This protein causes vasodilatation, extravasation of plasma proteins, and reduction of pain threshold.[61] Bradykinin performs its actions through binding with two receptors, B1 and B2. B1 is primarily associated with chronic pain syndromes, while B2 is constitutively present in tissues and is overexpressed in acute pain situations,[62] including pulpal pain.[63] It has been shown that bradykinin can act synergistically with neuropeptides, thrombin, and other factors to enhances the production of a number of eicosanoids such as PGE2, and cytokines, such as IL-1 and TNF-α.[64–67] Once released, bradykinin levels are controlled by a number of kininases, and its effects on pain are controlled by endogenous opiates and steroids. Nonsteroidal anti-inflammatory drugs have also been shown to reduce bradykinin levels.[68] It has been shown that sex steroids such as estrogen enhance bradykinin activity in peripheral nerves of the female host.[69]

The Coagulation Cascade and Fibrinolytic Systems

Tissue injury and severe inflammation frequently result in hemorrhage, at macroscopic or microscopic levels. Therefore, one of the principal initial mechanisms of healing is the coagulation cascade. Traditionally, the coagulation cascade has been divided into intrinsic and extrinsic pathways (Figure 4-8). This was due to the fact that the intrinsic pathway could occur in vitro by the Hageman factor, in the absence of thromboplastin. However, there are many interactions between both pathways, and the distinction is now less critical.[14] Through a series of localized enzymatic activations, the Hageman factor/thromboplastin result in the formation of factor X, this in turn results in the formation of thrombin from prothrombin. Thrombin (activated Factor II) has a number of critical functions, including positive feedback activation of a number of other factors such as factors VIII and V, but essentially forms the insoluble fibrin clot from the soluble protein fibrinogen. In order to limit the size of the clot, and prevent disseminated coagulation, the fibrinolytic system is activated and results in the formation of plasmin.

Complement System

The complement system consists of a series of ~45 proteins that play a number of critical roles in the immune system (Figure 4-9). Complement is activated through one of the three main pathways. The classical pathway involves antigen-antibody reaction and results in activation of C1 to form a molecule commonly referred to as C3 convertase. Certain microbial cell wall molecules such as LPS or antigen antibody complexes can activate the alternate pathway, in which C3 through co-enzymatic interaction from Factors B and D, and a protein called properdin, result in the formation of another C3 convertase of a different structure. The lectin pathway is activated by mannose-binding lectin (MBL), which together with ficolin 1, 2, or 3, form the ficolin-associated serine proteases (MASPs). Collectin 11 is also thought to activate the lectin pathway.[70] The enzyme C3 convertase, in whichever formulation, mediates the formation of C3b from C3, with C3a as a by-product. Together

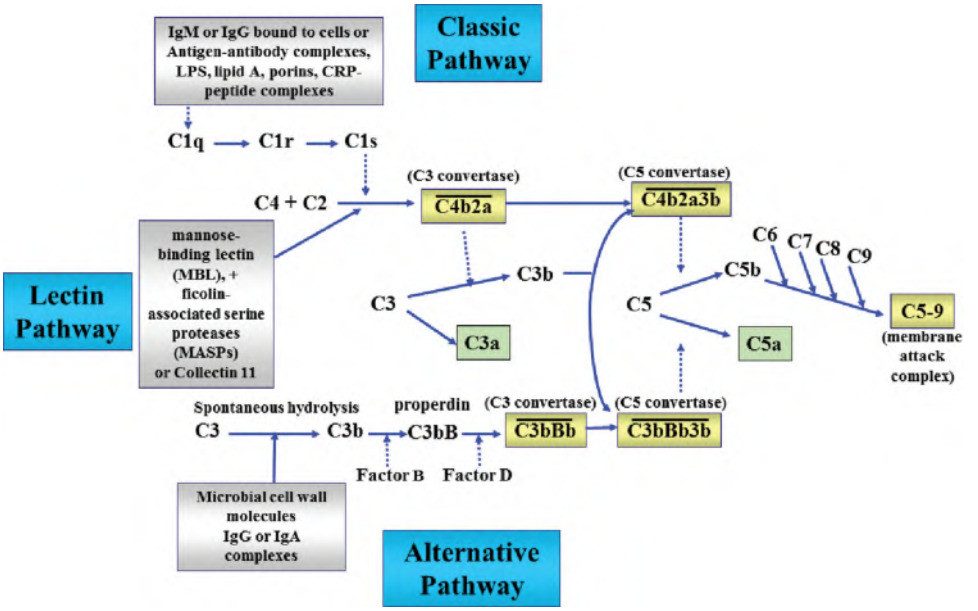

FIGURE 4-9 Complement cascade.

with C5 convertases, C3b activates C5 to C5b, with C5a as a by-product. C5b combines with C6, C7, C8, and C9 to form a membrane attack complex that forms holes through the bacterial cell membrane resulting in death of the cell. Complement molecules may also act as an opsonins for phagocytosis, as discussed previously. C3a and C5a are known as anaphylatoxins because they cause degranulation of mast cells with the release of histamine, which causes vasodilatation and increased vascular permeability. C5a activates the lipoxygenase pathway of arachidonic acid in neutrophils and monocytes, and is itself a potent chemotactic agent for these cells.

It has recently been revealed that some microbial virulence factors exploit the complement cascade to promote inflammation. This process creates an environment conducive to the formation of dysbiotic microbiota, such as that which characterizes periodontal disease.[70] For example, *Porphyromonas gingivalis* produces protein esterase virulence factors called gingipains that act as an agonist to the C5aR (the receptor for C5a).[71] In small amounts, gingipains activate complement and induce an inflammatory process, that is conducive to dysbiotic microbial proliferation. In addition, it was recently shown in two different animal models that C3 is required for induction of periodontal disease with bone loss.[72]

Matrix Metalloproteinases

Matrix metalloproteinases (MMPs) are a number of endopeptidases (Table 4-6) that are responsible for tissue remodeling during structural development, and tissue break down during pathological states such as the inflammatory and neoplastic changes, and caries. In addition to soft tissues, they are present in dentin, and their release during acid etching procedures may degrade collagenous matrix, which would degrade the bond to restorative materials with time. Occasionally some of these enzymes, such as collagenase, may be of microbial origin. Host MMPs are typically modulated by levels of cytokines and growth factors in various tissues. They are upregulated by IL-1β and TNF-α, and growth factors such as platelet-derived growth factor (PDGF), epidermal growth factor (EGF), and nerve growth factor (NGF). They are abrogated by IFN-γ and TGF-β.[61] MMP levels may allow the differentiation for levels of inflammation or correlate with certain histopathological diagnoses. For example, MMP-9 and MMP-13 were shown to be more prevalent in periapical granulomas than cysts.[73] MMP-9 levels may aid in the diagnosis of irreversible pulpitis, or correlate with symptomatic apical periodontitis.[74–76] Finally, MMP-1 gene polymorphism was shown to be associated with the risk for periodontal disease.[77]

MMPs are inactivated by a group of molecules collectively known as tissue inhibitors of metalloproteinases (TIMPs), and by α_2-macroglobulin (that inhibits neutrophil elastase, see below). MMPs and TIMPs have been identified in the dental tissues during health and disease.[78–82] It is noteworthy though, that in the oral cavity MMPs can be released in saliva, and so the sources of MMPs that degrades dentin matrix in caries may be salivary rather than pulpal. Recently,

TABLE 4-6 Commonly Identified Matrix Metalloproteinases That Are Zinc-dependent Endopeptidases

Common Name	MMP
Collagenases	1, 8, 13,18
Gelatinases	2, 9
Stromelysins	3, 10, 11
Membrane Type MMPs (MT MMPs)	14, 15, 16, 17, 24, 25
Minimal domain MMPs	7, 26
Others MMPs	12, 19, 20, 21, 22, 23, 27, 28

TABLE 4-7 Summary of the Origin and Actions of the Commonly Investigated Cytokines[1,3,6,35]

Cytokine	Special function*	Secreted by Interleukins	Effector Function
IL-1α, IL-1β	Pro-inflammatory; bone resorptive	Mono, MΦ, DC, NK, B, Endo	Co-stimulates T activation by enhancing production of cytokines including IL-2 and its receptor; enhances B proliferation and maturation; NK cytotoxicity; induces IL-1, 6, -8, TNF, GM-CSF and PGE_2 by MΦ; pro-inflammatory by inducing chemokines and ICAM-1 and VCAM-1 on endothelium; induces fever, APP, bone resorption by osteoclasts.
IL-1 receptoran-tagonist (IL-1Ra)	Anti-inflammatory	MΦ	Competitive antagonist of IL-1
IL-2	Pro-inflammatory	Th1	Induces proliferation of activated T- and B-cells; enhances NK cytotoxicity and killing of tumor cells and bacteria by monocytes and MΦ.
IL-3		T, NK, MC	Growth and differentiation of hematopoietic precursors; MC growth
IL-4	Anti-inflammatory; bone modulatory	Th2, Tc2, NK, T, MC	Induces Th2 cells; stimulates proliferation of activated B, T, MC; upregulates MHC class II on B and MΦ, and CD23 on B; downregulates IL-12 production and thereby inhibits Th1 differentiation, increases MΦ phagocytosis; induces switch to lgG1 and lgE.
IL-5	Anti-inflammatory	Th2, MC	Induces proliferation of eosino and activated B; induces switch to lgA.
IL-6	Pro-inflammatory; bone modula-tory, systemic inflammation	Th2, Mono, MΦ, DC, BM stroma	Liver: Synthesis of acute phase proteins. Differentiation of myeloid stem cells and of B into plasma cells; enhances T proliferation.
IL-7		BM and thymic stroma	Induces differentiation of lymphoid stem cells into progenitor T and B; activates mature T.
IL-8		Neutro; Mono, MΦ, Endo	Chemokine:mediates chemotaxis and activation of neutrophils.
IL-9		CD4+ T cells	Induces proliferation of thymocytes; enhances MC growth; synergizes with IL-4 in switch to lgG1 and lgE.
IL-10	Anti-inflammatory; bone modulatory	MΦ, T cells (mainly regulatory T cells)	Inhibits IFNγ, IL-2, Th1 cells; downregulates MHC class II and cytokine (including IL-12) production by mono, MΦ and DC, thereby inhibiting Th1. differentiation; inhibits T proliferation; enhances B differentiation.
IL-11	Bone-resorptive	BM stroma	Promotes differentiation of pro-B and megakaryocytes; induces APP, production of platelets.
IL-12	Pro-inflammatory	Mono, MΦ, DC, B	Critical cytokine for Th1 differentiation; induces proliferation and IFNγ production by Th1, $CD8^+$and NK; enhances NK and $CD8^+$ T cytotoxicity.
IL-13	Anti-inflammatory	Th2, MC	Inhibits activation and cytokine secretion by MΦ; co-activates B proliferation; upregulates MHC class II and CD23 on B and mono; induces switch to lgG1 and lgE; induces VCAM-1 on Endo.
IL-15		T, NK, Mono, MΦ, DC, B	Induces proliferation of T-, NK an deactivated B and Cytokine production and cytotoxicity in NK and $CD8^+$ T; chemotactic for T; stimulates growth of intestinal epithelium.
IL-16		Th, Tc	Chemoattractant for CD4+ T, mono and eosino; induces MHC class II.
IL-17	Pro-inflammatory	Th17, innate lym-phoid cells	Pro-inflammatory; stimulates production of cytokines including TNF, IL-1β, -6, G-CSF, and chemokines.
IL-18	Bone-resorptive	Mono, MΦ, DC, keratinocytes, osteoblasts	Induces IFNγ production by T; enhances NK cytotoxicity. Mono: enhances GM-CSF, TNF, IL-1β.
IL-19		Mono, MΦ	Modulation of Th1 activity
IL-20		Mono, Keratinocytes	Regulation of inflammatory responses to skin
IL-21		Th2 cells, Th17 cells, Tfh cells	Regulation of hematopoiesis; NK differentiation; B cell activation, differentiation; T co-stimulation
IL-22		Th17 cells	Inhibits IL-4 production by Th2; epithelial cells to produce defensins.
IL-23		DC, MΦ	Induces proliferation and IFNγ production by Th1; induces proliferation of Th17 and memory cells.
IL-24		Th2, Mono, MΦ	Induction of TNF, IL-1, IL-6, anti-tumor activity
IL-25		Th1, MΦ, MC	Induction of IL-4, IL-5, IL-13 and Th2-associated pathologies
IL-26		T, NK	Enhanced production of IL-8 and IL-10 by epithelium
IL-27		DC, Mono	Induction of TH1 responses, enhanced IFN-γ production

(*Continued on facing page*)

TABLE 4-7 (*Continued*) Summary of the Origin and Actions of the Commonly Investigated Cytokines

Cytokine	Special function*	Secreted by Interleukins	Effector Function
IL-28		Mono, DC	Type 1 IFN-like activity, inhibition of viral replication
IL-29		Mono, DC	Type 1 IFN-like activity, inhibition of viral replication
IL-31		Th2 cells	Promotes inflammatory responses in skin
IL-32		NK, T	Promotes inflammation. Role in activation-induced
IL-33		Endothelial cells, smooth muscle cells, keratinocytes, fibroblasts	Th2 development;induction of IL-4, IL-5, IL-13, activation of innate lymphocyte cells.
		Tumor Necrosis Factors	
TNF-α	Pro-inflammatory; bone resorptive	Th1, Mono, MΦ, DC, MC, NK, B	Tumor cytotoxicity; cachexia (weight loss due to muscle fat catabolism); induces cytokine secretion; induces E-selectin on endo; activates MΦ; antiviral, hypothalamus: fever.
Lymphotoxin (LTα, TNF-β, TNFSf1)	Pro-inflammatory	Th1, Tc	Tumor cytotoxicity; enhances phagocytosis by neutro and MΦ; involved in lymphoid organ development, antiviral
Lymphotoxin-αβ (LTαβ)		T, NK, follicular B, lymphoid inducer cells.	Lymphoid organogenesis, chemokines
		Interferons	
IFNα		Leukocytes	Inhibits viral replication; enhances MHC class I
IFNβ		Fibroblasts	Inhibits viral replication, enhances MHC class I
IFNγ	Pro-inflammatory	Th1, Tc1, NK	Inhibits viral replication, enhances MHC class I and II; activates MΦ; induces switch to lgG2a; Th1 differentiation, increases antigen presentation and processing by T cells; antagonizes several IL-4 actions; inhibits proliferation of Th2.
		Colony stimulating factors	
GM-CSF	Pro-inflammatory	Th, MΦ, Fibro, MC, Endo	Stimulates growth of progenitors of mono, neutro, eosino and baso; activates MΦ.
G-CSF	Pro-inflammatory	Fibro, Endo	Stimulates growth of neutro progenitors.
M-CSF	Pro-inflammatory	Fibro, Endo, Epith	Stimulates growth of mono progenitors.
Stem cell factor (SLF)		BM stromal cells	Stimulates stem cell division (c-kit ligand).
		Others	
TGFβ		Treg cells, B, MΦ	Inhibition of proliferation and effector functions of T cells; differentiation of Th17 and Treg B cells, chemo attraction of mono and MΦ but also anti-inflammatory; promotes tissue repair and angiogenic factors.
LIF		Thymic epith, BM stromal cells	Stem cells: block in differentiation
Osteoprotegrin (OPG)		osteoblasts	Inhibits osteoclast differentiation
Oncostatin M	Bone marrow stromal cells	T, MΦ	Regulation of hematopoietic cytokines in endothelial cells; inhibits cancer cell proliferation.

APP, acute-phase proteins; B, B-cell; baso, basophil; BM, bone marrow; Endo, Endothelium; eosino, eosinophil; Epith, epithelium; Fibro, fibroblast; GM-CSF, granulocyte-macrophage colony-stimulating factor; IL, interleukin; LIF, leukemia inhibitory factor; MΦ, macrophage; MC, mast cell ; Mono, monocyte; neutro, neutrophil; NK, natural killer; SLF, steel locus factor; T, T-cell; TGFβ, transforming growth factor-β. IL-8 is a member of the chemokine family.

1,10-phenanthroline-5,6-dione (Phendione), a metal chelator and an inactivator of MMPs, was shown to have antimicrobial agent against *Enterococcus faecalis* and to be an inhibitor of human MMP-2 in the root canal.[83]

It has been documented through a significant body of evidence that tetracyclines, and some tetracycline analogues and chemically-modified tetracyclines may degrade MMPs at subantibiotic levels.[84,85] This is thought to be one of a number of anti-inflammatory properties that are exhibited by some antibiotics.[61]

Neutrophil and Macrophage Elastases

During pulpal and periapical tissue inflammation, a number of other proteases may contribute to the tissue destruction frequently encountered. Among these protolytic enzymes are neutrophil elastases that are neutral serine proteases,

released from PMNs or macrophages during activation of these cells. Neutrophil elastase can cleave a wide variety of structurally important proteins and glycoproteins, such as elastin, type III and IV collagen, fibronectin, and core proteins of proteoglycan molecules. It has been reported that neutrophil elastase was increased in periapical lesions that were symptomatic,[86] and that its levels although correlated with prostaglandin E2, did not change following root canal instrumentation.[87] Differential expression of neutrophil and macrophage elastases showed the latter to increase earlier in periapical lesion induction, but both appeared to be expressed in high levels.[88]

Nitric Oxide

Nitric oxide (NO) is a gaseous free radical that has been described to have several pro-inflammatory and modulatory effects in tissues. It is produced through a reaction that involves the enzyme NO synthase (NOS) and several cofactors including NADPH. Several NOS isoforms are present, including inducible (iNOS), neuronal (nNOS), and endothelial (eNOS). The nNOS and eNOS are constitutively expressed in the respective tissues and are calcium dependent, whereas the iNOS is induced in macrophages and a number of other cells, primarily by cytokines such as IL-1 and TNF-α or microbial products such as LPS, and are calcium independent.[61] Because it is relatively volatile and short lived, the detection of NO is frequently assessed through the presence of NOS, particularly iNOS.

Under normal conditions, NO is primarily involved in vasodilatation through the relaxation of smooth muscles and prevention of adhesion of leukocytes to endothelial wall, which is considered as an anti-inflammatory function. However, during inflammation, including that of the pulp and periapical tissues, and due to the action of cytokines, large amounts of NO are released.[89–94] NO contributes to the killing of microbial cells and tumor cells by macrophages through its interaction with super-oxide free radical[14] as follows:

$$NO + O_2^{\cdot -} \rightarrow OH^{\cdot} + NO_2$$

NO is also an antagonist for platelet activation, adhesion, and degranulation. Moreover, it was shown that NO can regulate the proliferation, growth, and apoptosis of pulp cells *in vitro*.[95]

Oxygen-Derived Free Radicals

These are highly reactive, short-lived molecules produced by macrophages and neutrophils in normal and pathological states. The most important molecules are the superoxide anion ($O2^{\bullet -}$), which eventually is converted to hydrogen peroxide (H_2O_2), and the hydroxyl radical ($OH^{\bullet}$) and toxic NO derivatives.[14]

The main role that these molecules play is to increase the inflammatory response by increasing cytokines, chemokines, and adhesion molecules. However, in large amounts they cause thrombosis and tissue damage. The antioxidant activity is mediated through enzymes such as catalase, glutathione peroxidases, and superoxide dismutase (SOD). There are two main forms of SODs: copper–zinc (Cu, Zn-SOD) found primarily in cytoplasm and manganese (Mn-SOD) found primarily in mitochondria. While the presence of SOD in normal pulp has been described several decades ago,[96,97] the effect of inflammation on the levels of both isoforms of this enzyme has been controversial. Several studies have shown an increase of SOD,[96,98] or Cu, Zn-SOD and Mn-SOD[99] in pulpal inflammation. In fact, one study showed an anti-inflammatory effect that Cu, Zn-SOD has pulpal inflammation in an animal model.[100] However, a more recent study showed that there is a reduction of Cu, Zn-SOD in human pulpitis, possibly due to depletion.[101] Using quantitative real-time reverse transcriptase polymerase chain reaction, that is more sensitive than traditional protein assays, it has been reported that the expression of mRNA for both enzymes does increase in pulpitis in patients and that the elevation of Mn-SOD is significantly higher than that of Cu, Zn-SOD.[102] It was also shown that TLR-2 is involved in the induction of reactive oxygen species (ROS) production by macrophages in response to several root canal bacteria, while only *Fusobacterium nucleatum*-induced ROS production by TLR-4-competent macrophages.[103]

Cytokines

Cytokines are low-molecular-weight proteins that stimulate or inhibit proliferation, differentiation, or function of immune cells. Over 40 cytokines have been described with various functions that mediate or modulate the immune system and bone resorption. Cytokines may exert their actions through autocrine (acting on receptors of the same cell that produced them), paracrine (acting on other cells in the local vicinity), and endocrine (acting systemically) modes. Because of the involvement of cytokines in several areas of interest in endodontics, their various roles and functions have been and will be described in various sections of this chapter and in other chapters dealing with pulpal and periapical pathosis. In order to avoid redundancy, a brief description of the main features of some cytokines will be described here, with other information provided elsewhere in the chapter. Table 4-7 presents a summary of the role, sources, and main actions of common cytokines.

Pro-inflammatory Cytokines

Pro-inflammatory cytokines include IL-2, IFN-γ, IL-1α, IL-1β, TNF-α, IL-6, IL-12, IL-17, and CSFs such as GM-CSF and M-CSF (Table 4-7). These cytokines are produced by a variety of cells in response to antigens, microbial cells and molecules, autoimmune factors, and neoplastic triggers, or as part of homeostatic function, and represent the effector arm of the immune response. Their effects include cell differentiation, proliferation, activation, bone resorption, chemokine production, and a number of other important functions to respond to and degrade the irritants.

TNF-α is the principal mediator of the response to gram-negative bacteria. LPS stimulate the functions of mononuclear phagocytes and act as a polyclonal activator of B cells, thus contribute to elimination of the invading bacteria. The major cellular source of TNF is the LPS-activated mononuclear phagocyte. Other sources include antigen-stimulated T cells, activated NK cells, and activated mast cells. IL-1 derived from mononuclear phagocytes, and its principal function, similar to TNF, is to mediate host inflammatory response in innate immunity. T cells are more effective than LPS at eliciting IL-1 synthesis by monocytes. IL-1 is made by a diverse cell types, including epithelium and endothelium, providing local sources of IL-1 in the absence of macrophage-rich infiltrates.

The effect of IL-1 is similar to TNF. At low quantity, it acts on endothelium to increase the expression of adhesion molecules and on monocytes to secrete chemokines. It shares many features of TNF but is also different, for example, it potentiates rather than suppresses the actions of CSFs on bone marrow cells (Table 4-7). The actions of IL-6 induce IL-1 production, causing hepatocytes to synthesize plasma proteins, such as fibrinogen, C-reactive protein, and serum amyloid proteins, which contributes to the acute phase response. It serves as a growth factor for activated B cells.[104,105]

One of the principle pro-inflammatory (also known as Type 1) cytokines is IL-2. This cytokine is produced by the activated CD4+ cells and activates monocytes and macrophages leading to the initiation of the immune response. Other important Type 1 cytokines include IFN-γ, IL-12, and IL-17 (Table 4-7). All these cytokines are clearly critical for the initiation of pulpal and periapical inflammation. However, it has been shown that the main cytokines involved in bone resorption and mediating pulp necrosis and periapical inflammatory response are IL-1 and TNF-α, and that these reactions fundamentally rely on the innate rather than the adaptive immune response.[106] (For Anti-inflammatory cytokines, see later sections in this chapter.)

Neuropeptides

Neuropeptides are proteins produced by neural tissues and are released at the sites of nerve endings. Under normal conditions, they function to maintain the vascular tone of blood vessels; thus, modulating the vascularity and regulating the innervation of the tissue. During an inflammatory response, there is vasodilatation, extravasation of plasma proteins, and sprouting of nerve endings within the inflamed tissue. These changes result in an increase in the levels of certain neuropeptides. It is now recognized that certain neuropeptides such as Substance P (SP) have decidedly pro-inflammatory actions, whereas a number of other neuropeptides such as vasoactive intestinal peptide (VIP), a-melanocyte-stimulating hormone, neuropeptide Y (NPY), and somatostatin are members of an anti-inflammatory repertoire of immune modulators.[107–109] The newly discovered neuropeptides, such as urocortin, adrenomedullin, and cortistatin, also appear to have anti-inflammatory actions in that they inhibit T-cell proliferation, Th1 response, and IL-2 production.[108] Calcitonin gene-related peptide (CGRP) may have pro-inflammatory as well as anti-inflammatory actions.[108] SP was the first neuropeptide described around 1931. It is localized in sensory nerves, is related to neurokinins A and B, and its receptors are NK1, NK2, and NK3.[110] CGRP (described in 1983) binds to CGRP1, CGRP2, and AM2, whereas NPY (discovered in 1982) is localized in sympathetic, cholinergic, sensory, enteric, and central neurons, and binds to Y1, Y2, Y4, and Y5 receptors.

With respect to pulpal and periapical region, denervation experiments have shown that, in the pulp, SP-, neurokinin A-, and CGRP-containing nerve fibers originate from the trigeminal ganglion and that NPY-containing nerve fibers come from the superior cervical ganglion. The origin of VIP-containing fibers may be parasympathetic nerve fibers since it has been associated with acetylcholine in other tissues.[61,111]

Since the dental pulp is highly innervated, neuropeptides play a significant role in mediating and modulating the inflammatory response in it. The neuropeptides that have been studied the most in endodontic pathosis are SP and CGRP, and VIP and NPY are studied to a lesser degree. In the normal rat molar pulp, SP and CGRP were shown to be present in close proximity to macrophages, an association that was more prevalent in the odontoblastic layer than in the central pulp.[112] During inflammation, sprouting of pulpal nerve fibers was shown to be associated with an increased expression of SP and CGRP closely surrounding the areas of irritation.[113,114] However, severe irritation by experimental pulp exposures caused a drop in the overall pulpal levels of SP and CGRP, possibly due to the depletion of neuropeptide stores in the nerve endings.[115] CGRP addition to human pulp cells *in vitro* caused a two-fold increase in the levels of bone morphogenetic protein 2 (BMP-2), a member of TGFβ superfamily, which has the capacity to induce dentin regeneration.[116] Clearly, these findings suggest that neurogenic inflammation effected by neuropeptides plays an active and dynamic role in modulating pulpal inflammation.

Neuropeptides are thought to contribute to the inflammatory process in a number of ways. SP and CGRP,[117] together with VIP, are potent vasodilators; however, NPY is a vasoconstrictor.[111] SP also causes increased vascular permeability and plasma extravasation.[118]

Neuropeptides may reduce the threshold of pain in the pulp, accounting for symptoms associated with certain cases of pulpitis. The association of SP with the extent of caries and pain from caries was investigated in permanent molars of children.[119] The percentage of neuronal areas that positively stained for SP was found to be significantly more in gross carious lesions than in moderate or no lesions and significantly more in painful lesions. These findings were true at all levels of coronal pulp studied that included the pulp horn, subodontoblastic nerve plexus, and mid-coronal pulp. The fact that not all cases of irreversible pulpitis are associated

with pain may be explained by the actions of inhibitors of neurotransmitters such as γ-aminobutyric acid (GABA) or gastrin-releasing peptide (GRP). GABA-like and GRP-like immunoreactivity has been identified in the dental pulp.[120] It was also recently shown that NPY is capable of inhibiting capsaicin-sensitive afferent neurons at the spinal level, perhaps explaining the mechanisms for spinal NPY-induced anti-nociception.[121] Local opioid receptors in the pulp may also play a role in suppressing pulpal pain.[122,123] On the other hand, it was recently shown that dental pulp nociceptors, collected from patients with caries, express TLR-4 and CD-14,[124] that TNFα enhances the sensitivity of trigeminal neurons,[125] and that LPS senstizesTRPV1 receptors via action onTLR-4.[126] Collectively, these findings indicate a direct mechanism for bacteria and bacterial LPS to activate these nociceptors.

Neuropeptides may contribute to the inflammatory process by a number of other mechanisms. These include the release of inflammatory mediators, such as histamine, PGE2, collagenase, IL-1, IL-6, and TNFα, the enhancement of chemotaxis, phagocytosis, and the expression of adhesion molecules, and lymphocyte proliferation and IL-2 production.[127] The interactions between eicosanoids, bradykinin, and various neuropeptides suggest synergistic action with CGRP[128] or modulatory action with NPY[107] and illustrate the complex interplay between various elements of the immune response. The neuropeptides, SP, CGRP, VIP, and NPY, may also modulate the expression of angiogenic growth factors.[129] It is interesting to note that animal studies have shown a reduction of the neuropeptides SP and CGRP with age.[130]

Toll-like Receptors

Innate immunity has a considerable specificity achieved through the recognition of multiple microbe-specific surface molecules by PRR[131] present on host cells, such as TLRs and the receptors for complement. The PRR-recognized molecules on the microbes include surface proteins, nucleic acids, and carbohydrates (e.g., lipopolysaccharide, PG, LTAs). This is known as microbe-associated molecular patterns.[131,132]

TLRs, as pattern-recognition receptors, are a family of 12 members identified in mammals so far and are further divided into several subfamilies (Table 4-8). The subfamily of TLR1, TLR2, and TLR6 recognizes lipids, whereas the highly related TLR7, TLR8, and TLR9 recognize nucleic acids. TLR4 recognizes ligands such as lipopolysaccharide (LPS), fibronectin, and heat-shock proteins, all of which have different structures. TLRs are expressed on various immune cells, including macrophages, DCs, B cells, specific types of T cells, and on nonimmune cells, such as fibroblasts and epithelial cells. Expression of TLRs is modulated rapidly in response to pathogens, a variety of cytokines, and environmental stresses. TLRs may be expressed extracellularly or intracellularly. For example, TLRs 1, 2, 4, 5, and 6 are expressed on the cell surface, others (TLRs 3, 7, 8, and 9) are found almost exclusively in intracellular compartments such as endosomes.[131,132]

TABLE 4-8 Examples of TLR Recognition of Microbial Components

Molecule	Microorganism type/species	TLR
LPS	G(–) bacteria	TLR4
LTA (lipoteichoic acid)	Group B *Streptococcus*	TLR6/2
Peptidoglycan	G(+) bacteria	TLR2
Flagellin	Flagellated bacteria	TLR5
CpG-DNA	Bacteria and mycobacteria	TLR9
Not determined	Uropathogenic bacteria	TLR11
Zymosan	*Saccharomyces cerevisiae*	TLR6/2
Mannan	*Candida albicans*	TLR4
Viral DNA	Virus	TLR9

NOD-like Receptors

Similar to TLR family, NOD-like receptors or NLRs are cytoplasmic PRRs, together with retinoic acid-inducible gene 1-like receptor and TLR, make up the innate pathogen pattern-recognition system.[133] There are 22 members of NLRs in humans. They respond to intracellular pathogens as well as play important roles in distinct biological processes ranging from regulation of antigen presentation, sensing metabolic changes in the cell, modulation of inflammation, embryo development, cell death, and differentiation of the adaptive immune response.[133]

NLRPs are a subgroup of NLRs constituted by proteins such as NLRP1, 3, 4, 6, 7, and 12 that are involved in the formation of multi-protein complexes termed inflammasomes. These complexes consist of one or two NLR proteins, the adapter molecule apoptosis-associated speck-like containing a Caspase activation and recruitment domains (CARD) and pro-caspase-1. These inflammasomes might sense microbial products and a variety of stress and damage associated-endogenous signals.[134]

AGE/RAGE

Advanced glycation end products (AGE) are a group of heterogenous compounds formed in the hyperglycemic environment of patients with diabetes mellitus. The receptor for these compounds has the acronym RAGE. Diabetes mellitus (DM) affects an estimated 350 million cases worldwide,[135] and 29.1 million people or 9.3% of the U.S. population, of whom 8.1 million are undiagnosed <http://www.cdc.gov/diabetes/pubs/statsreport14/national-diabetes-report-web.pdf>. AGEs are thought to be involved in the pathogenesis of types 1 and 2 DM, and may also be oxidative derivatives resulting from diabetic hyperglycemia.[136] Glucose exhibits a slow glycation rate, and glucose-6-phosphate or glyceraldehyde-3-phosphate, which are intracellularly present at small levels, can form AGEs at a faster rate. The chemical transformation of amine-containing molecules by reducing sugars (glycation)—whether on proteins, lipids or nucleotides,

results in AGEs or the so-called: Maillard products.[136] Several molecules such as carboxymethyllysine (CML), pentosidine, or derivatives of methylglyoxal (MG), such as MG-H1, are well-studied AGE compounds, and serve as AGE markers. AGEs could also be introduced through food processing, dry heat cooking, and cigarette smoking.

In addition to mediating pancreatic β-cell toxicity, AGEs can promote recruitment and activation of T cells and macrophages.[137] At least two types of cellular AGE receptors have been characterized: RAGE and AGER1. AGER1 binds, degrades AGEs, and protects against oxidant injury. However, in excess of external AGEs, AGER1 is depleted.[136] Furthermore, the consequence of AGE-RAGE interaction is the development of cellular oxidant stress, a means by which cell signaling pathways may be activated, thereby resulting in altered cellular phenotype and cellular dysfunction.[137] AGE-RAGE axis in endothelial cells could induce the expression of genes for vascular endothelial growth factor, enhance vascular permeability, angiogenesis, and local inflammation. The production of several cytokines, such as TNF-α, IL-1β, IL-6, and monocyte chemotactic protein-1, is induced by the AGE–RAGE in monocytes and macrophage. RAGE promoter assays showed that AGE-RAGE signaling promotes the transcriptional upregulation of the RAGE gene through the activation of NF-κB.[138]

Defensins

Human β-defensins (hBDs or DEFB) is a potent antimicrobial peptide effective against a broad spectrum of bacteria, including Gram-positive bacteria, Gram-negative bacteria, and fungi. Among these microbes, *Streptococcus mutans* and *Lactobacillus acidophilus* that are associated with dental caries, and certain strains of periodontal bacteria such as *Aggregatibacter actinomycetemcomitans* and *P. gingivalis* have been shown to be sensitive to hBD-2.[139,140] hBDs are expressed mostly in epithelia.[27,141–144] Besides the cloned, sequenced, and well-characterized hBD-1, -2, -3, and -4, there appears to be at least 28 more hBDs existing based on the genomic surveys.[145] Fourteen hBDs or DEFB gene transcripts (DEFB-1, -4, and DEFB-103 to -114) have been detected in gingival keratinocytes.[146] Periodontal bacteria *F. nucleatum* and *A. actinomycetemcomitans* induce hBD-2 and hBD-3, respectively, in oral epithelial cells.[147]

Growth Factors

Growth factors are peptide molecules that transmit signals between cells and tissue, which may result in differentiation, proliferation, or activation of cells or inhibition of these functions. In general, growth factors promote and accelerate the structural development or the healing of tissues following injury. Some growth factors, such as angiogenic growth factors, have also been implicated in the pathogenesis of neoplastic changes within tissues. Most growth factors are named after the original tissue in which they were identified, a fact that could be misleading because the growth factors typically are ubiquitous in that they perform their actions in a wide range of tissues. As mentioned for cytokines, growth factors can perform their actions in an autocrine, paracrine, or endocrine manner. Table 4-9 lists a classification and nomenclature of some of the main classes of growth factors.

Many growth factors are not only mediators of hard tissue matrix deposition but also become entrapped within the matrix as it becomes calcified. This has led to a great

TABLE 4-9 Classification of the More Commonly Recognized and Studied Growth Factors

Superfamily	Family	Abbreviated Name
Transforming growth factor-β	Transforming growth factor-β	TGF-β
	Inhibins	Inhibin/Activin
	Bone morphogenetic proteins	BMP
	Vg-1	Vg-1, GDF-1, DPP
Platelet-derived growth factor	Platelet-derived growth factors	PDGF
	Vascular endothelial growth factors	VEGF
	Connective tissue growth factors	CTGF
Epidermal growth factor	Epidermal growth factor	EGF
	Transforming growth factor-*a*	TGF-*a*
Enamel matrix derivatives	Amelogenin	Collectively known as EMD
	Enamelin (Enam)	
	Tuftelin	
Other large peptide growth factor families	Fibroblast growth factors	FGF
	Insulin-like growth factors	IGF
	Nerve growth factor	NGF
	Hepatocyte growth factor	HGF
	Tumor necrosis factors*	TNF

*As noted before, TNF-α and TNF-β are usually classified as pro-inflammatory cytokines, but sometimes considered within growth factor Classifications.[148]

interest in osteoinductive growth factors such as the bone morphogenetic proteins BMP-2 or BMP-7, enamel matrix derivatives (EMDs), PDGF, and insulin-like growth factor (IGF). These proteins have been extensively investigated with respect to their ability to induce, potentiate, and accelerate bone regeneration, whether as purified proteins or as components of demineralized freeze-dried bones.[18,149–154]

There have been considerable interest in whether BMPs,[155–159] TGF-β,[160–163] EMD[164–166] or other growth factors may enhance the formation of reparative dentin following pulp exposure. *In vivo* trials in animal models have initially been conducted via protein deposition directly on experimentally exposed pulp.[156,159,167] However, more recently, gene transfer strategies have been employed to assure prolonged effect of the induced protein,[157,163] or transplantation of cells in which the growth factor genes have been induced *in vitro* to a polyglycolic acid scaffold in the pulp.[168] However, it was later realized that in the presence of inflammation, as induced by applying LPS to the model, these strategies fail to produce the desired outcome.[155]

Within a pathological pulpal or periapical tissue, or even during an orthodontic movement, growth factors are frequently expressed and are thought to modulate the inflammatory process and aid in the healing that ensues after the removal of irritants.[18,169–174] For example, it has been shown that TGF-β1 down-regulates the expression of matrix metalloprotein-8 (MMP-8) in human odontoblasts and dental pulp cells *in vitro*.[79]

Vascular endothelial growth factor (VEGF) has also been identified as a critical growth factor for the pathogenesis and sustenance of periapical lesions.[175–177] This growth factor most likely also plays an important role in healing of lesions following treatment, although this has not been adequately described.

MOLECULAR MEDIATORS OF ANTI-INFLAMMATORY REACTIONS

Membrane Phospholipids/Arachidonic Acid Metabolic Pathways

Inflammatory process is fine-tuned by pro-inflammatory and anti-inflammatory mediators. There is more information known about pro-inflammatory mediators, whereas less is known regarding anti-inflammatory factors. In fact, in the pathways leading to the production of pro-inflammatory mediators such as prostaglandins and leukotrienes, there is also production of anti-inflammatory mediators such as lipoxins (Figure 4-10). In the host defense mechanism, acute inflammation executed by neutrophils is resolved by proceeding to chronic inflammation in which pro-inflammatory mediators are produced, or to resolution in which protective mediators are released.[178–180]

This pro-resolution against acute inflammation is an active process that involves the activation of specific biochemical and cellular programs of resolution. Lipoxins are potent counter-regulatory signals for endogenous pro-inflammatory mediators, including lipids (e.g., leukotrienes) and cytokines (TNF-α, IL-6), resulting in inhibition of leukocyte-dependent inflammation. Lipoxins are rapidly biosynthesized in response to stimuli, act locally, and then are rapidly enzymatically inactivated.[179,181–183] Another important group of lipid mediators that possess anti-inflammatory and pro-solution action is resolvin, which protects healthy tissue during immune-inflammatory responses to infection or injury, promoting inflammation resolution and tissue healing.[184] Resolvins are derived from omega-3 (ω-3) fatty acids, docosahexaenoic acid (DHA), and eicosapentaenoic acid (EPA), and have subtypes including the E series (RvE1-3, from EPA), the D series (RvD1 and RvD2, from DHA), and aspirin-triggered (AT) forms (AT-RvD1-6).[179,184]

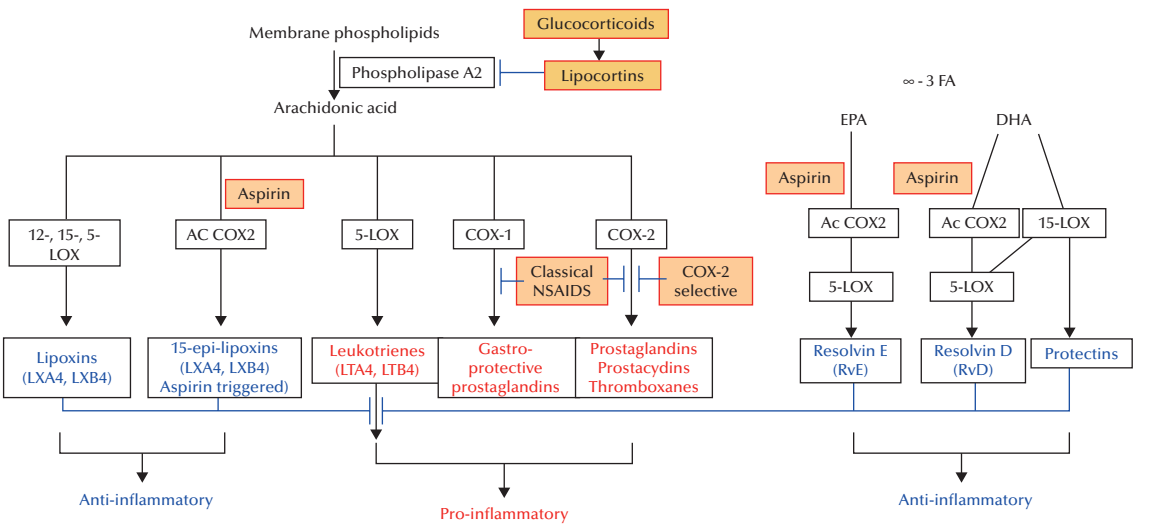

FIGURE 4-10 Pathways of lipid mediator production from membrane phospholipids and pathways of omega-3 (ω-3) fatty acids metabolism. LOX: lipoxygenase; COX: cyclooxygenase; NSAIDS: nonsteroidal anti-inflammatory drugs; LX: lipoxin; DHA: docosahexaenoic acid; EPA: eicosapentaenoic acid.

They are generated via COX2-/LOX-mediated pathways in which another molecule belonging to a new family of mediators named protectins are also produced that possess similar anti-inflammatory functions as resolvins (Figure 4-10).[185]

Anti-inflammatory Cytokines (IL-10, TGF-β, and Regulatory T Cells)

Several cytokines have predominantly anti-inflammatory effects including IL-1Ra, TGF-β, and IL-10 (Table 4-7). Monocytes and B cells are the major sources of IL-10 in human subjects. The primary T-cell source for IL-10 is regulatory T lymphocytes (Treg), as mentioned above. IL-10 inhibits production of many pro-inflammatory cytokines such as IL-1β, IL-6, IL-8, IL-12, IFN-γ, and TNF-α. In addition, IL-10 inhibits MHC class II, CD23, intercellular adhesion molecule 1, and CD80/CD86 expression by DCs and other APCs. IL-10 plays an important role in human allergic disease. Asthma and allergic rhinitis are associated with diminished IL-10 expression in the allergic airway by both alveolar macrophages and DCs. Newer members of the IL-10 family include IL-19, IL-20, IL-22, IL-24, and IL-26; however, none of these cytokines significantly inhibits cytokine synthesis, an activity that remains unique for IL-10.[186] Treg populations can be classified into naturally occurring Foxp3+/CD4+/CD25+ Treg subset (CD4+CD25+ Treg) and antigen-driven Treg producing IL-10 (IL-10-Treg) and TGF-β (TGF-β-Treg). The production and action of these two cytokines are inter-related and likely involve a positive feedback loop, in which IL-10 enhances the expression of TGF-β and vice versa.[186–188]

INTRACELLULAR SIGNALING PATHWAYS OF INFLAMMATORY MEDIATORS

TLR Signal Transduction Pathways

Upon binding the ligands, TLRs activate the same signaling molecules that are used for IL-1R signaling,[189] triggering downstream signaling cascades that leads to the activation of pro-inflammatory cytokine and chemokine genes. Stimulation of most TLRs leads to Th1 rather than Th2 differentiation. Thus, innate immunity is a key element in the inflammatory response as well as the immune response against pathogens. Microbial cell-wall components stimulate immune cells and serve as PAMPs, recognized by individual TLRs. LPS, also known as an endotoxin, is generally the most potent immune-stimulant among these cell-wall components. Earlier studies have shown that endotoxin plays an important role in initiating and perpetuating of periapical inflammation and bone resorption.[190-193] LPS liberated from Gram-negative bacteria binds LPS binding proteins (LBP) that present in the bloodstream, and then binds to CD14 expressed on the cell surface of phagocytes. Subsequently, LPS is transferred to MD-2, which associates with the extracellular portion of TLR4, followed by oligomerization of TLR4, a key molecule of LPS signaling.[194,195] LPS does not have a direct apoptotic effect on osteoblasts or PDL cells, but can activate macrophages to induce apoptosis in osteoblasts and PDL cells via released TNF-α.[196]

NF-κB and NF-κB-Activating Pathways

The transcription factor NF-κB plays a pivotal role in a series of cellular processes, particularly those involved in inflammation, immunity, cell proliferation, and apoptosis. It consists of homo- or heterodimers of a group of five proteins, NF-κB1 (p50), NFκB2 (p52), p65/RelA, c-Rel, and RelB. In resting, NF-κB is in the cytoplasm and bound by the inhibitory proteins IkBs. Upon cell stimulation, IκBs are rapidly phosphorylated and degraded, freeing the NF-κB to translocate into the nucleus and bind onto the transcriptional regulatory sequence motifs of multiple target genes. NF-κB activation is divided into two main pathways: the classical and the alternative pathways of NF-kB activation. The classical pathway is induced by a variety of innate and adaptive immunity mediators including pro-inflammatory cytokines (TNFα, IL-1β), TLRs, and antigen receptors (TCR, BCR) ligation. While these NF-κB inducers signal through different receptors and adaptor proteins, all converge to the activation of IκB-kinase (IKK) complex, which phosphorylates IκBa leading to its degradation. The freed NF-κB then translocates into the nucleus where it activates the transcription of target genes such as cytokines, chemokines, adhesion molecules, and inhibitors of apoptosis (Figure 4-3). The alternative pathway of NF-κB activation is important for secondary lymphoid organ development, homeostasis, and adaptive immunity. It is induced by B-cell activating factor, lymphotoxin-β, CD40 ligand, and human T cell leukemia (HTLV) and Epstein-Barr (EBV) virus. It enhances NF-κB inducing kinase (NIK)- and IKKa-dependent processing of p100 into p52, that binds DNA in association with its partners, such as RelB. These stimuli also activate the classical pathway.

NF-κB-inducers trigger the formation of ROS, this may be the reason why such a diversity of inducers activate NF-κB via the same IKK-dependent pathway. Both IL-β and TNF-α activation of NF-κB involves ROS.

INFLAMMATION-INDUCED BONE RESORPTION

Formation of periapical lesion as the result of pulpal infection involves features that are somewhat different from a typical microbial infection of other tissues. Pulp tissue ends and transits into the periodontal ligament tissue at the apical cemento-dentinal junction. Apical periodontal ligament begins to be affected as the pulpal inflammation spread toward it. Inflamed apical portion of the pulp and the periodontal ligament releases factors that induce bone resorption to build and maintain a defense line against the incoming microbial invasion. The resorbed bone space is replaced with abscesses and/or granulomatous tissue rife

with inflammatory cells and fibroblasts. Proliferation of epithelial cell rest of Malassez leads to scattered epithelial cell clusters within the inflamed periapical lesion. With time, these epithelial cell clusters may form a cyst.[197–203] A true periapical cyst occurs in 15% of the periapical lesions examined based on the findings by Nair et al.,[203] who also found that 52% (133/256) of these lesions demonstrated epithelial proliferation. For more details, see Chapter 6.

The epithelial cells in the PDL are involved not only in the formation of cysts, but may also be in the regulation of cementum and bone metabolism as they express osteopontin and osteoprotegerin that are not expressed in gingival epithelial cells.[204] Based on animal studies, periapical lesions expand in the early phase of the lesion formation and become self-limited at later stage.

Microbial Role in the Pathogenesis of Periapical Lesion

Although inflammation is generally associated with infection, inflammation can be the mere result of an aseptic trauma or a deregulated immune reaction. The classical study by Kakehashi et al.[205] verified this notion that without the presence of microbes, exposed pulp tissue will heal after a transient inflammatory response without the development of periapical lesions. Endodontic infection is characterized as a polymicrobial infection with predominately anaerobes, many of which are gram negative. The damage of host tissues from infection is the result of 1) direct infliction from microbial products such as collagenase, trypsin-like enzymes, and fibronectin-degrading enzymes; and 2) host immune response that leads to destruction of tissue such as periapical bone.

As mentioned above, host innate immunity exerts certain extent of specificity to the microbial components via PRRs such as TLRs. 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 meningitis*. There is a reduced expression of IL-1 and IL-12 and decreased periapical bone destruction at 21 days, in TLR-4 deficient mice when subjected to pulpal exposure and infection with a mixture of four anaerobic pathogens, *Prevotella intermedia, F. nucleatum, Streptococcus intermedius* (G+), and *Peptostreptococcus micros* (G+). Also, no dissemination of infection occurs in the TLR-4 deficient mice.[206]

However, if the pulp is exposed to the microorganisms in the oral environment, these LPS hyporesponsive mice do not show significant difference in the level of cytokine production or periapical bone destruction from the normal controls.[207] Studies have indicated that the variations of microflora obtained from molar surfaces of mouse within a same experimental group are as great as the variation between mice in the different groups[208] that could contribute to the discrepancies of findings by different investigators.

Components such as LTA of Gram-positive bacterial cell walls can also stimulate innate immunity in a similar manner as 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 PG. The importance of TLR2 in the host defense against gram-positive bacteria has been demonstrated using TLR2-deficient (TLR2–/–) mice that were found to be highly susceptible to challenge with microbes,[209–211] and developed significantly larger periapical lesions with more osteoclasts than controls.[211]

LTA can cause apoptotic cell damage including that of *E. faecalis* that induces apoptosis in osteoblasts, osteoclasts, periodontal ligament fibroblasts, macrophages, and neutrophils. It also stimulates leukocytes to release mediators that are known to play a role in various phases of the inflammatory response including TNF-α, IL-1β, IL-6,[212] IL-8,[213] and PGE2. These factors have all been detected in periapical samples, and each has well-known tissue-damaging effects.[214]

Role of Immune Cells in Periapical Lesion Formation

Immune cells present in human periapical lesion consist of lymphocytes, macrophages, plasma cells, neutrophils, and natural killer (NK) cells with the former two types as the majority.[215,216] Within lymphocytes, T cells are in greater number or equal to B cells.[217] APCs and T suppressor/cytotoxic cells are associated with both pre-existing and newly formed epithelium.[216]

Using rodents as study models, the dynamics of periapical lesion development in rodents are characterized by having an active bone resorption phase within 15 days after pulp exposure followed by a chronic phase with little lesion expansion.[218–221] Th cells dominate during the active phase, whereas increased numbers of Ts cells are associated with chronicity. The subpopulations of specific Th cells involved in the periapical lesion development remain unclear.

The role of lymphocyte subpopulations in the formation of periapical lesions was studies using lymphocyte-deficient rodents. T cell-deficient rodents develop periapical lesion in a similar manner compared to the normal counter animals except some minor differences. Wallstom et al.[222] used athymic rat model (T cell-deficient) and found no significant difference between periapical tissue responses of the conventional and athymic groups. Tani et al.[223] used nude mice (T cell-deficient) and immunostaining to study the involvement of lymphocytes in the kinetics of periapical lesion development. They found that at 2 weeks after pulp exposure, nude mice develop larger periapical lesion than normal mice, but the lesion sizes are similar between the two experimental groups at 4 and 6 weeks after pulp exposure. At 8 week, however, normal mice have slightly larger lesion size. No T cells and reduced number of B cells are present in the lesions of nude mice. T cells (more Th than Ts) begin to emerge in lesions after 4 weeks in normal mice.[223] Fouad[106] employed severe combined immunodeficiency (SCID, T and B cell-deficient) mice as a study model and found that the SCID mice lesions are significantly smaller than the controls

at only the 3-week period. In these studies, pulp was exposed to oral microbes.

By using RAG-2 SCID mice (both T and B cell-deficient) and an infection protocol—equalized bacterial challenge of the exposed pulp with mixed microbes *Prevotella intermedia, F. nucleatum, P. micros,* and *S. intermedius* and the access sealed, Teles et al.[224] found that approximately one-third of the RAG-2 mice develop endodontic abscesses, while no immunocompetent controls have abscesses. In another study, specific knock-out (k/o) mice RAG 2, Igh-6 (B cell-deficient, Ig heavy chain k/o), Tcrb Tcrd (T cell-deficient, β & δ chain TCR k/o), and Hc^0 (C5 deficient) were used to determine which immune element is important for the defense mechanism in endodontic infection. Their results demonstrate that B cells, not T cells or C5, play a pivotal role preventing dissemination of endodontic infection.[225]

CD4+CD25hiFoxp3+ Treg cells are present in human periapical lesions expressing IL-10 and TGF-β. Tregs in the periapical lesions inhibit the proliferation of T-cells, at least in part, by the production of IL-10.[226] Various T cell subsets including Th1, Th2, Th9, Th17, Th22, Thf, Tr1, and Tregs have been examined in human periapical lesions by detecting the cytokines/markers expression. TNF-α, IFN-γ, IL-17A, and IL-21 levels are significantly higher in active lesions, while levels of IL-4, IL-9, IL-10, IL-22, and FOXp3 are higher than those in active lesions.[227] In a rat model, the presence of Th17 and Treg cells during development of periapical lesion was studied. IL-17–positive cells (Th17) markedly increased during early and mid-phase of active lesion expansion; whereas Foxp3-positive cells (Treg) remained at low levels until late phase and then dramatically increased thereafter.[48]

Mechanisms of Bone Resorption—Regulation of Osteoclast Formation and Activation

Under normal conditions, osteoclastogenesis mainly occurs within the bone marrow as a continuing process of bone modeling and remodeling. Compared to the generalized bone loss seen in osteoporosis, localized bone loss in jawbones resulting from endodontic infection is a combination of a focal inflammatory immune reaction and localized osteoclastogenesis. Inflammatory cells, including lymphocytes and macrophages, produce cytokines, such as tumor necrosis factor (TNF) and IL-1, and chemokines to recruit and activate more inflammatory cells. Lymphocytes, monocytes, and DCs found in periapical granulomas synthesize receptor activator of nuclear factor-κB ligand (RANKL). There is a significantly higher RANKL gene expression in the granulomas compared with the control tissues both in humans and rats.[7,228,229]

Macrophage CSF (M-CSF) and nuclear factor-κB ligand (RANKL) are two cytokines essential for promoting the differentiation of osteoclast precursors (OCPs) into mature osteoclasts. OCPs, generated in the bone marrow from hematopoietic stem cells, mobilize to diseased sites through

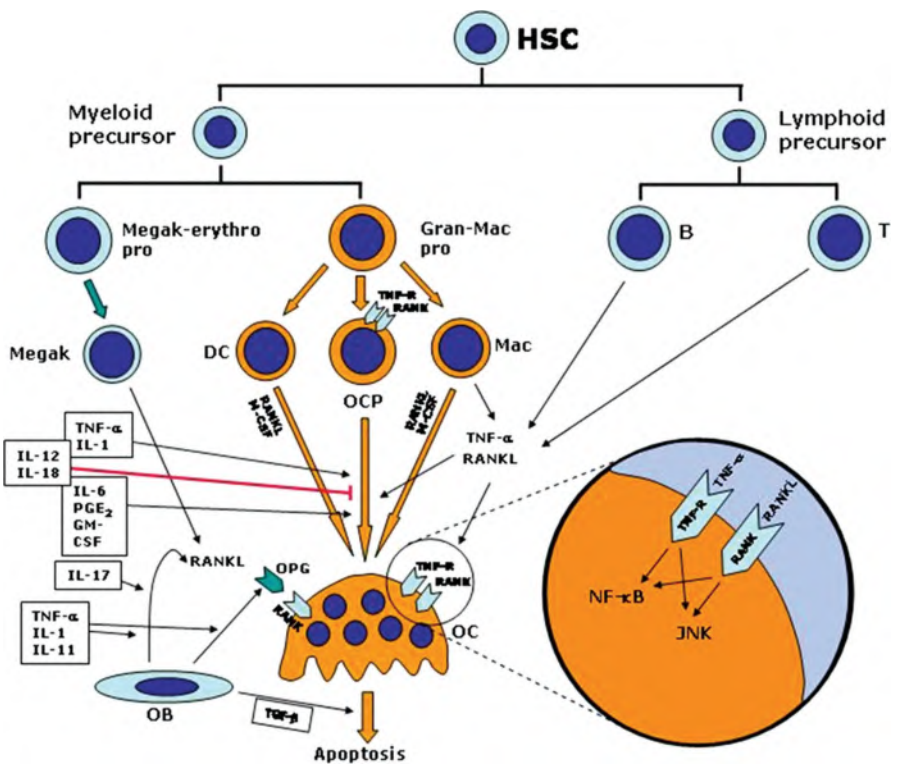

FIGURE 4-11 Regulations of osteoclast differentiation and the involved key cytokines. HSC: hematopoietic stem cells; OCP: osteoclast precursor; OC: osteoclast; OB: osteoblast; DC: dendritic cells; GC: granulocyte; Mac: macrophage; E: erythrocyte; OPG: osteoprotegerin.

the blood stream and differentiate into osteoclasts to resorb bone. OCPs migrate along chemokine gradients.

The RANKL/RANK system is essential for mature osteoclast formation, a critical player in T-lymphocyte-mediated osteoclastogenesis, and induces OCPs to produce pro-inflammatory cytokines and chemokines.[230–232] Mature B and T cells do not form osteoclasts but affect osteoclastogenesis indirectly by producing RANKL or osteoprotegerin (OPG), a soluble decoy RANKL receptor (Figure 4-11). OPG binds RANKL thereby preventing RANKL activity. OPG is expressed in human pulp of healthy and inflamed tissues[233]; however, its role in pulp tissue is unclear. The number of RANKL-positive cells and the ratio of RANKL/OPG in chronic apical lesion are significantly higher than those in healthy periapical tissues. The number of RANKL-positive cells is higher in lesions with severe inflammatory infiltration than in those with light inflammatory infiltration.[234] The influence of immune cells on osteoclastogenesis becomes significant when they accelerate osteoclast formation, and then both cell types contribute to the pathogenesis of the disease, as seen in inflammatory arthritis or periodontal disease.

The RANKL/RANK system is essential for osteoclast formation. RANKL not only delivers a final differentiation signal, but also activates osteoclasts and promotes their survival. RANKL is expressed by almost all cell types in the body. In the immune system, RANKL is expressed by activated T cells, B cells, and DCs. OPG is produced by cells of mesenchymal origin. RANKL and OPG expression is regulated by many factors, and the ratio of RANKL/OPG controls osteoclastogenesis. Periodontal ligament cells produce both RANKL and OPG.[235] In contrast to broad expression of RANKL, RANK, the receptor for RANKL, is only identified on mature osteoclasts, DCs, and OCPs, that are therefore the major target cells for RANKL and OPG. TNF is overproduced by many cell types at inflammatory foci, including macrophages, T cells, DCs, osteoclasts, and OCPs.[236]

Inhibition of Bone Resorption by Bisphosphonates

Bisphosphonates (BPs) have a unique high-affinity binding property to hydroxyapatite mineral, generating high local concentrations of drug on bone surfaces where these agents can preferentially interfere with osteoclast-mediated bone resorption. BPs comprise amino- and non-amino-containing compounds. Aminobisphosphonates such as pamidronate, alendronate, risedronate, and zoledronate exert their inhibitory effects on osteoclast function by inhibiting farnesyl pyrophosphate synthase, an enzyme in the mevalonate pathway necessary for lipid modification (prenylation) of small GTP binding proteins. Interference with the activity of the binding proteins alters cytoskeletal organization and intracellular trafficking in osteoclasts, resulting in inhibition of osteoclast function. Ruffled border formation is a process that is highly dependent on cytoskeletal function, strongly regulated by geranylgeranylated GTP binding proteins, such as Rac, Rho, and so on. Non-aminobisphosphonates such as etidronate and clodronate are metabolized to non-hydrolyzable analogues of ATP and act as inhibitors of ATP-dependent enzymes, leading to enhanced osteoclast apoptosis (Figure 4-12).[237]

The pathophysiological mechanism underlying ONJ may be multifactorial including altered bone remodeling, oversuppression of bone resorption, angiogenesis inhibition, soft tissue BPs toxicity, occurrence of constant microtrauma, inflammation or infection, and dento-alveolar surgery.[238–241] Precipitating factors of ONJ: severe periodontitis, spontaneous exposure, periodontal surgery, dental implants, and root canal surgery (in the order of prevalence).[242–244]

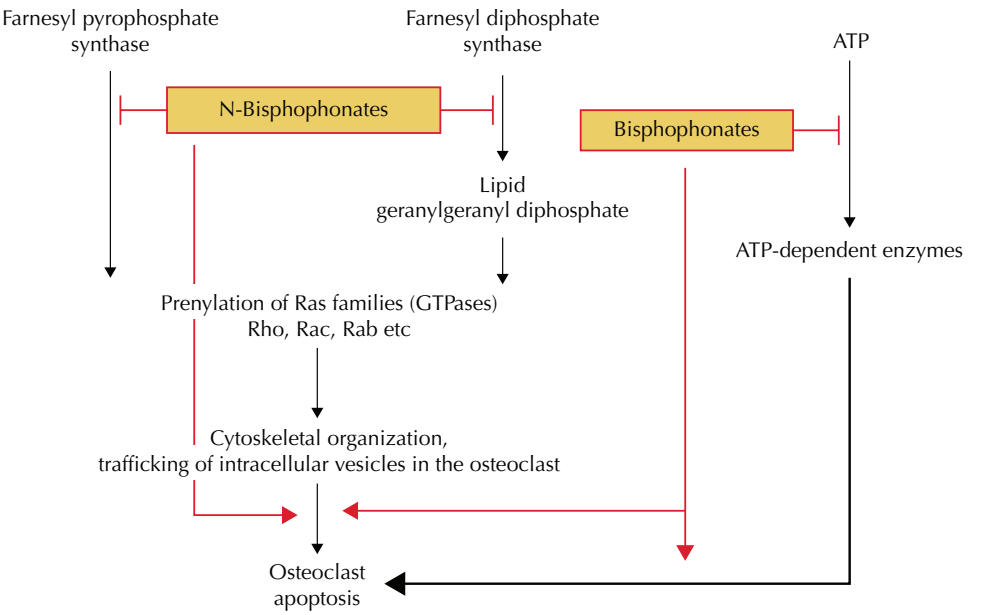

FIGURE 4-12 Metabolism of bisphosphonate (BP)-induced osteoclast apoptosis.

Roles of Cytokines in Periapical Bone Resorption

Many cytokines are involved in the initiation and maintenance of inflammation, some of which are also responsible directly or indirectly for the induction of bone resorption. Periapical bone resorption induced by cytokines is a destructive and an undesirable process indicative of the magnitude of endodontic infection. On the other hand, cytokines are also important for the establishment of local immune defense mechanism. The role of specific cytokines in a variety of immune and inflammatory diseases, including endodontic pathoses, is difficult to elucidate due to the intricate and overlapping functions of these inflammatory cytokines and mediators.

Cytokines TNFα, IL-1, IL-6, and IL-11 are involved in promoting bone resorption and mediating inflammation both systemically and locally. However, IFN-γ, IL-12, and IL-18 that may otherwise have pro-inflammatory actions, do not appear to augment periapical lesion development.[245] In human dental granulomas, there is a correlation between the intensity of inflammatory infiltrate and the percentage of mononuclear cells positive for IL-4, suggesting a predominant Th2 response. There is also a correlation between the frequency of cells expressing IL-6 and LT-alpha, suggesting a synergistic activities IL-6 and LT-alpha in granulomas. The numbers of inflammatory cells expressing the anti-inflammatory molecules far outnumbered the cells that expressed pro-inflammatory cytokines.[246,247]

In mice, TNFα and IL-1α are highly expressed in areas that contain a mixed inflammatory infiltrate or fibroblasts in periapical lesions. The number of cells expressing these two cytokines is proportional to the lesion size.[106] The expression of IL-11 was not modulated by pulp exposure. Most of the Th1-type cytokines, including IL-2, IL-12, and IFN-γ, showed an increase in mRNA and/or protein expression in periapical lesions after pulpal exposure; the expression of Th2-type cytokines (IL-4, IL-6, IL-10, IL-13) was similarly increased, but had declined at the latest time-point (day 28). There was a lack of correlation between IL-1a and Th2-type anti-inflammatory mediators, including IL-4, -6, and -10. These results indicate that a cytokine network is activated in the periapex in response to bacterial infection, and that Th1-modulated pro-inflammatory pathways may predominate during periapical bone destruction.[248–253]

The role of specific cytokines in the development of periapical lesions has been studies using mice with gene knock-outs of different cytokines or their receptors, such as IL-1, IL-6, and TNF.[208,254,255] Mice with functional deletions of receptors to IL-1 (IL-1RI(−/−)), TNF (TNFRp55(−/−)-p75(−/−)), or both (TNFRp55(−/−)-IL-1RI(−/−)) infected with multiple anaerobic pathogens after pulp exposure lead to increased polymorphonuclear and mononuclear phagocyte recruitment in the periracicular lesions and bacterial penetration into host tissue. Osteolytic lesion formation is also greater in animals lacking TNF and/or IL-1 receptors than in control mice indicating that IL-1 or TNF receptor signaling is not required for bacteria-induced osteoclastogenesis and bone loss, but does play a critical role in protecting the host against mixed anaerobic infections. Although TNFα enhances osteoclastogenesis and mediates lipopolysaccharide-induced bone loss, deletions of TNFRp55(−/−) does not reduce bacteria-induced bone resorption.[254] Between IL-1 and TNFα, IL-1 receptor signaling is more important than TNF receptor signaling in preventing the spread of infection into surrounding fascial planes.[256]

TABLE 4-10 Mediators That Promote or Inhibit Bone Resorption

Promote bone resorption	IL-1, TNF-α, PGE2, IL-6, IL-11, RANK/RANKL, M-CSF, IGF-I
Inhibit bone resorption	IL12, IL-18, IFN-γ, IL-4, OPG[258]
Dual functions	PGs, TGF-β, glucocorticoids

IL-6 production in mouse periapical lesions reaches its peak (~2-fold higher than base-line) on day 14 after pulpal exposure and then decreases to the basal level after 28 days.[250] Depleting IL-6 negatively affects the host defense mechanism against local infection in the periapical area.[208,255] In IL-$6^{-/-}$ mice, periapical lesions rapidly develop in week 2, whereas development of the lesion is one week slower in the IL-$6^{+/+}$ mice.[208] The increased bone resorption in IL-6-deficient animals correlated with increases in osteoclast numbers, as well as with elevated expression of bone-resorptive cytokines IL-1alpha and IL-1beta, in periapical lesions and with decreased expression of the anti-inflammatory cytokine IL-10. These data demonstrate that endogenous IL-6 expression has significant anti-inflammatory effects in modulating infection-stimulated bone destruction *in vivo*.[255]

The functional role of the Th2-type cytokines IL-4 and IL-10 in infection-stimulated bone resorption *in vivo* has been assessed using gene knock-out mice. IL-10(−/−) mice have significantly greater infection-stimulated bone resorption compared with wild-type mice, while IL-4(−/−) have no increased resorption. IL-10(−/−) mice show markedly elevated IL-1α production within periapical inflammatory tissues (>tenfold), whereas IL-4(−/−) exhibit decreased IL-1α production. These findings suggests that IL-10, but not IL-4, is an important endogenous suppressor of infection-stimulated bone resorption *in vivo*, likely acting via inhibition of IL-1α.[257]

The functional roles of the Th1 cytokine IFN-γ and IFN-γ-inducing cytokines IL-12 and IL-18 has also been determined with a similar approach using IL-12−/−, IL-18−/−, and IFN-γ−/− mice.[245] Although *in vitro* evidence has demonstrated that IL-12 and IL-18 have inhibitory effects on osteoclast formation,[258] no difference was found in the infection-stimulated periapical bone resorption between knock-out and wild-type control mice.[245] Therefore, it appears that there is a functional redundancy in pro-inflammatory pathways. A summary of bone-resorption related factors is presented in Table 4-10.

HYPERSENSITIVITY REACTIONS

Hypersensitivity reactions represent a group of immunological responses where the reaction observed by the host far exceeds that normally triggered in the immune response described before. This results in excessive tissue damage and even death in certain cases. There are four general types of immunological hypersensitivity reactions. They vary in their severity and extent from a mild subclinical response to a life-threatening reaction. It is important for the clinician to understand the underlying mechanisms of these conditions in order to recognize and manage these reactions in their patients effectively.

Type I: IgE-mediated degranulation of mast cells and basophils, with release of histamine. This may be either systemic (anaphylaxis) or local (atopy). Type I hypersensitivity is fundamentally caused by the initial sensitization of the patient to a particular allergen. Some of the common allergens here include penicillin, certain foods, pollen, and insect bite. Upon exposure to a second challenge to this allergen, cross-linking of IgE on the surface of mast cells occurs, and release of C3a and C5a complement molecules cause degranulation of mast cells and release of inflammatory mediators. The most important of these mediators is histamine; however, other mediators including eicosanoids, Th2 cytokines, and chemokines are released. The patient experiences vasodilatation, which may be local or systemic, as well as bronchoconstriction, which causes wheezing and may cause asphyxia. The patient is considered to have a medical emergency that should be immediately managed with subcutaneous injection of epinephrine. A proportion of patients with chronic exposure to allergens develop asthma.

Type II: Antibody-dependent cytotoxic hypersensitivity. Antigens on the cell surface can cause cell damage through the interaction with inflammatory cells that have FcγR or C3b receptors. Recall from the discussion of phagocytosis that this reaction primes the microbial surface by opsonins that aid in phagocytosis. In this case, however, phagocytosis does not occur, occasionally because the target is too large for phagocytosis such as for large target cells or in parasitic infections. Examples of type II reactions include blood factor (ABO) or rhesus (Rh) factor incompatibility and autoimmune hemolytic anemia.

Type III: Immune complex-mediated hypersensitivity. Excessive accumulation of antigen–antibody complexes in tissues, such as when there is excessive chronic accumulation of antigen, can stimulate an immune response through phagocyte-, complement-, and/or cytokine-mediated reactions. Frequently, neutrophils and macrophages attempt to perform phagocytosis, but due to the large amount of antigen, such as a microbial colony, they spill their proteases and oxygen- and nitrogen-reactive intermediates in the tissue causing tissue damage. Examples of this reaction include arthus reactions, serum sickness, systemic lupus erythematosus, and immune complex glomerulonephritis.

Type IV: Cell-mediated (delayed-type) hypersensitivity. This reaction is mediated by Th1, CD4+, or CD8+ cells, causing cytokine-mediated tissue damage. Examples of this reaction include allergic reactions to bacteria, viruses and fungi, contact dermatitis reactions, chronic granulomas, such as tuberculosis and leprosy. In the skin tuberculin test (Mantoux reaction), the subcutaneous administration of antigen causes accumulation of mononuclear inflammatory cells within 24 to 48 hours.

Other Types of Hypersensitivity

Stimulatory Hypersensitivity

The prime example of this reaction is Graves' disease, in which plasma cells continue to produce a self-antibody against the thyroid-stimulating hormone (TSH) receptor in the thyroid gland, which itself stimulates the gland to keep producing thyroxine, and does not respond to negative feedback mechanisms.

Innate Hypersensitivity Reactions

The severe disseminated reaction to LPS in Gram-negative bacteria is known as septic shock syndrome. In this reaction, LPS causes the release of systemic cytokines IL-1, TNF-α, and IL-6, which in excessive amounts activate compliment, cause respiratory distress by excess circulating neutrophils, and disseminated intravascular coagulation. Gram-positive septic shock has also been recognized due to enterotoxins of streptococci or staphylococci.

SYSTEMIC INFLAMMATION AS A CONSEQUENCE OF ENDODONTIC INFECTION

As noted before, during an acute infection or after tissue injury, there is an increase in the concentration of pro-inflammatory cytokines in the blood, primarily IL-1, TNFα, and IL-6. IL-1 and TNFα cause fever through their action on the thermoregulatory center in the hypothalamus due to the release of local eicosanoids.[14] IL-1 causes an increase in IL-6 that acts on hepatocytes to synthesize a number of plasma proteins called acute phase proteins, and the reaction is known as acute phase reaction. Among the most intensely secreted proteins are C-reactive protein (CRP), mannose-binding lectin (MBL), serum amyloid P component (SAP), and a1-acid glycoprotein. CRP and MBL are important in fixing complement and deposit C3b, that is opsonins, on the microbial cell.[6] SAP can, together with chondroitin sulfate, bind to lysosomal enzymes such as cathepsin B released in inflammation. This complex becomes a component of the amyloid fibrillar deposits that accompany chronic infections.[6] Other proteins that are released in moderate amounts in an acute phase reaction include fibrinogen, fibronectin, haptoglobin, angiotensin, ceruloplasmin, complement proteins C3, C9, and factor B, a1-proteinase inhibitors and a1-antichymotrypsin. Finally, cytokines may also act on bone marrow to cause the increased

production of white cells causing leukocytosis, where white cells may reach 15,000 to 20,000/mL rather than the 4,000 to 10,000/mL normal count.[9]

Acute endodontic infection can cause systemic inflammation with elevation of CRP and SAA.[259–261] These factors appear to subside shortly after the acute infection resolves.[259,260] Chronic periapical lesions in humans or animal models do not appear to be associated with CRP or SAA.[262,263] However, a number of other factors, including markers of inflammation, have been shown to increase with the presence of periapical lesions. Recently, a systematic review and meta-analysis in which the changes in 31 different systemic molecular markers were analyzed in both acute and chronic periapical infections.[264] The results showed that CRP, IL-1, IL-2, IL-6, asymmetrical dimethylarginine (ADMA), IgA, IgG, and IgM were increased in patients with apical periodontitis compared to controls. The meta-analyses further showed that: serum IgA, IgG, and IgM increased in humans with AP compared to healthy controls and that serum CRP, IgA, IgE, IgG, and IgM were not significantly different before and after treatment.

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Chapter 5

Pulpal Pathosis

Graham R. Holland, Tatiana M. Botero, Stephen B. Davis

The pulp may be subjected to a number of insults: microbial, chemical, physical, trauma, and iatrogenic.

Most pulps being treated are already damaged. It should be assumed that every tooth that is being restored due to caries or leakage has an inflamed pulp, and its treatment should be undertaken cautiously.

Microorganisms are responsible for most pulpal disease. The classic study by Kakehashi et al.[1] proved that exposed pulps in gnotobiotic (germ free) rats did not become inflamed, whereas similarly exposed pulps in rats with a full oral flora became inflamed. Dental caries and the pulpal inflammation beneath it are clearly microbial in origin. Pulpal injury beneath restorations is mostly microbial and not due to cytotoxicity of the materials. Microorganisms and their byproducts enter between the restoration and the dentin as a result of microleakage.[2–4] The toxins from the carious lesion reach and affect the pulp well ahead of the microbes themselves.

Antigen-recognition cells and antigen-presenting cells initiate the immune response either the innate (nonspecific), in naïve tissue, or the specific in tissue that has been previously exposed to the antigen. Vascular changes are an essential part of the response but these changes have to take place within hard tissue walls with the blood vessels entering from only one end.

DENTAL CARIES ARE THE MOST COMMON SOURCE OF BACTERIA AND THEIR BY-PRODUCTS AFFECTING THE PULP

Dental caries begins beneath a biofilm of dental plaque when environmental factors favor the growth and metabolism of acidogenic bacteria. The population of bacteria that are present in carious lesions is mixed and variable. Chhour et al.[5] demonstrated the presence of 75 species in 10 samples of carious dentin. Up to 31 taxa were represented in each sample. A diverse collection of lactobacilli were found to comprise 50% of the species, with prevotellae also being abundant, comprising 15% of the species. In another study, significant positive associations were found between *Micromonas micros* and *Porphyromonas endodontalis* detection and inflammatory changes in the pulp.[6] These anaerobes have been strongly implicated in endodontic infections that occur as sequelae to carious pulpitis. Accordingly, the data suggest that the presence of high levels of these bacteria in carious lesions may be indicative of irreversible pulpal pathosis. In a clinical setting, however, it is very difficult to estimate the nature and size of the tissue injury.

Diverse groups and different proportions of species are found in carious lesions. Thus, it has not been possible to produce an antibiotic to inhibit the progress of caries or a vaccine to prevent it. The bacteria inhabiting the margins between restorations and dentin are similarly highly variable. A variety of products are released or formed on the death of the bacteria. These include acids and proteinases that dissolve and digest the enamel and dentin, as well as toxins including lipopolysaccharide (LPS) and lipotechoic acid (LTA). LPS results from the breakdown of the walls of gram-negative bacteria and LTA from the breakdown of gram-positive bacteria. As gram-negative bacteria predominate in the plaque over carious lesions, it may be assumed that LPS is the primary toxin. LPS is a generic term and there are many variations of its molecular structure, and differing virulence, depending on the species of bacteria producing it. The speed of progress of both dental caries and pulpal inflammation is variable, perhaps as a result of variations in the quantity, nature, and combination of toxins.

Although bacteria can themselves readily travel within dentinal tubules (Figure 5-1), toxins pass through dentin and enamel well ahead of them. Thus, the inflammatory response of pulp is to the toxins rather than to the bacteria themselves. Bacteria only invade the pulp at a late stage of the caries process that clinically would present as a carious exposure. For more details, see Chapter 3.

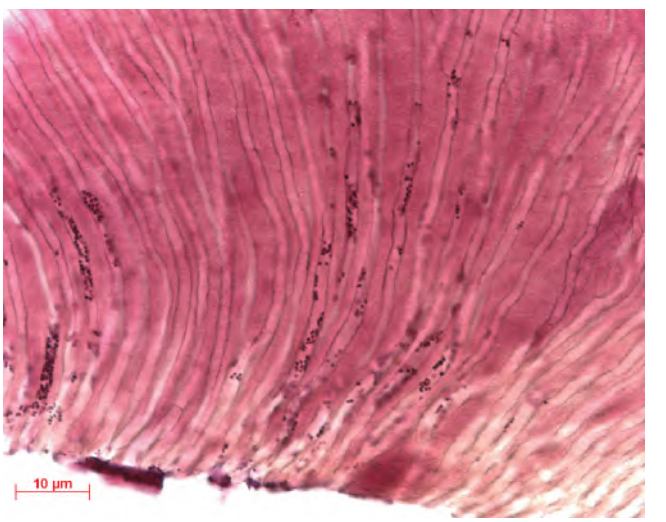

FIGURE 5-1 Bacteria in dentinal tubules in vitro. (Brown and Brenn[197]). (With permission from Dr. Christine M. Sedgley, Portland, OR, U.S.A.)

BACTERIA AND THEIR BY-PRODUCTS CAN REACH THE PULP BY MICROLEAKAGE AROUND RESTORATIONS AND FROM OTHER SOURCES

Anomalous Crown Morphology, Fractures, and Cracks

Any feature that allows the accumulation of bacteria will increase the likelihood of pulpal inflammation. In most cases, the inflammation will be secondary to caries. In cracked teeth, if they are symptomatic, the cracks will be lined with bacteria that have easy direct access to the pulp (Figure 5-2).[7]

Periodontal Disease

Biofilms form on the root surface of periodontal pockets. Their flora is similar to dental caries and would, presumably, release similar toxins. The presence of caries itself is not commonly found in this situation, but toxins may enter the pulp via lateral canals or by diffusing along dentinal tubules. For more details, see Chapter 36, "Endodontic–Periodontal Interrelationship." The outward flow of dentinal fluid would oppose this, and the relative impermeability of radicular dentin would prevent or reduce it. It has been demonstrated by the examination of extracted teeth with periodontal disease, but no caries, that pathologic changes do occur in the pulp when periodontal disease is present.[8]

On removing the cementum layer, periodontal scaling might be thought to allow greater ingress of microorganisms and toxins. This has not been supported experimentally.[9] The fact that the pulp has considerable powers to oppose invasion via the dentinal tubules has been clearly demonstrated by observations showing bacteria invading dentinal tubules of devitalized teeth much more readily than the tubules of vital control teeth.[10] Teeth with periodontal disease reaching the apex often remain vital. The consensus seems to be that periodontal diseases may lead to some pulpal changes but they rarely, if ever, lead to necrosis unless there is apical involvement.

FIGURE 5-2 Bacteria within a crack in dentine extending from the crack into dentinal tubules (Brown and Brenn). (With permission from Dr. Henry Trowbridge, San Francisco, CA, U.S.A.)

Blood Stream (Anachoresis)

Bacteria in circulating blood may be deposited in the pulp. An experimental study on dogs, in which some pulps were exposed and capped and then bacteria released into the blood stream, showed accumulations of bacteria in the capped pulps but not in normal pulps.[11] Whether this phenomenon is of clinical significance is unknown.

THE IMMUNE RESPONSE IN THE DENTAL PULP IS THE SAME BASIC PROCESS THAT OCCURS ELSEWHERE BUT IN A UNIQUE ENVIRONMENT

All connective tissues are capable of mounting an immune response that will follow the same basic pattern but is affected by local factors. The pulp's response differs from that in some other tissues. Because bacteria enter the tissue only at a very late stage, the pulp's blood supply is limited and the tissue is within a low-compliance chamber. If the tissue is presented with a new antigen, the innate immune response is initiated and is followed by the specific response a few days later. If the tissue is presented with an antigen it has met before, the specific response will be initiated immediately.

The tissue response to foreign antigens was described in molecular terms in Chapter 4, "Inflammation and Immunological Responses." The current chapter relates these processes to histologic and physiologic changes in the pulp and to the tissue's attempts at repair.

ANTIGEN RECOGNITION IN THE DENTAL PULP

All three antigen-presenting cell types expressing the type II major histocompatibility complex (MHC) surface proteins, macrophages, dendritic cells, and B lymphocytes are present and active in pulp's response to bacteria and toxins. In addition, it is now becoming clear that odontoblasts, being in the ideal position to detect antigens via Toll-like receptors (TLRs), produce a variety of cytokines and chemokines.[12]

In a normal healthy pulp, *macrophages* (Figure 5-3)[13] are present in a resting form, as monocytes. Macrophages require stimulation by microorganisms or cytokines before they express type II MHC molecules.[14] At rest, they are found predominantly around blood vessels,[15] although a few are distributed throughout the tissue.

Dendritic cells (Figure 5-4) are characterized by the shape that provides their name. They form a network throughout the pulp concentrating around blood vessels (Figure 5-5)[15,16] and the odontoblast layer. Some of the dendritic cells in the odontoblast layer extend their processes into the dentinal tubules (Figure 5-6).[17] They constantly express the MHC molecules on their surface without provocation. Recent studies[18] indicate that there are two populations of dendritic cells in the dental pulp, one beneath the odontoblast layer and the other around the blood vessels (Figure 5-7). The number

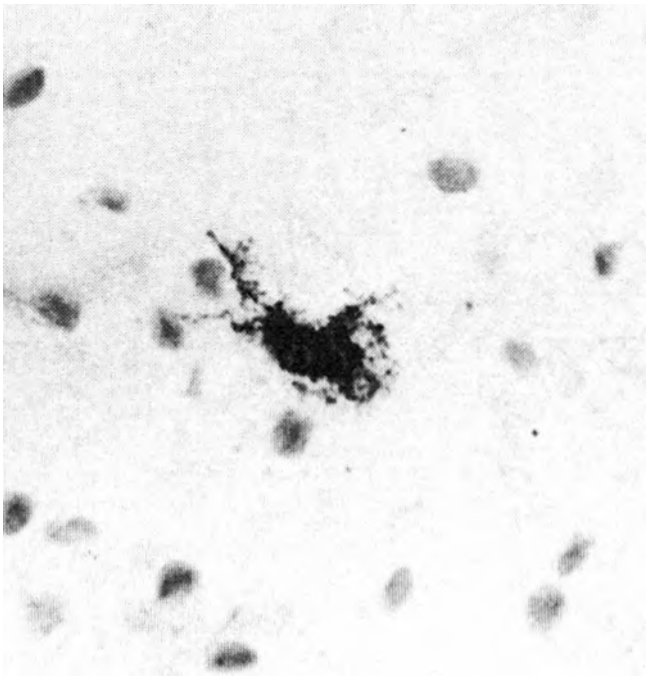

FIGURE 5-3 Macrophage from the central pulp (Immunohistochemistry). (Reproduced with permission from Jontell M et al.[13])

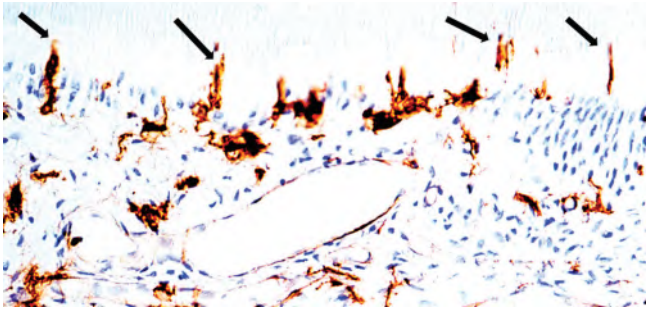

FIGURE 5-4 Dendritic cells (brown) in and around the odontoblast layer. Some dendritic cells (arrows) have processes extending into dentinal tubules (Immunohistochemistry with blue counterstain). (Reproduced with permission from Yoshiba K et al.[17])

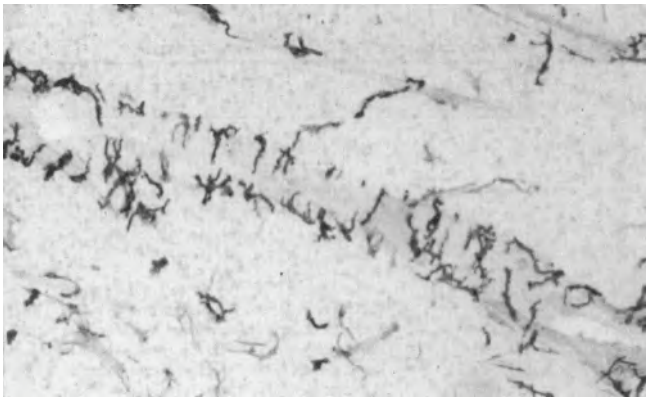

FIGURE 5-5 Dendritic cells surrounding and adjacent to blood vessels. Immunohistochemistry. (Reproduced with permission from Jontell et al.[13])

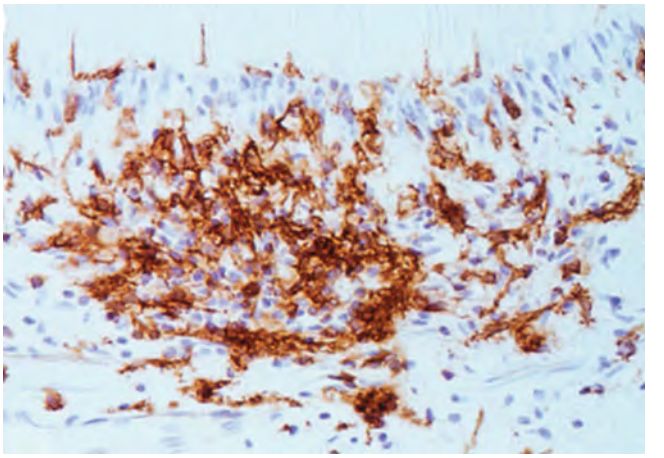

FIGURE 5-6 Dendritic cells (brown) congregating in an area of inflammation. (Reproduced with permission from Yoshiba et al.[17])

of dendritic cells increases in the pulp when it becomes inflamed and they accumulate beneath the carious lesion (Figure 5-6).[17]

B cells are specialized lymphocytes that will become plasma cells. They secrete antibodies during the specific immune response and express the MHC molecules. They have also been reported in the normal pulp but are rather rare.[19,20] Their role in the initial stages of antigen recognition and presentation in the pulp is unclear. Occasional T cells are found in normal pulp (Figure 5-8) and may be activated by antigen-presenting cells locally.

In the pulp, there is a close anatomic relationship between nerve fibers and dendritic cells (Figure 5-9), and both increase in parallel when the pulp is inflamed.[17,21] The sympathetic nervous system has recently been shown to have a modulating influence on pulpal inflammation.[22–24] The sympathetic system inhibits the production of proinflammatory cytokines, although stimulating the production of antiinflammatory cytokines.[22] In addition, T lymphocytes and other leukocytes produce antinociceptive molecules such as β-endorphin and somatostatin during inflammation (Figure 5-10), which reduce the excitability of pain fibers.[25]

Odontoblasts are the first cells to encounter an antigen diffusing along the dentinal tubules. Veerayutthwilai et al.[12] described how several markers of innate immunity have been demonstrated on odontoblasts and that their processes include chemokines and TLRs. Odontoblasts respond differentially to the toxins produced by gram-positive and gram-negative bacteria. A gram-negative toxin activating TLR-4 up-regulates mRNAs for interleukin-1β, interleukin- 8, tumor necrosis factor-α, and chemokines and TLRs. A synthetic peptide mimicking a toxin from gram-positive bacteria activating TLR-2 downregulates these mRNAs. Interleukin-1β, tumor necrosis factor-α, and chemokine (C-C motif) ligand 20 were also up-regulated from sixfold to 30-fold in odontoblast preparations from decayed teeth. These data show that odontoblasts express microbial pattern recognition receptors in situ, allowing differential responses to gram-positive and gram-negative bacteria. They also suggest that pro-inflammatory cytokines and innate immune

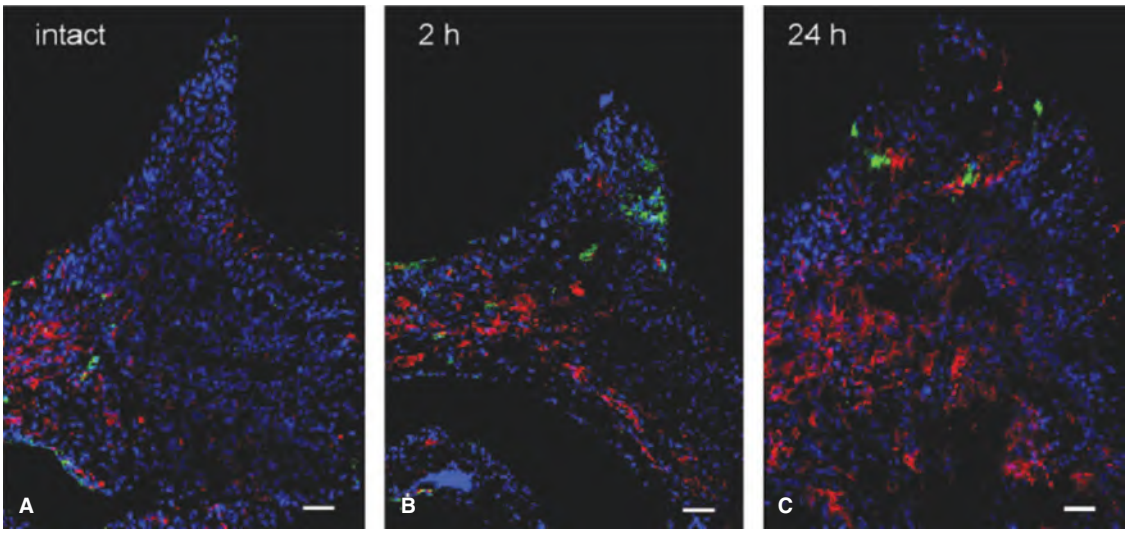

FIGURE 5-7 Two different types of dendritic cells (here stained red or green) differentiated by immunohistochemistry. The nuclei of odontoblasts and fibroblasts are stained blue. In this experiment, the pulp was injured and the three images were taken at different time points after the injury. (Reproduced with permission from Zhang et al.[18])

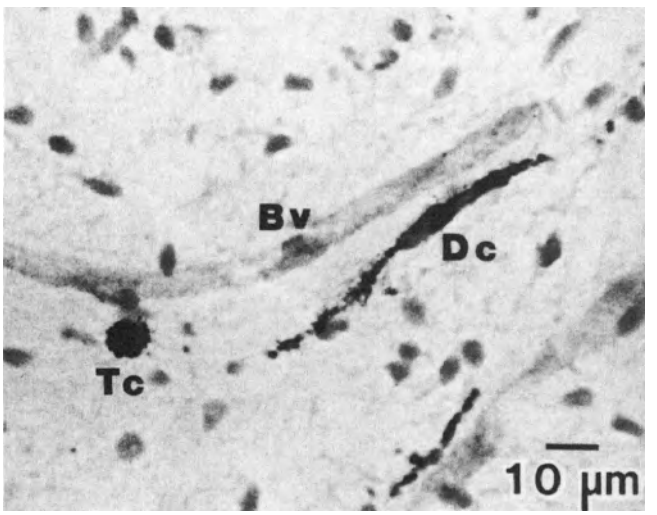

FIGURE 5-8 Dendritic cell (Dc) and a T-helper cell (Tc) adjacent to a blood vessel in the pulp (Immunohistochemistry). (Reproduced with permission from Jontell et al.[13])

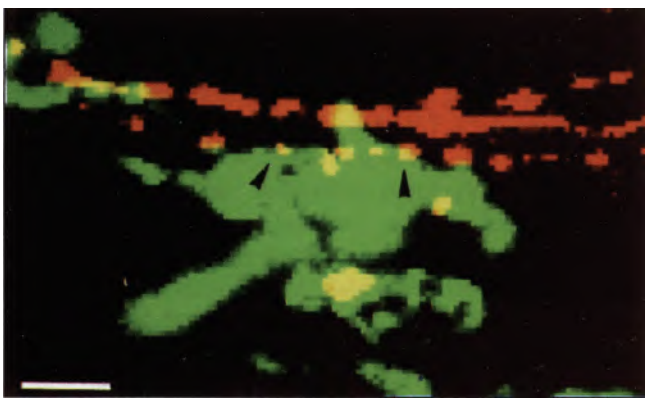

FIGURE 5-9 Confocal microscope image of a dendritic cell (red) and a nerve fiber (green). Where they are close together is yellow. (Reproduced with permission from Okiji et al.[15])

responses in decayed teeth may result from TLR signaling (Figure 5-11).[12] Other studies on odontoblasts *in vitro* suggest that in response to a bacterial toxin they produce chemokines that attract dendritic cells.[26]

Process of Antigen Recognition

Dendritic cells and macrophages bind to phagocyte antigen that is then processed intracellularly, bound to MHC molecules, and moved to the cell membrane for recognition by T cells. B cells bind antigen to specific cell surface receptors. All the antigen-presenting cell types enter the blood stream and carry the surface molecules to the lymph nodes where T cell activation takes place, although some may occur locally (Figure 5-8).[16] Each type of antigen-presenting cell carries different types of antigen. The T cells respond not only to the antigen but also to the modified complex in the cell membrane of the antigen-presenting cells. Being stationary, the odontoblast does not participate directly in the activation of T cells but, presumably, activates dendritic cells.

HISTOLOGIC CHANGES IN THE EARLY STAGES OF CARIOUS ATTACK DURING THE NONSPECIFIC IMMUNE RESPONSE ("ACUTE INFLAMMATION")

The classic description of the histology of the inflamed dental pulp beneath caries was published by Seltzer et al. in 1963.[27] They attempted, with limited success, to find a relationship between the clinical presentation of the patient and the histology of the pulp. In doing so, they looked at many examples of pulpitis at many different stages. This and other studies[19,20] have made it possible to put together a hypothetical description of pulpal changes during the progression of dental caries.

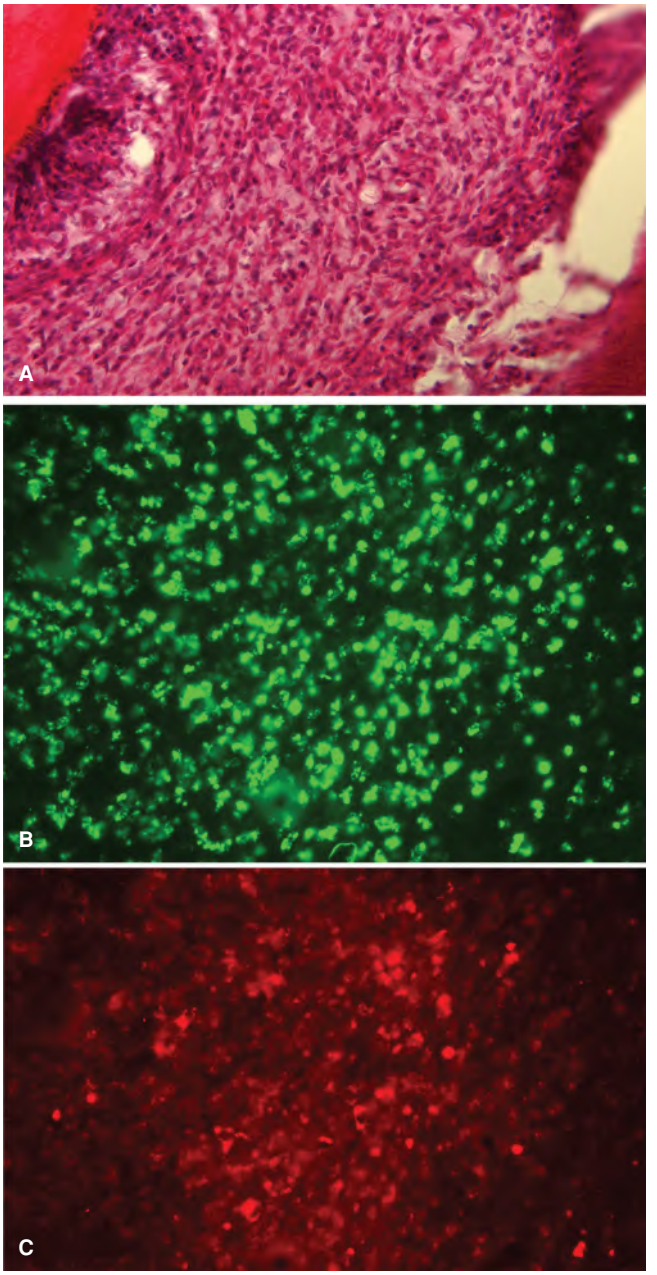

FIGURE 5-10 ***A.*** Area of inflammation in a pulp exposed 1 week earlier and left untreated (hematoxylin and eosin). ***B & C.*** Same section of the pulp photographed through different color filters after immunofluorescent staining. In ***B,*** the staining for β-endorphin is seen and in ***C,*** the staining for T lymphocytes. Some cells are labeled by both stains indicating that T lymphocytes produce β-endorphin. (Reproduced with permission from Mudie and Holland.[25])

The description is very much hypothetical as dental caries is a highly variable disease and its progress has never been followed continuously in a single tooth. This description was obtained largely by examining paraffin-embedded sections stained with hematoxylin and eosin under the light microscope. More recently, other approaches have been used, particularly immunohistochemistry, electron microscopy, and molecular techniques. However, the original microscopic description provides a framework on which newer data may be hung.

Cariogenic bacteria in the dental plaque produce a mixture of acids and enzymes that dissolve the mineral elements of enamel and dentin and then digest the organic matrix. The initial removal of mineral makes the enamel more permeable and the bacterial toxins will diffuse well ahead of cavitation. Once the dentin is reached, the toxins and, much later, the bacteria themselves will travel along the dentinal tubules (Figure 5-1). Clearly, variations in the composition and thickness of enamel and dentin, and particularly the patency of the dentinal tubules, will determine the rate at which these toxins reach the pulp. In vital teeth, this movement will be opposed by the outward flow of dentinal fluid. However, toxins reach the pulp at a very early stage relative to surface changes. Brannstrom and Lind[28] looked at a large number of extracted teeth and found that in ~50% of teeth with white spot lesions and no cavitation, there was histologic evidence of inflammation in the underlying pulp (Figure 5-12).

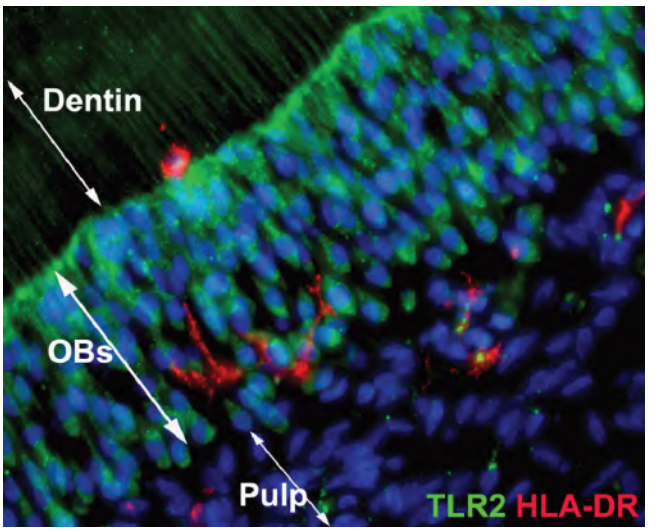

FIGURE 5-11 Immunohistochemistry showing TLRs (green) for bacterial components on odontoblasts (blue nuclei). Some dendritic cells are also stained (red). TLRs when activated by bacterial components lead to the release of chemokines that participate in the immune response. (Reproduced with permission from Zhang et al.[18])

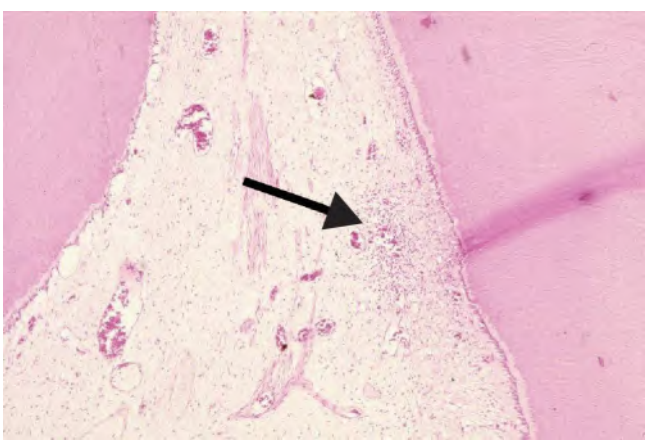

FIGURE 5-12 Early inflammatory response (arrow) beneath a non-cavitated carious lesion (hematoxylin and eosin).

The first cells contacted by the toxic bacterial broth, diffusing down the dentinal tubules, will be the odontoblasts. Some studies describe structural changes in the odontoblast layer even at an early stage. This may not be truly happening when seen in hematoxylin and eosin-stained sections. All histologic procedures are subject to artifacts, and man-made changes are not linked to any biologic change. Teeth that have been examined in this way have probably been extracted with forceps and compressed in the process, causing aspiration and damage to the odontoblast layer. Teeth are then fixed, usually in formaldehyde, which often causes the pulp to shrink away from the dentin. The later stages of preparation, demineralization, embedding in wax, and sectioning may also cause damage (Figure 5-13). From studies using other techniques, we now know that odontoblasts respond to toxins and can create a reactionary dentin barrier. It is more than likely that the morphologic changes reported in the odontoblast layer early in the carious process are artifactual.

With hematoxylin and eosin-stained sections, dendritic cells cannot be distinguished, but macrophages and neutrophils can be distinguished. The macrophages (and dendritic cells) will recognize a foreign antigen and carry its details to the lymph nodes, although this is a histologically invisible process. Resting macrophages (monocytes) enlarge and become active. Neutrophils leave the blood stream by diapedesis and migrate to the scene of action (Figure 5-14). This accumulation of phagocytic cells is dominated numerically by neutrophils, recognizable by their lobed nuclei, and is the key morphologic feature in the early response to caries. Mast cells may also be present,[29,30] but they are difficult to detect in routine preparations due to their extreme fragility. They are most common in surface tissues in response to inhaled or ingested antigens. At the same time, vascular and then neural changes (described elsewhere in this chapter) occur. Nerves are not well visualized in hematoxylin and eosin-stained sections, and observations of changes in them during inflammation require other techniques. Sometimes an apparent dilation of local blood vessels may be seen.

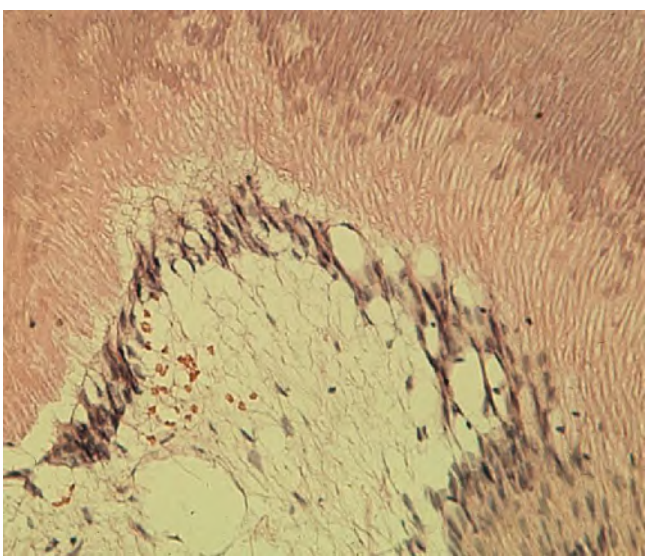

FIGURE 5-13 Poorly fixed section of pulp and dentin. The vacuoles and disruption of the odontoblast layer are artifacts (hematoxylin and eosin).

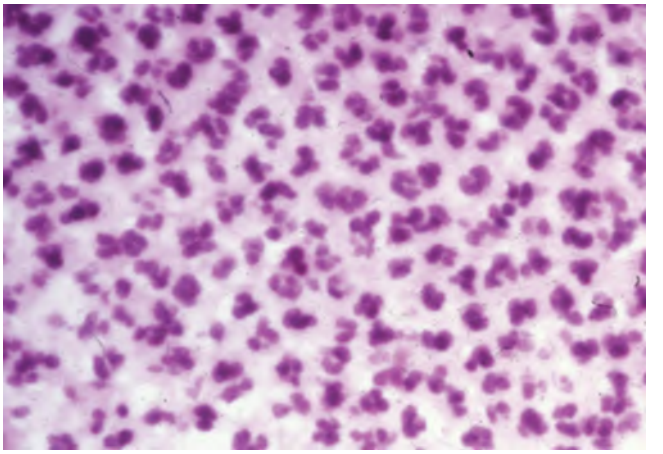

FIGURE 5-14 Polymorphonuclear leukocytes in early inflammation (hematoxylin and eosin). (With permission from Dr. Henrry Trowbridge, San Francisco, CA, U.S.A.)

HISTOLOGIC CHANGES IN THE LATER STAGES OF CARIOUS ATTACK DURING THE SPECIFIC IMMUNE RESPONSE ("CHRONIC INFLAMMATION")

The immediate "inflammatory" phase of the immune response begins very shortly after the antigen arrives in the tissue. If the body has been exposed to the antigen on a previous occasion, then the record of that exposure will have been maintained by the memory T cells. The production of lymphocytes synthesizing specific antibodies will begin very quickly (within a few hours). If the antigen has not been encountered before, new clonal lines of lymphocytes will be developed, which takes several days. In either case, the production of specific lymphocytes will take place in the lymph nodes. With a known antigen, however, there may also be a second challenge, a local interaction between antigen-presenting cells and resident T lymphocytes.[16]

Whether the antigen is new or returning, the histologic appearance of the tissue will be the same, only the time scale will be different. At this stage, the appearance of the pulpal response is characterized by the dominating presence of lymphocytes (Figure 5-15). The numbers of lymphocytes present increases with the severity of clinical symptoms.[21]

A commonly used definition of chronic inflammation, often called a granuloma, is "inflammation of slow progress marked chiefly by the presence of lymphocytes and the formation of reparative tissue" (Figure 5-15). This is commonly seen in the dental pulp of extracted carious teeth, some of which may have been asymptomatic.

The pulp is a remarkably robust tissue and can, by the production of tertiary dentin, protect itself from persistent

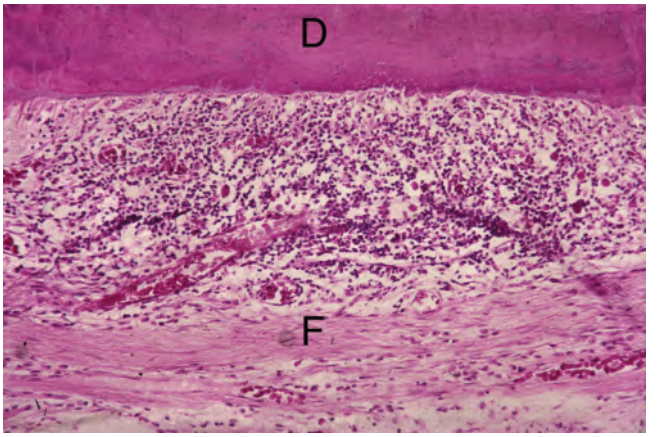

FIGURE 5-15 Chronic inflammation (D = dentin). An attempt at fibrous repair (F) is made at the same time as inflammation. The inflammatory cells are predominantly lymphocytes (hematoxylin and eosin). (With permission from Dr. Henry Trowbridge, San Francisco, CA, U.S.A.)

carious attacks. If the carious lesion is not treated, however, the increasing quantity of irritants will eventually cause irreversible changes. These are at first limited in size and may even form a "pulpal abscess." The bulk of the tissue around it appears healthy (Figure 5-16). This local necrosis, unless checked, will progress gradually throughout the tissue and into the periradicular tissues. The progress of tissue damage in the pulp is determined by the presence and spread of microbial toxins and is not due to the "strangulation" of blood vessels.

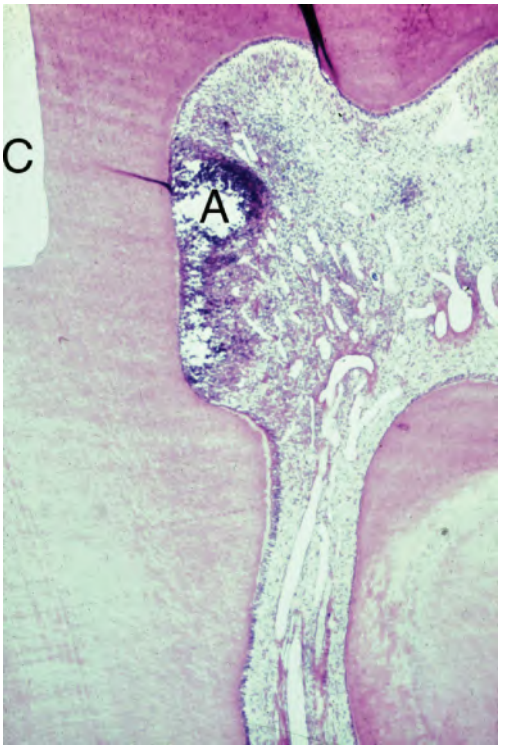

FIGURE 5-16 Pulpal abscess (A) beneath a deep cavity preparation (C). Away from the diseased area, the appearance of the pulp is normal (hematoxylin and eosin). (With permission from Dr. Henry Trowbridge, San Francisco, CA, U.S.A.)

HEMODYNAMIC CHANGES IN THE PULP DURING CARIES

An understanding of the vascular component of the immune response in the dental pulp is critical to planning or executing vital pulp therapy. Unfortunately, there are relatively few studies on pulpal vascular changes under carious lesions in humans.[31–36] Several investigators have looked at the effect of experimentally induced inflammation in animal models.[37–41] There are some excellent reviews of the vascular physiology of the dental pulp[40] as well as the description given in Chapter 2, "Structure and Function of the Pulp–Dentin Complex."

Blood Flow

Measuring blood flow in the dental pulp, even in experimental animals, is a difficult procedure, and data generated are a challenge to interpret. Using plaque extract to initiate inflammation in a rat incisor model, Kim et al.[42] reported a 40% increase in blood flow in a "moderately inflamed" pulp but a 35% reduction in a "partially necrotic" pulp. Another study by Bletsa et al.,[41] in a similar model, showed that the application of LPS to the pulp resulted, after 10 minutes, in a reduction in blood flow that continued for the 3-hour duration of the experiments. This was interpreted as a limited ability of the pulp to respond.

Interstitial Fluid Pressure

The healthy dental pulp has an interstitial pressure of 5 to 10 mm Hg.[37,43] One of the key changes during inflammation is the movement of fluid from within the capillaries into the interstitial space. By the laws of physics, increasing the amount of fluid in a rigid chamber leads to an increase in pressure. It was assumed, for a long time, that there was such a pressure rise in the pulp and that it would cause compression of the blood vessels leading to vascular stasis and necrosis. It was even suggested that this pressure change could lead to strangulation of the vessels at the apex causing necrosis in areas of the pulp not directly affected by the bacterial toxins.

Experimental work by Tonder and Kvinnsland[37] (later Heyeraas) and Van Hassel[43] measured interstitial fluid pressure in the pulps of experimental animals and showed that although the pressure increased in the area immediately beneath an injury a short distance (2 mm) away, the pressure stayed within normal limits. Even within areas of raised interstitial fluid pressure, capillaries remain patent because of a balancing increase in intra-luminal pressure. The increased interstitial fluid pressure may be beneficial as, when combined with increased capillary permeability, it would lead to an accelerated rate of fluid exchange across the capillary membrane.[40] Any net increase in tissue fluid, especially macromolecules, will be removed by the lymphatics. The presence of lymphatics in the dental pulp has been controversial, but studies using ultrastructural and histochemical techniques

have established their existence.[44–46] Necrosis occurs beneath persisting carious lesions only when bacterial toxins poison cells directly, spreading throughout the pulp.

Changes in blood flow and pressure are an integral part of the immune/inflammatory response and thus a component of the normal process of limiting the extent of damage and initiating repair. The detailed physiology of the microcirculation is complex and varies considerably between tissues (e.g., brain, kidney).[41] In the dental pulp, it is particularly difficult to study. This contributes to the difficulties of precise diagnosis. Although there have been many attempts to measure pulpal blood flow clinically, none has been validated as completely accurate.

NEURAL CHANGES DURING PULPAL INFLAMMATION

Sympathetic nerves control blood flow by constricting precapillary sphincters[47–49] and by interacting with other elements of inflammation. In other sites, sympathetic activity inhibits the production of proinflammatory cytokines although stimulating the production of anti-inflammatory cytokines[50] and is involved in the recruitment of inflammatory cells to the area.[23,51,52] Sympathetic nerves have an inhibitory effect on odontoclasts and stimulate reparative dentin production, although they have no apparent effect on the degree of inflammation induced by mustard oil.[53] There is less abscess formation 4 days after deep cavity preparation in normal teeth when compared with sympathectomized teeth.[51] Nor are there differences in the extent or severity of inflammation after pulp exposure injury in sympathectomized rat molars after a waiting period of >20 days.[51]

Afferent sensory fibers from the trigeminal system also play an important role in response to toxins and injury. The afferent fibers release two important neuropeptides, substance P and calcitonin gene-related peptide (CGRP). Both cause vasodilatation and increased capillary permeability. Sympathetic activity causes vasoconstriction and also reduces the release of peptides from afferents. Thus, the sympathetics and the afferents seem to act in opposing directions.

In injured pulps, there is an increased expression of nerve growth factor (NGF) and its receptors.[54,55] A concomitant sprouting of the afferent terminals and increased presence of substance P and CGRP[55–60] also takes place. Sprouting of sensory nerve fibers occurs as early as 1 day after dental injury[61] and decreases 3 to 4 weeks after pulp exposure injury, a time when sympathetic nerve sprouting occurs.[62–65]

Pulpal injury and inflammation are also associated with neural changes outside the pulp itself. In the trigeminal ganglion after pulpal injury, the expression of various neuropeptides increases,[66,67] Trk-B receptors (associated with pain) increase,[68] and nitric oxide synthesis is enhanced.[69] There are also detectable changes in the supporting cells of the ganglion.[70] In the trigeminal nucleus (pars caudalis has been the most studied), pulpal injury and inflammation lead to degenerative changes[71] and to cFos expression, an indication of possible changes in the pain system.[72] Perhaps, most significantly from a clinical point of view are changes in the nucleus related to central sensitization.[73–77] This may help explain the variable presentation of pulpitis in terms of pain. For more details, see Chapter 7, "Dental Innervations and Pain of Pulpal Origin."

ANTI-INFLAMMATORY AND ANTINOCICEPTIVE MECHANISMS IN THE DENTAL PULP

During inflammation, there are important interactions between the nervous and immune systems.[78] Neuroimmune interactions control pain through the activation of opioid receptors on sensory nerves by immuno-derived opioid peptides.[79] Opioid-containing leukocytes migrate to peripheral sites of inflammation. On exposure to stress, opioid peptides are released, bind to opioid receptors on peripheral sensory neurons, and induce endogenous antinociception. Although much of the research in this area has been done on skin and joints (in relation to arthritis), the efficacy of adding as little as 1 mg of morphine to the local anesthetic used in oral surgery has been demonstrated.[80] Although no published studies focus directly on the effect of local opioids on pulpalgia, the underlying elements have been demonstrated. Opioid receptors are present on pulpal nerves[22,81] and β-endorphin has been shown to be present in, at least, lymphocytes in the inflamed pulp (Figure 5-10).[25] For more details, see Chapters 2 and 7. It is important to see this as a part of the pulpal response to injury. The extent of this antinociceptive mechanism may be variable depending on, for one possibility, the exact nature of the antigen presented. This may, in part, explain the wide variation in the pain experienced as a result of pulpal inflammation.

LESS COMMON PULPAL RESPONSES: CALCIFICATION AND RESORPTION

Calcification of the pulp takes a variety of forms. Discrete pulp stones occur in a large proportion of the population. In a study of young adults (dental students), 46% of the group had radiographic evidence of pulp stones in at least one tooth. Ten percent of all the teeth included in that study contained a pulp stone.[82]

The vast majority of pulp stones are found in molars (Figure 5-17). Pulp stones can be classified as true or false as they can resemble tubular regular dentin or bone with cells embedded in it. They are also classified as free, adherent, or embedded when they become part of the dentinal walls (Table 5-1).[83] Although the etiology of pulp stone formation is unknown, there is evidence that pulp stones are more common in patients with atheromatous cardiovascular disease (74%) than in those without (39%).[84] Pulp stones are not considered pathological, but act as an obstacle in locating and instrumenting root canals.

A higher incidence of pulpal mineralization is associated with some genetic disorders, such as Ehlers–Danlos syndrome[85] and amelogenesis imperfecta.[86] A generalized, more

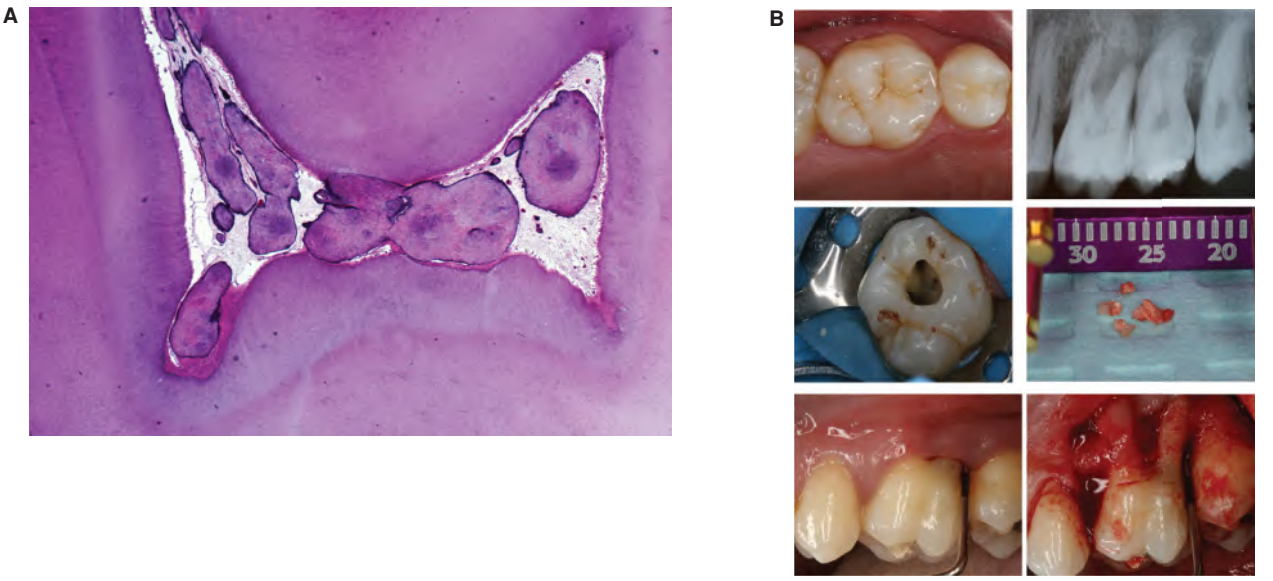

FIGURE 5-17 ***A.*** Multiple pulp stones in the pulp chamber of a molar (hematoxylin and eosin). With permission from Dr. Henrry Trowbridge, San Francisco, CA, UA. ***B***. Pulp stones from a periodontally involved maxillary molar of a healthy 28-year-old patient. Tooth responded normal to cold and electric pulp tests. Root canal treatment was done since the distal root was planned for amputation. Multiple calcifications were found during access opening. No pulp stones could be detected in the adjacent teeth. (With permission from Dr. Hector Rios and Dr. Tatiana Botero, Ann Arbor, MI, U.S.A.)

TABLE 5-1 Terminology

Pulp stone	True	Made of dentine and lined by odontoblasts.
	False	Formed from degenerating cells which mineralize.
	Free	Stone not related to pulp space wall, surrounded by soft tissue.
	Adherent	Stone attached to wall of pulp space, not fully enclosed by dentine.
	Embedded	Stone enclosed within canal wall, less attached than the above.
Denticle		An alternative term for pulp stone, more usually a calcification filled with epithelial remnants surrounded by odontoblasts.
Fibrodentine		Material produced by fibroblast-like cells against dentine prior to differentiation of a new generation of odontoblast-like cells.
Dystrophic calcification		Inappropriate biomineralization of the pulp in the absence of mineral imbalance.

Classification for dental pulp stones. Reproduced with permission from Goga R et al.[83]

diffuse dystrophic mineralization of the pulp may occur after trauma[87,88] and is one of several good reasons for follow-up radiographs. In these patients, a characteristic color change is noticeable as they become more yellow while the pulp chamber and canal space are filled by reactionary and reparative dentin. The majority of these teeth will not require endodontic therapy. Only <10% might become necrotic and develop a periapical lesion.[89,90]

Internal resorption has been considered an alternative sequela to trauma although most of the literature on this condition consists of case reports (Figure 5-18). The difficulty in establishing a correlation with trauma is probably related to the length of time between the traumatic incident and the diagnosis.

The internal root resorption is asymptomatic but progressive. This lesion may eventually perforate the root or crown. This type of resorption can be extensive and may

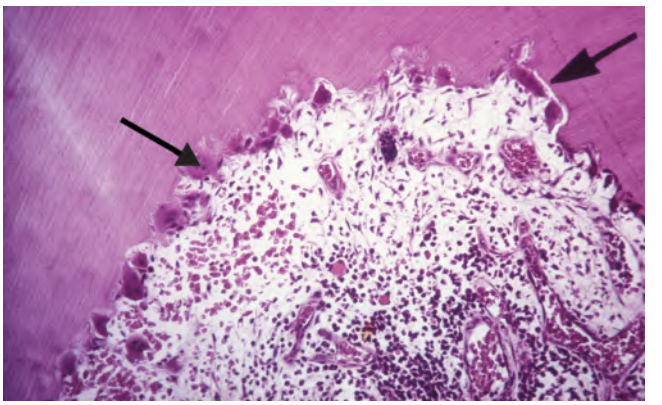

FIGURE 5-18 Internal resorption. Multinuclear odontoclasts can be seen within lacunae in the dentin (arrows) (hematoxylin and eosin). (With permission from Dr. Henrry Trowbridge, San Francisco, CA, U.S.A.)

become a coronal pink discoloration that is often misdiagnosed as caries. Internal resorption has also been found as microscopically small lesions in teeth with pulpitis and necrosis.[91] Damage to the predentin layer leads to a direct contact of the inflamed tissue and the exposed dentin. The odontoclast (differentiated from circulating monocytes) would start resorbing dentin and replacing it with granulation tissue. Although internal irresorption can be transient or progressive. When internal resorption is diagnosed, the pulp should be extirpated to stop this irreversible process.[92,93] For more details, see Chapter 15, "Pathologic Tooth Resorption."

WHEN THE PULPAL RESPONSE SUCCEEDS: REPAIR AND REGENERATION

The immune response, including inflammation, is only one part of the pulp's total response to toxins or injury. When effective, this response neutralizes and removes any foreign material and allows recovery, repair, and regeneration. The repair process can take several forms. The most obvious distinctions relate to the severity of the injury. When no cells are killed, the original odontoblasts can form reactionary (tertiary) dentin (Figure 5-19). When the odontoblasts are killed, new dentin-forming cells develop from stem cells and form reparative (tertiary) dentin (Figure 5-20).[94,95] The repair of larger areas of damage is more variable and depends on the nature of any clinical intervention. It seems that normally organized pulp tissue does not re-form. An example of extensive tissue damage from which the pulp recovers is the pulp polyp (Figure 5-21). Rapidly advancing caries in young molars can result in collapse of the crown with a large but self-cleansing pulp exposure. Epithelial cells from the oral mucosa settle on the exposed pulp and form an epidermis (Figure 5-22). This phenomenon attests to the pulp's ability to respond to injury but is futile as the molars involved are often not restorable.

Sometimes, the progress of caries and the defensive response can reach a stalemate. The irritant continues but is contained. This, by one definition, is chronic inflammation with injury and repair occurring at the same time. Many inflammatory cells, largely lymphocytes, are present as well as areas of fibrous tissue representing an attempt to "wall off" the diseased tissue.

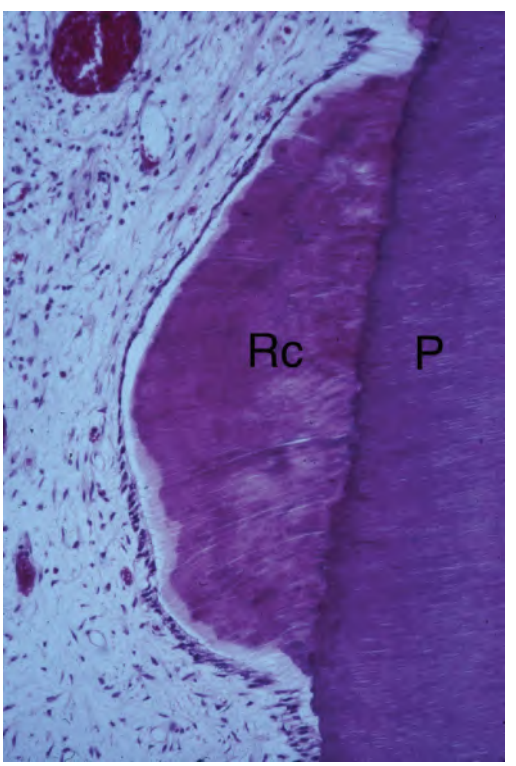

FIGURE 5-19 Reactionary (tertiary) dentin (Rc) developed beneath a deep cavity preparation. The tubules in the reactionary dentin are continuous with those of the primary dentin (P) (hematoxylin and eosin). (With permission from Dr. Henry Trowbridge, San Francisco, CA, U.S.A.)

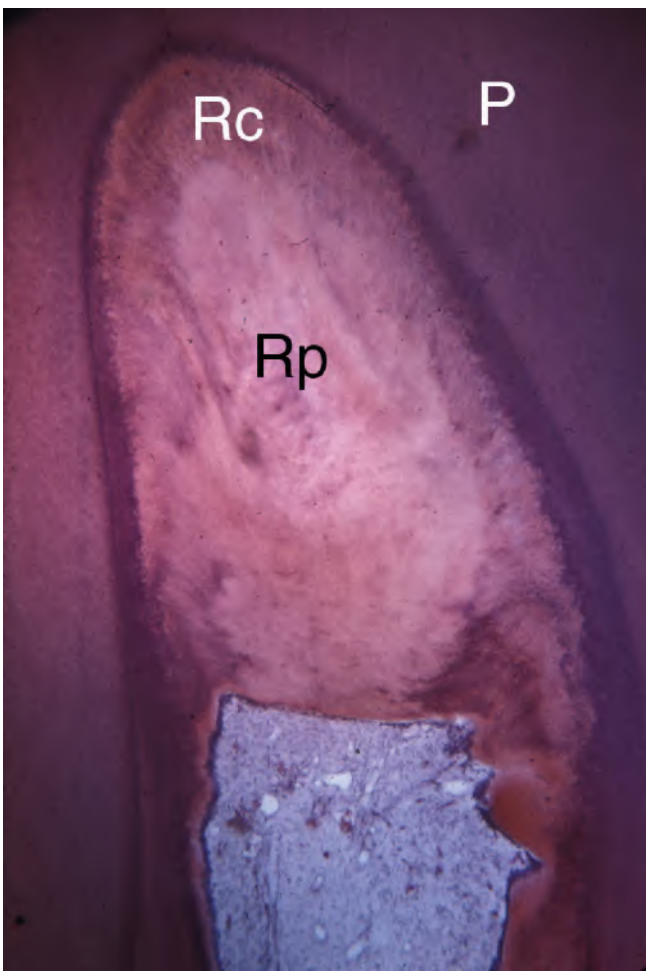

FIGURE 5-20 A layer of reactionary (tertiary) dentin (Rc) formed beneath an injury has then become severe enough to kill the original odontoblasts. It resulted in the formation of atubular reparative (tertiary) dentin (Rp), which has been formed by new odontoblasts (P = primary dentin) (hematoxylin and eosin). (With permission from Dr. Henrry Trowbridge, San Francisco, CA, U.S.A.)

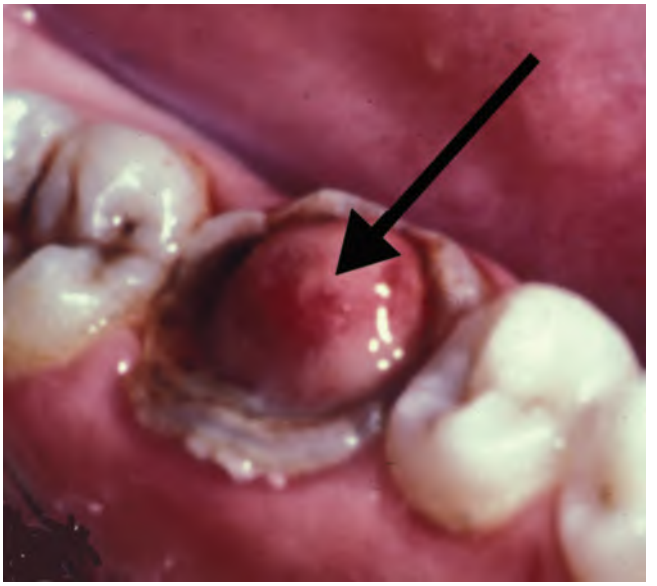

FIGURE 5-21 Pulp polyp, also known as hyperplastic pulpitis (arrows). Caries of the mandibular first permanent molars in a young patient has proceeded so quickly that the walls of the cavity collapsed, leaving a largely self-cleansing area. The pulp survives and its surface colonized by epithelial cells shed from the buccal mucosa. (With permission from Dr. Henry Trowbridge, San Francisco, CA, U.S.A.)

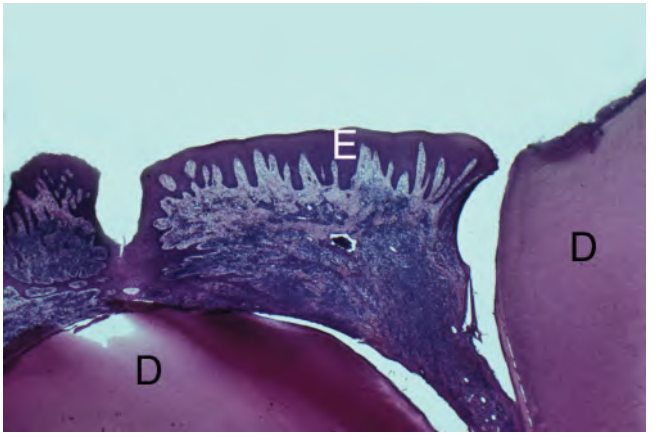

FIGURE 5-22 Structure of a pulp polyp. The epithelial cells shed from the buccal mucosa form an epithelium (E) that is normal in appearance. Dentin (D) is present on each side of the polyp that has a dense infiltrate of inflammatory leukocytes (hematoxylin and eosin). (With permission from Dr. Henry Trowbridge, San Francisco, CA, U.S.A.)

ENCOURAGING A SUCCESSFUL RESPONSE

From a clinical standpoint, it is important to recognize that the pulp has a strong innate ability to respond and repair. Most of our strategies for encouraging pulpal repair involve removal of the irritant and diseased tissue and prevention of further injury. Calcium hydroxide, for example, probably does nothing directly to stimulate reparative dentin formation but is permissive of the pulp's inherent activity. For >50 years, calcium hydroxide has been considered the standard of care for direct and indirect pulp capping. The advent of new materials derived from mineral trioxide aggregate (MTA) classified as bioceramics or tricalcium silicate cements set by a hydration reaction forming calcium silicate hydrate and calcium hydroxide ($CaOH_2$). Bioceramic cements have stimulatory activity, but their main property are due to the high level of biocompatibility and their success in preventing recontamination.[96] With the discovery of biologically active molecules such as growth factors and matrix proteins, there are now more possibilities of stimulating repair.[97,98] The development of cultures of pulpal stem cells from human permanent teeth (DPSC)[99] or deciduous teeth (SHED)[100] and supporting scaffolds may allow tissue regeneration. Neither of these approaches is, at present, ready for clinical adoption.[101]

FACTORS LIMITING THE PULP'S RESPONSE

The only significant factor that limits the pulp's ability to respond to injury is age. The older pulp has a reduced number of cells,[102] innervation,[103] and vascularity,[104,105] but the immune response remains active.[94,95]

CLINICAL PROCEDURES

Local Anesthetics

Local anesthetics reduce pulpal blood flow by approximately half when they contain vasoconstrictors.[106,107] This effect is almost due to the vasoconstrictor. It is important to remember that when preparing a cavity in an anesthetized tooth, the pulp is in a suboptimal condition to respond. Much of what we know about the pulp's response to cavity preparation and materials has been from animal experiments in which general, rather than local, anesthetics were used.

Heat During Cavity and Crown Preparation

Experimental Observations

The basic effect of heat on mineralized tissue has been best documented on bone.[108–111] From this work, a value of 10°C above body temperature is frequently cited as the threshold to cause irreversible damage to vital tissues.[108] Higher temperatures and increased application times lead to greater damage. An increase of surface temperature to 47°C held for only 1 minute, resulted in minor reversible injury. Increasing the duration to 5 minutes caused irreversible damage to calcified bone and marrow. Raising the temperature to 50°C but reducing the duration back to 1 minute produced injury similar to the injury caused by raising the temperature to 47°C at 5 minutes. Temperatures of 60°C and higher, will result in bone necrosis.[108] Severe cold will also damage tissue. The formation of water crystals

within cytoplasm will kill a cell. This occurs at a temperature of ~2°C below zero,[112] a temperature never likely to be experienced in the mouth.

Moderate elevation of temperature on the external surface of a tooth can raise the internal temperature of the pulp to the point that injury occurs. A temperature increase of 5.5°C above body temperature, as observed by Zach and Cohen,[113] is frequently cited as the threshold for damage to the pulp. This observation was made after applying a soldering iron preheated to 275°C to the teeth of monkeys for a limited time. When the same temperature was applied for a longer period of time, generating an 11.1°C increase in pulp temperature, 60% of pulps became necrotic. However, this very abrupt application of high heat may not translate directly to clinical situations.

Baldissara et al.[114] applied a gradually increasing heat stimulus until the patient indicated sensitivity then sustained it for 30 seconds. The temperature change across the dentin was measured after extraction. The maximum temperature increase was 14.7°C reaching a measured temperature of 50.9°C. Histologic examination of the heated pulp revealed neither necrosis nor alteration of the odontoblast layer. All the patients remained symptom free throughout the duration of the study, and all teeth responded to cold testing just before extraction.[114] These *in vivo* experimental observations were made on un-restored, noncarious human teeth, scheduled for extraction for orthodontic treatment. As these patients are of young age, their pulps will have a good blood supply and will not have been affected by previous injury. This model may not be fully revealing the effects of heat on the mature pulp.

The more moderate pulp responses observed by Baldissara et al.[114] are derived from earlier studies. Pulps from human subjects that were examined immediately and 1 month after a 30-second application of 150°C to 0.5-mm-thick dentin were found to have localized areas of injury although no pulps were completely necrotic.[115] The most common histologic observation was a breakdown of the odontoblast layer. Some of the pulps had localized areas of necrosis. The affected areas were not surrounded by inflammatory cells. None of these injuries was symptomatic. Other studies have reported similar results.[116,117] It appears that the dental pulp has some capacity to adapt to a moderate, gradually increase in temperature.

Cutting Dentin

The amount of heat produced during cutting of dentin is determined by the sharpness of the bur, the amount of pressure exerted on the bur, and the length of time the cutting instrument contacts tooth structure. The temperature on the surface of a rotating dental bur in contact with dentin has been reported to be as high as 417°C.[116] High-speed air-driven handpieces require an effective method of cooling the tooth in order to safely use these handpieces. The two cooling methods most frequently applied are air cooling and water cooling. Pulp temperatures with air cooling alone rise to 60°C, whereas with water cooling, it falls to 26°C (11°C below body temperature).[118]

Damage has been observed in the pulps of teeth cut with air cooling only.[119] Odontoblasts were dislocated into dentinal tubules and there were vacuoles throughout the odontoblast layer. By contrast, with water-cooling, displacement of odontoblasts occurred in less than half of the teeth and was less marked when it did occur. Fifteen days after cavity preparation, inflammation was noted in all the pulps under cavities prepared with air-cooling only. On the other hand, inflammation occurred in only 25% of water-cooled group. In both groups, recovery occurred within 200 days with the formation of tertiary reparative dentin. Dentin is a good insulator and thus careful cutting is not likely to damage the pulp unless the thickness of dentin between preparation and pulp is <1.0 mm.[119] Even then, the response should be mild.

The "blushing" of dentin during crown preparation is thought to be due to frictional heat resulting in vascular injury (hemorrhage) in the pulp.[115] The dentin takes on an underlying pinkish hue soon after the operative procedure. The most intense level of frictional heat is generated with a large diamond bur when teeth are prepared for a full crown. Crown preparation performed without the use of a coolant leads to a marked reduction in pulpal blood flow, presumably because of vascular stasis and thrombosis.[120]

The safest way to prepare tooth structure is to use ultra-high speeds of rotation (100,000–250,000 rpm), with an efficient water-cooling system, light pressure, and intermittent cutting. During cutting at high speeds, the revolving bur creates an area of turbulence that tends to deflect a stream of water. Therefore, an air-water spray with sufficient volume and pressure must be used if the coolant is to overcome the rotary turbulence. The bur–dentin interface should be constantly wet. Cavity preparation with a low-speed handpiece, sharp bur, and light, intermittent pressure is only slightly more injurious than cutting at high speeds. Hand instruments and low-speed cutting are relatively safe ways to finish a cavity preparation, rather than using a high-speed handpiece with the water coolant shut off.[116,117]

Lasers

The use of laser beams to fuse enamel and reduce the likelihood of carious invasion has been suggested.[117] Different lasers with different energy levels may also be used to remove caries. Certain types of lasers can generate a marked increase in temperature within dentin and pulp tissue.[121] Proper power setting, time of application, and use of water spray will moderate the temperature increase to levels below the heat threshold of pulp damage.[122,123]

Pins

Pulp damage may result from pinhole preparation or pin placement. Coolants do not reach the depth of the pin preparation. During pinhole preparation, there is always the risk

of pulp exposure. Furthermore, friction-locked pins often produce micro-fractures that may extend to the pulp, subjecting the pulp to irritation and the effects of microleakage. The use of pins should be discouraged and, with the introduction of newer adhesive materials, their use is no longer necessary.[124,125]

Cavity Cleansing

A prolonged blast of compressed air, aimed onto freshly exposed vital dentin, will cause a rapid outward movement of fluid in patent dentinal tubules.[126] Tubule diameter midway between the pulp and the dentin-enamel junction (DEJ) is only 1.5 μm. Therefore, the removal of fluid from the tubules by a blast of air activates strong capillary forces. These, in turn, lead to a rapid outward flow of dentinal fluid.[127] Fluid removed from the tubules at the dentin surface is replaced by fluid from the pulp.

The rapid outward flow of fluid in the dentinal tubules stimulates nociceptors in the dental pulp producing pain. Rapid outward fluid movement may also result in odontoblast displacement.[127] Odontoblasts are dislodged from the odontoblast layer and drawn outward into the tubules. Within a short time, the displaced cells undergo autolysis and disappear. When the pulp has not been severely injured either by caries or by other factors, displaced odontoblasts are replaced by new cells derived from stem cells deeper in the pulp. In this way, the odontoblast layer is reconstituted by "replacement" odontoblasts capable of producing tertiary dentin. Even vigorous drying of dentin alone does not result in severe injury to the underlying pulp.

Drying agents containing lipid solvents, such as acetone and ether, have been used to clean cavity floors. Because of their rapid rate of evaporation, application of these agents to exposed dentin produces strong hydrodynamic forces in the tubules causing odontoblast displacement. Cavities should be dried with cotton pellets and short "soft" blasts of air rather than harsh chemicals. Disinfectants have, in the past, been used in cavity cleansing. There seems to be no particular benefit to this, and they are now rarely used as they are potentially toxic to the pulp.

Etching Dentin/Smear Layer

Cutting dentin results in a smear layer on the cut surface consisting of fragments of microscopic mineral crystals and organic matrix.[128–134] This layer may interfere with the adhesion of restorative materials although some newer bonding agents bond well to the smear layer. Acidic cavity-cleansing products and chelating agents have been used to remove the smear layer, but the use of these depends on the nature of the restorative material. The smear layer does have one desirable property. By blocking the orifices of dentinal tubules, the smear plugs greatly decrease the permeability of dentin. While the smear layer is semipermeable, it is not a barrier to microorganisms or their products.

Complete dissolution of the smear layer opens the dentinal tubules thereby increasing the permeability of dentin. If the dentin is left unsealed, the diffusion of irritants to the pulp may intensify and prolong the severity of pulpal reactions. Available experimental evidence is contradictory. Some evidence indicates that etching, as a step in restoration, may reduce microleakage.[135] Other evidences suggest that etching causes pulpal damage, when the thickness of the remaining dentin is <300 μm (RDT).[136]

In the absence of microleakage, acid etching of dentin does not appear to produce injury to the pulp because calcium and phosphate ions are released, producing a buffering action. Even when placed in deep cavities, acid etchants produce only a small increase in hydrogen ion concentration in the pulp.[137]

Impressions, Temporary Crowns, and Crown Cementation

Temperatures of up to 52°C have been recorded in the pulp during impression taking with modeling compound. Modeling compound may be damaging because of the combination of heat and pressure.[138–141] Fortunately, this technique is no longer used. Rubber base and hydrocolloid materials do not injure the pulp. The heat generated during the exothermic polymerization of autopolymerizing resins may injure the pulp. Cooling is strongly recommended when provisional crowns are fabricated directly. Before cementing provisional crowns, the crown preparation should be carefully lined with temporary cement to minimize microleakage. The cemented temporary crown should be in place for a short period of time; temporary cements are not stable and will eventually wash out. Microleakage around temporary crowns is a common cause of postoperative sensitivity. During the cementation of crowns, inlays, and bridges, strong hydraulic forces may be exerted on the pulp as cement compresses the fluid in the dentinal tubules.[142] In deep preparations, this can result in a separation of the odontoblast layer from the predentin. Vents in the casting will allow cement to escape and facilitate seating. The pulps of heavily restored teeth that have overcome all these procedures are at risk of developing necrosis.[143]

DENTAL MATERIALS

Microleakage

The most important characteristic of any restorative material, related to its effect on the pulp, is its ability to form a seal that prevents the leakage of microorganisms and their byproducts onto dentin and then into the pulp.[2,136,144]

Cytotoxicity

Certain restorative materials are composed of chemicals having the potential to irritate the pulp. However, when placed in a cavity, the intervening dentin usually neutralizes or prevents leachable ingredients from reaching the pulp in a high enough concentration to cause injury. For example, eugenol in zinc oxide-eugenol (ZnOE) is potentially irritating but

very little can reach the pulp. Phosphoric acid is a component of silicate and zinc orthophosphate (ZnOP) cements and was thought to be highly injurious to the pulp. However, the buffering capacity of dentin greatly limits its penetration. It is now clear that the problems reported following use of these materials were due to their high degree of shrinkage and subsequent microleakage.[4]

Clearly, the thickness and permeability of dentin between a material and the pulp affect the response to the material. In addition, the penetration of some materials through dentin may be limited by the outward flow of fluid through the tubules that will be increased if the pulp is inflamed.[145] This is a factor that has been overlooked in many *in vitro* studies focusing on the passage of materials through dentin.

Many studies on cytotoxicity focus on isolated cell types in culture and do not take into account the immunocompetent cells present in the intact pulp. Materials may have a differential effect on these cells by either stimulating or inhibiting their activity.[146] Cytotoxicity tests can be used as an initial screening but they are not conclusive in determining their biocompatibily.[147]

Materials are more toxic when they are placed directly on an exposed pulp. Cytotoxicity tests carried out on materials *in vitro*, or in soft tissues, may not predict the effect these materials have on the dental pulp. The toxicity of the individual components of a material may vary.[148,149] A set material may differ in toxicity from an unset material. The immediate pulpal response to a material is much less significant than the long-term response. Upon examination a few days after placement, there may be a strong inflammatory response. After a few months, the inflammatory response may have receded and repair has taken over. The best measure of long-term response is the thickness of tertiary dentin laid down by the affected pulp.

Heat Upon Setting

Many restorative materials have exothermic setting reactions. Temperature increase during setting may be over 10°C[150] but can be limited to 2°C to 3°C.[141,151] Cooling includes the use of both well directed air and water spray.

Some luting cements generate heat during setting; it has been suggested that this might cause pulpal injury. The most exothermic luting material is ZnOP cement. However, during setting, an intrapulpal temperature increase of only 2°C was recorded.[152] Heat of this magnitude is not sufficient to injure the pulp.

Desiccation by Hygroscopy

Some hygroscopic materials may potentially cause injury by withdrawing fluid from dentin. However, little relationship exists between the hydrophilic properties of materials and their effect on the pulp. Moisture absorbed by materials is probably much less than that removed from dentin during cavity drying.[151]

SPECIFIC MATERIALS

Zinc Oxide–Eugenol

ZnOE has many uses in dentistry, with a long history as a temporary filling material, cavity liner, cement base, and luting agent for provisional cementation of castings. Before the introduction of calcium hydroxide, ZnOE was the material of choice for direct pulp capping.[136,153–155]

Eugenol, the most active ingredient in ZnOE, is a phenol derivative and is toxic when placed in direct contact with tissue.[156] It also possesses antibacterial properties.[157] The use of Eugenol in pain control is attributed to its ability to block the transmission of nerve impulses.[158] A thin mixture of ZnOE significantly reduces intradental nerve activity when placed in a deep cavity preparation in cat's teeth; a dry mix of ZnOE has no effect.[159]

When included in cements to temporize crown preparation, some eugenol does reach the pulp, but the amounts are small and unrelated to RDT. In *in vitro* studies, "desensitizing" agents do not seem to reduce the penetration of eugenol, although time does.[160] The movement of eugenol is by a hydrolytic mechanism that is dependent on the presence of fluid. With little fluid available, release is low.[153,154]

The most important property of ZnOE is that it provides a tight marginal seal preventing microleakage. Its superiority as a temporary restorative material is enhanced by its antimicrobial properties.[161]

Zinc Phosphate Cement

ZnOP was a popular luting and base agent. It has a high modulus of elasticity and therefore, was commonly used beneath amalgam restorations before glass ionomer cements were available. The phosphoric acid liquid phase was formerly thought to injure the pulp. Recent studies have shown that this is not the case. Cementation of castings with ZnOP is well tolerated. ZnOP is more likely than glass ionomer to produce pulpal sensitivity at the time of cementation and up to 2 weeks after cementation. However, 3 months after cementation, there is no difference in sensitivity.[162–165]

Polycarboxylate Cement

When placed in cavities or used as a luting cement, zinc polycarboxylate does not irritate the pulp. In cementing well-fitting crowns and inlays, neither polycarboxylate nor ZnOP cements contract enough to permit the ingress of bacteria. Consequently, it is unnecessary to apply a varnish or liner to cavity walls; doing so reduces cement adhesion.[165–167]

Glass Ionomer Cements

The original use of the glass ionomer cement was as an esthetic restorative material, but now it has also found use as liners, luting agents, and pulp-capping materials (sometimes in conjunction with calcium hydroxide). When placed on exposed pulps in non-carious teeth, glass ionomer cement shows a degree of bacterial microleakage similar to that of

composite resins but less than half of the calcium hydroxide cement.[168,169] The incidence of severe pulpal inflammation or necrosis caused by glass ionomer cement on exposed healthy pulps is similar to that for calcium hydroxide but greater than that for composite resins.[169] When placed in cavities in which the pulp is not exposed, and where there is a narrow RDT (0.5–0.25 μm), both calcium hydroxide and composite resin show faster deposition of tertiary dentin than glass ionomer cements.[170]

When used as a luting agent, the pulpal response to glass ionomer cements is similar to that of polycarboxylate and ZnOP cements.[163] For some time after their introduction as luting agents, glass ionomer cements were associated with post-cementation sensitivity. With the use of appropriate technique, the incidence of sensitivity using these agents is not greater than that with other commonly used luting agents.[164]

Calcium Hydroxide

Since its introduction in dentistry by Herman in 1927, calcium hydroxide has been the most widely used material for direct and indirect pulp capping procedures. Calcium hydroxide ($CaOH_2$) has high pH (12.5) and dissociates into Ca^{2+} and OH^{2-} that not only gives antibacterial properties[171,172] to the formulation but also provides a stimulus to induce tertiary reactionary or reparative dentin secretion.[173] When in contact with the pulp, a zone of necrosis is formed and a mild inflammatory reaction occurs. Cells from the core zones migrate, proliferate, and differentiate as odontoblast-like cells to form reparative dentin and gradually forms a dentinal bridge.[174,175] Coronal seal is a priority for success as calcium hydroxide lacks sealing properties.

Mineral Trioxide Agregate and Calcium Silicate Cements

Ideally, materials use to fill root-end preparations should be hydrophilic, which can set in the presence of water. MTA was the first hydrophilic cement developed 20 years ago from a Portland cement formulation. MTA contains tricalcium silicate, dicalcium silicates, and bismuth oxide as radiopacifier.[176,177] The hydration reaction with water or water-containing liquids generates a calcium silicate gel that gets harder after 4 hours or longer. Calcium hydroxide is also released for as long as the reaction is active. After setting, MTA creates a good seal to dentin and materials such as glass ionomer cements, copolymers, and composites. The composition, constant high pH, and the property to release calcium provides MTA several advantages over other materials as a biocompatible pulp capping material.[178,179]

The exposure of MTA to lights can lead to discoloration in the dentin due to the presence of several metals in its original formulation (gray-colored MTA). The long setting time, the sandy consistency, and the possible discoloration of MTA-derived cements have forced the development of newer materials. Some new materials are now available including Endo-Sequence (Brasseler, Savannah, GA, USA), Biodentine, (Septodont, Lancaster, PA, USA), white-colored MTA plus (Denstply, Tulsa, OK, USA), and Theracal (Bisco, Schaumburg, IL, USA) among others.[96,180,181] Most of these new formulations have improved the original MTA cement although more research is needed.

Amalgam

Amalgam alloy has been widely used for restoring posterior teeth. There is shrinkage during setting, which results in microleakage.[182] This decreases as corrosion products accumulate between the restoration and the cavity walls and can be further reduced by the use of liners.[183] Amalgam is the only restorative material in which the marginal seal improves with time. Esthetics and public concern with the mercury content of amalgams have lead to an accelerated use of composite resins as posterior restorative materials instead of amalgam. Their use, however, is more technique sensitive. In deep cavities in posterior teeth, composites have been associated with more pulpal injury than amalgams because of microleakage.[184]

Restorative Resins

The first adhesive-bonded resin composite systems that were developed contracted during polymerization resulting in gross microleakage and bacterial contamination of the cavity. Bacteria on cavity walls and within axial dentin are associated with moderate pulpal inflammation. Over a period of time, some composites absorb water and expand; this tends to compensate for initial contraction. To limit microleakage and improve retention, enamel margins are beveled and acid etched. When compared with unfilled resins, the newer resin composites present a coefficient of thermal expansion similar to that of dentin. With the development of hydrophilic adhesive bonding systems, the problem of marginal leakage appears to have diminished.[184–186]

Although restorative resins themselves do not release substantial heat upon setting, the heat of photo-curing lights can be transmitted through the resin and affect dentin. Time of exposure, thickness of dentin, and type of photo-curing light are factors to consider. Temperatures at the surface of the tips of various light curing units can increase from 49.6°C to 100°C above the temperature before activation.[187] In spite of these very high temperatures, *in vitro* and *in vivo* investigations have consistently demonstrated pulp temperatures below the level that will cause pulp damage.[188–191]

Pulp Capping Materials

Indirect or direct pulp capping is a very common procedure in operative dentistry and requires knowing the status of the pulp, the history of previous injuries, the status of root formation, the periapical condition, and future restoration plans. Calcium hydroxide was the first material introduced as a capping agent. It was considered as the material of choice for many years due to its impressive antimicrobial action as

well as its potential to induce dentinal bridge formation.[171] Many other capping materials have been explored such as hydroxyapatite, dentin chips, growth factors (BMP-1, -7, TFG-B-1,-3, FGF, IGF[192–202] glass ionomers, and composite resins.[197–200] The release of bioactive molecules is a key factor for the reparative dentinogenesis induced by any of these capping agents.[200] As previously described new tricalcium silicate-based materials such as Biodentine and Theracal have shown successful results for patients and clinicians.[201,202] For more details, see Chapter 27, "Vital Pulp Therapy."

POLISHING RESTORATIONS

Polishing glass ionomer and composite restorations does not cause an increased temperature at the pulp-dentin interface.[198] Polishing amalgam restorations, however, can produce temperatures that may be damaging. With high contact pressure, high speed, and no coolant, *in vitro* temperature increases of >20°C have been recorded within 30 seconds.[122] However, a handpiece rotating at 2,500 rpm that was held for 60 seconds with a relatively light (55 gm) force generated a maximum temperature increase of only 6.2°C.[136] This occurred when a Burlew disk without coolant was used as the polishing agent. With air cooling, the increase was 2.0°C. When polishing an amalgam restoration with continuous pressure and no water coolant, it is recommended to not exceed 4,000 rpm.[203] With use of coolant, light pressure, and intermittent contact during polishing, there is a low likelihood of heat-generated pulp damage.

POST-RESTORATIVE HYPERSENSITIVITY

Many patients complain of hypersensitivity after a restorative procedure.[150,164] This may be due to any one or an accumulation of the factors listed above. Discomfort is usually of short duration. If pain is prolonged, a pre-existing pulpitis may have been exacerbated. If delayed in onset by days, the cause may be microleakage of microbial toxins under a poorly sealed temporary restoration. The absence of postoperative sensitivity after restoration with modern composites of both class I and II preparations has been demonstrated in clinical studies, suggesting that variations in technique may be responsible for the anecdotal reports of sensitivity.[199,205] The use of hydroxymethacrylate and glutaraldehyde "desensitizer" does not reduce the incidence of sensitivity.[204] Self-etching, self-priming dentin-bonding systems reduce the incidence of sensitivity following the restoration of deep carious cavities.[130] If pain is evoked by biting on a recently restored tooth, an intracoronal restoration may be exerting a strong shearing force on the dentin walls of the preparation. It is more likely to be due to injury to the periodontal ligament resulting from hyperocclusion. Hyperocclusion from an extracoronal restoration is not injurious to the pulp but may cause a transient hypersensitivity.

VITAL TOOTH BLEACHING

Overnight external bleaching of anterior teeth with 10% carbamide peroxide causes mild pulpitis that is reversed within 2 weeks.[206] In vitro studies show that the principle bleaching agent, hydrogen peroxide, can reach the pulp after application to the enamel.[207] Whether this occurs *in vivo* is unknown. Outward fluid flow in dentinal tubules and other factors would reduce the effect. Both short-term and long-term (9–12 years) clinical observations on bleached teeth reported no significant pulpal changes.[208] Heat-activated bleaching agents can cause intrapulpal temperatures to rise by 5–8°C when measured *in vitro*.[209]

THERMAL SENSIBILITY TESTING

Thermal sensibility testing utilizes media at temperatures that certainly have the potential to damage tissues. Heated gutta-percha reaches a temperature of ~200°C just before the smoke point, and a cold-test substance such as carbon dioxide snow has an inherent temperature of –78°C. A heat-testing device such as the System B (SybronEndo, Orange, CA, USA) with the appropriate tip is recommended to be set at a temperature of 200°C. The question must be asked whether these extreme temperatures are transmitted to the dental pulp during clinical testing and whether there is any likelihood that the tissue may be damaged.

Rickoff et al.[210] reported combined *in vivo* and *in vitro* findings with no damage to pulp tissue following use of flame-heated gutta-percha and carbon dioxide snow. Gutta-percha was heated to the smoke point and then applied to a tooth surface *in vivo*. The gutta-percha remained in contact for 1–10 seconds. For cold testing, a carbon dioxide pencil of 3.5 mm diameter was held to the tooth for 5 seconds to 5 minutes. The teeth were extracted and examined histologically. To measure the temperatures generated at the pulpal–dentin junction (PDJ), a thermistor was placed onto the dentin surface of an extracted tooth and the temperature protocol repeated. Internal temperature was recorded as the external surface was heated to different temperatures. The maximum PDJ temperature reached with heat testing was 39.9°C (10-second application) and for cold was 9.5°C (5-minute application). These temperatures do not reach the thresholds at which tissue damage will occur. The time of heating falls within the range of likely clinical application.[210]

Augsburger et al.[211] observed similar temperature decreases. The greatest temperature drop in their *in vitro* evaluation was 6.3°C. This fall in temperature occurred in a tooth with a full gold crown that received a 5-second application of carbon dioxide snow. The greatest temperature drop in noncarious teeth was 2.5°C (also with carbon dioxide). They also tested water and skin refrigerant that had maximum temperature drops at the PDJ of 2.8°C and 1.8°C, respectively. The greatest temperature transference between adjacent teeth with metallic restorations was a decrease of 1.7°C between a tooth with a gold crown and one with an amalgam. Rickoff et al.[171] observed a similar, very small

temperature drop of 0.9°C, after applying carbon dioxide to four premolar teeth *in vitro*. *In vivo* histologic analysis revealed no alteration from normal tissue appearance after a 2-minute[212] or a 5-minute[213] application of carbon dioxide. When carbon dioxide was held to a tooth for 20 minutes, the pulp showed necrosis of odontoblasts.[213] It is, therefore, safe to conclude that heat and cold testing within normal clinical parameters will not damage the dental pulp.

TOOTH MOVEMENT

Orthodontic tooth movement of a routine nature does not cause clinically significant changes in the dental pulp. For more details, see Chapter 39, "Interrelationships of Endodontics and Orthodontics in Treatment Planning and Patient Treatment."

Responses to pulp testing, especially electrical, may be unreliable.[214] The heavy forces used to reposition impacted canines frequently lead to pulp necrosis or calcific metamorphosis.[215] Intrusion but not extrusion reduces pulpal blood flow for a few minutes as the pressure is applied.[211] Capillaries proliferate in the pulps of moving teeth.[216] A variety of growth factors are produced including vascular endothelial growth factor (VEGF) that may explain this increase in vascularity.[217–219]

REMOVAL OF ORTHODONTIC BRACKETS

Some of the methods of removing brackets at completion of orthodontic treatment have the potential to cause injury to the pulp.[210–224] Brackets may be simply ground off, or pincher-type pliers may be used to mechanically remove the bracket. A third method of bracket removal is use of an electrothermal device (ETD) that heat-softens the bonding composite expediting removal of the bracket. Heat transferred from the ETD has been measured as high as 45.6°C[225] on the pulpal side of dentin in *in vitro* experiments, though most reports have been in a lower range.[220,226–228] When residual composite is removed by grinding, pulp-side temperature actually decreased below ambient temperature when water coolant was used.[220] Metal brackets have a greater capacity to transmit heat to inner dentinal surfaces than ceramic brackets.[217]

In vivo evaluation has recorded much lower temperatures and has reported limited pulpal damage. Jost-Brinkman et al.[229] observed no pulpal inflammation 4 weeks after electrothermally debonding brackets from human teeth. The maximum temperature recorded was 6.9°C over a period of 43 seconds. There has been consensus that electrothermal debonding does not cause gross damage or necrosis of dental pulp. However, there may be limited peripheral disruption of odontoblasts with slight inflammation.[220,223,224]

Periodontal Disease

There is continuing controversy about the effect of periodontal disease on the dental pulp. The majority of evidence shows that, as long as the periodontal involvement does not reach the apical foramen of the tooth, there is minimal pulpal involvement. A histological study on 60 caries-free teeth with different degree of periodontitis demonstrated that the cumulative effect of periodontal disease on the pulp were mainly calcification, reactionary dentin formation and mild pulpal inflammation. These pulpal changes were found mostly when microorganisms were present in the dentinal tubules or lateral canals. Necrosis or total disintegration of the pulp was found only when the main apical foramen was involved.[229,230] The flushing action of the dentinal fluid is the primary protective mechanism of the healthy vital pulp to counter microorganisms and their toxins.[231] For more details, see Chapter 36.

Ultrasonic Scaling

Activation of ultrasonic instruments generates large amounts of heat. Although dentin is not a particularly good thermal conductor, this heat can still transfer through dentin to affect pulp tissue. Ultrasonic scaling of roots requires prolonged contact of an ultrasonic device and the potential for pulp damage exists. As with other heat-generating dental procedures, proper cooling with water prevents gross pulpal damage. In live dogs, ultrasonic scaling did not create heat-induced pulpal damage when water coolant was used. Interestingly, histologic examination did reveal acute pulpitis, but it was attributed to the vibratory effects of the ultrasonic device rather than the temperature.

An interesting phenomenon of ultrasonic dental scalers is that the load of force with which the ultrasonic tip is held against the tooth has a much greater effect (×4) on the heat generated in the dentin substrate than the power setting.[232,233] One investigation found greater heat generation with diamond sonic tips versus smooth surface tips and greater heat production with heavy versus light force (1N and 0.3N).[234] A diamond tip with heavy pressure resulted with in vitro measurement of 36°C increase. When water coolant was used, all measurements were below 4.2°C increase. Proper water-cooling of both ultrasonic and sonic scalers will prevent excessive heat production in the pulp.

Systemic Disease

Although rare systemic disease may affect the pulp, more likely are that pulpal changes are coincidental with the presence of pulpal disease. One relationship that has been postulated is that between cardiovascular disease and pulpal mineralization. An extensive study of 1,432 teeth reported significantly, higher numbers of pulp stones in patients with cardiovascular disorders (15.86%) than other groups (9.35%).[235] Other studies, however, provide very different data. One[236] found that 74% of patients with reported cardiovascular disease had detectable pulp stones, while only 39% of patients without a history of cardiovascular disease had pulp stones.

In patients with diabetes or autoimmune disorders there was no difference in the incidence of pulp stones between

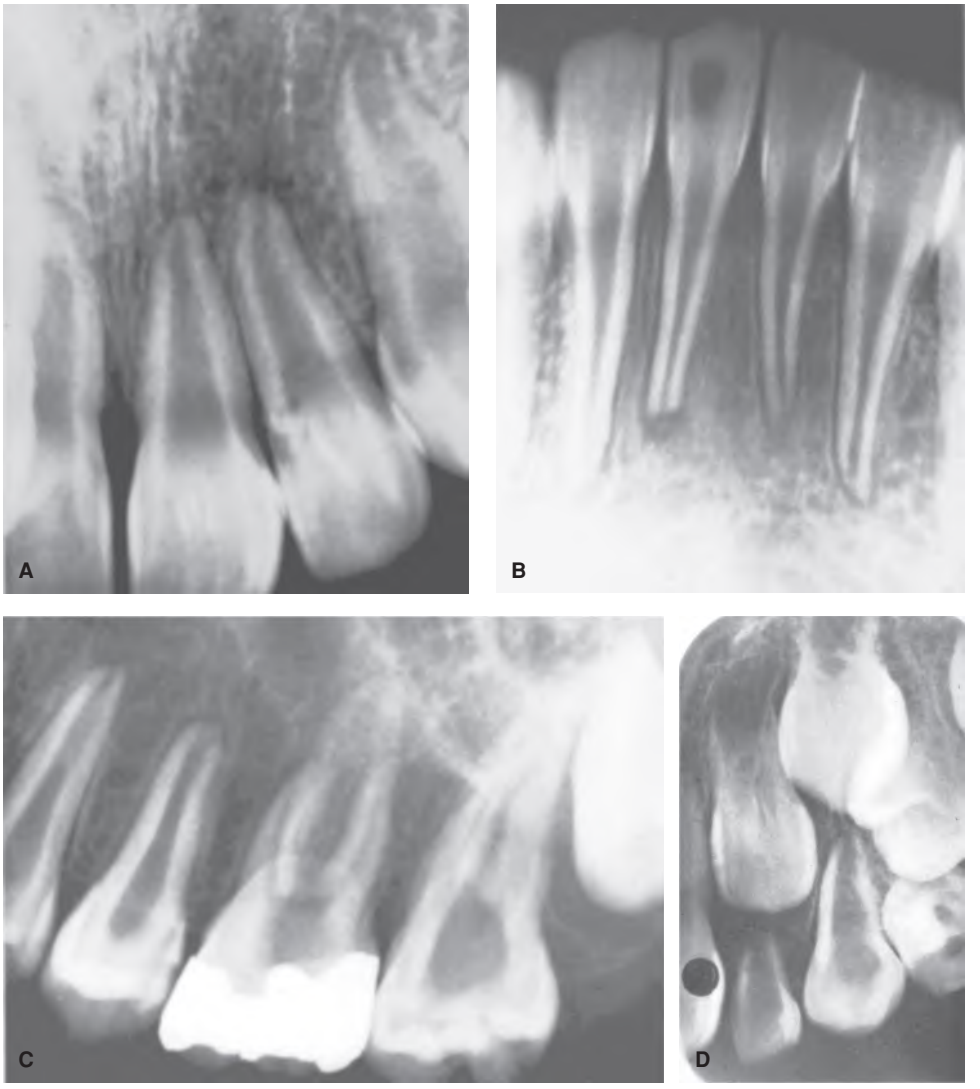

FIGURE 5-23 Unusual pulp dystrophy in *hereditary hypophosphatemia*. Incomplete calcification of dentin and large pulps leave these teeth vulnerable to infection and necrosis. ***A.*** Maxillary incisors at the age of 13. Note large pulp spaces. ***B.*** Mandibular incisors have been traumatized and pulp necrosis has developed. ***C.*** Large pulps can be seen in molar and premolar teeth. The pulp in the first molar later became necrotic. ***D.*** Large pulp size and shape are also apparent in the deciduous dentition. On the basis of these childhood radiographs, diagnosis can be made early about the patient's condition. Timely vitamin D therapy may prevent dwarfing.

patients suffering from diabetes or autoimmune disease and controls.[235] Pulp stones have also been noted in patients with genetic disorders such as dentin dysplasia and dentinogenesis imperfecta.[237]

An unusual and rare cause of pulpal dystrophy and necrosis is found in individuals afflicted with hereditary hypophosphatemia. This disease, which results in dwarfism and "tackle deformity" of the legs, was formerly called refractory rickets, renal rickets, or vitamin D-resistant rickets. Its involvement in dentistry was first reported by Everett and Ingle.[238] It is characterized dentally by the abnormally large pulps and incomplete calcification of the dentin (Figure 5-23). The pulps in these teeth appear to be fragile and succumb to minor irritating stimuli. In one report, the patient had 11 pulpless teeth requiring endodontic treatment.[239,240]

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Chapter 6

Periradicular Disease

Domenico Ricucci, Isabela N. Rôças, José F. Siqueira, Jr.

Endodontics is the specialty ultimately involved with the prevention and treatment of periradicular diseases. These diseases are pathologic processes essentially inflammatory in nature. They develop initially in the periodontal ligament (PDL) to affect bone and cementum, and are caused by microbial infection of the root canal space. For more details, see Chapter 3, "Microbial and Nonmicrobial Etiologies of Endodontic Diseases."

Because periradicular lesions usually occur around the root apex, they are also referred to as periapical lesions or apical periodontitis; the latter denomination was recommended by many authors and international endodontic associations. Apical periodontitis is the term preferentially used throughout this chapter. The reader should be aware that periradicular lesions of endodontic origin may sometimes develop laterally to the root, in association with large lateral canals, or in the furcal region, in association with furcal accessory canals. Periradicular diseases are related to the host innate and adaptive immune responses to intra-radicular bacteria in an attempt to contain the spread of the infection to the bone and other body sites.[1–3]

ETIOPATHOGENESIS

Periradicular Tissues Respond Promptly to Bacterial Invasion of the Pulp

The dental pulp is protected from oral bacteria by its natural encasement in mineralized tissues, including dentin, enamel, and cementum. Immediately after tooth eruption, homeostasis of the pulp is constantly at risk during one's lifetime by the loss of the hard tissues. Dental caries is by far the most common cause of pulp breakdown, followed by periodontal disease. However, other causes may threaten the pulp tissue, such as attrition, trauma, cracks, fractures, and restorative procedures. It is important to remember that microorganisms can easily colonize the tubules of exposed dentin[4] and sometimes reach the pulp via tubules, even before a direct pulp exposure is established. This is true for teeth with deep caries lesions[5,6] as well as for teeth with dentin cracks or attrition.[7,8]

The pulp responds to microbial infection of the dentin tubules by mounting an inflammatory response. This involves both innate and adaptive immune responses, triggering the tertiary dentinogenesis process and reducing the permeability of dentin to microbial cells and their metabolic products. Inflammatory changes, including vasodilation (hyperemia), increase vascular permeability, and migration of inflammatory cells can remain reversible for a long period of time.[9] Reversibility of pulpal inflammation can often be achieved after intervention, consisting of complete removal of the diseased tissues followed by placement of an adequate restoration.

Once microorganisms have invaded the pulp, via tubules from a thin residual layer of dentin or pulp exposure, a focus of necrosis usually develops. Initially, this is limited to a small portion of the pulp adjacent to the forefront of the bacterial infection. This area is typically surrounded by severe acute inflammation, then an area of chronic inflammation and finally, at some distance, a histologically normal tissue.[10] From a histologic point of view, the occurrence of a focus of necrosis, even of limited extension, constitutes the point of transition from a reversible to an irreversible inflammatory state.[6,9,10] This process may happen interchangeably with or without clinical symptoms.

Progression of Pulp Degeneration Process

The initial area of necrosis slowly expands to involve increasing areas of the coronal pulp; however, the layers necrosis—acute inflammation—chronic inflammation—uninflamed tissue are always observed. Tertiary dentin and dystrophic calcifications are commonly seen. Radiographic changes in the periapical area are not normally visible in the radiograph during initial stages of pulp tissue degeneration. However, histologic observation allows recognition of early reactions in the apical PDL in some cases with only partial necrosis in the pulp chamber.

The case shown in Figure 6-1 illustrates the condition in which early periapical disturbances are observed with only partial necrosis in the pulp chamber. It is the maxillary third molar of a 70-year-old woman seeking treatment for severe spontaneous pain originating from the upper left jaw and radiating to the ear. A radiograph (Figure 6-1A) revealed an extruded maxillary third molar, due to missing mandibular third molar. It had deep mesial carious lesion originating at the cemento-enamel junction (CEJ), very close to the pulp chamber. Radiographically, thickening of the apical periodontal ligament (PDL) space was present. Diagnosis of irreversible pulpitis was made. The tooth was extracted upon patient´s request. Some soft tissue remained attached to the root apex (Figure 6-1B and C). The root was separated from the crown and the two portions were processed for histology. Histologic sections of the crown, cut on a mesio-distal plan confirmed that the carious lesion was very close to the pulp chamber (Figure 6-1D), although a direct pulp exposure could not be seen in any of

the serial sections. A partly empty space was present in the pulp horn subjacent to the caries with amorphous debris heavily colonized by bacteria, surrounded by a severe concentration of inflammatory cells (Figure 6-1D-F). Bacteria were shown to enter the pulp horn through dentinal tubules (Figure 6-1G).

At a short distance away from the pulp horn, at the root canal orifice, a completely different picture could be observed, with intact odontoblast layers on opposite walls and absence of inflammation (Figure 6-1H). Longitudinal sections of the single conical root cut through the main foramen showed vital tissues in the foraminal area, connected to the periapical soft tissue (Figure 6-1I and L). Early resorption of the apical canal walls could be observed (Figure 6-1L and M). The soft tissue attached to the apical surface was thicker than a normal PDL, and showed abundance of collagen fibers, hyperemic vessels, and scattered chronic inflammatory cells (Figure 6-1J and K). Pulp stones (free and embedded) were present in the apical canal, together with hyperemic vessels (Figure 6-1N-P).

This case clearly confirms that when necrosis and bacterial colonization occur in a limited portion of the pulp chamber, periradicular inflammatory changes may already be observed. The occurrence of these inflammatory reactions

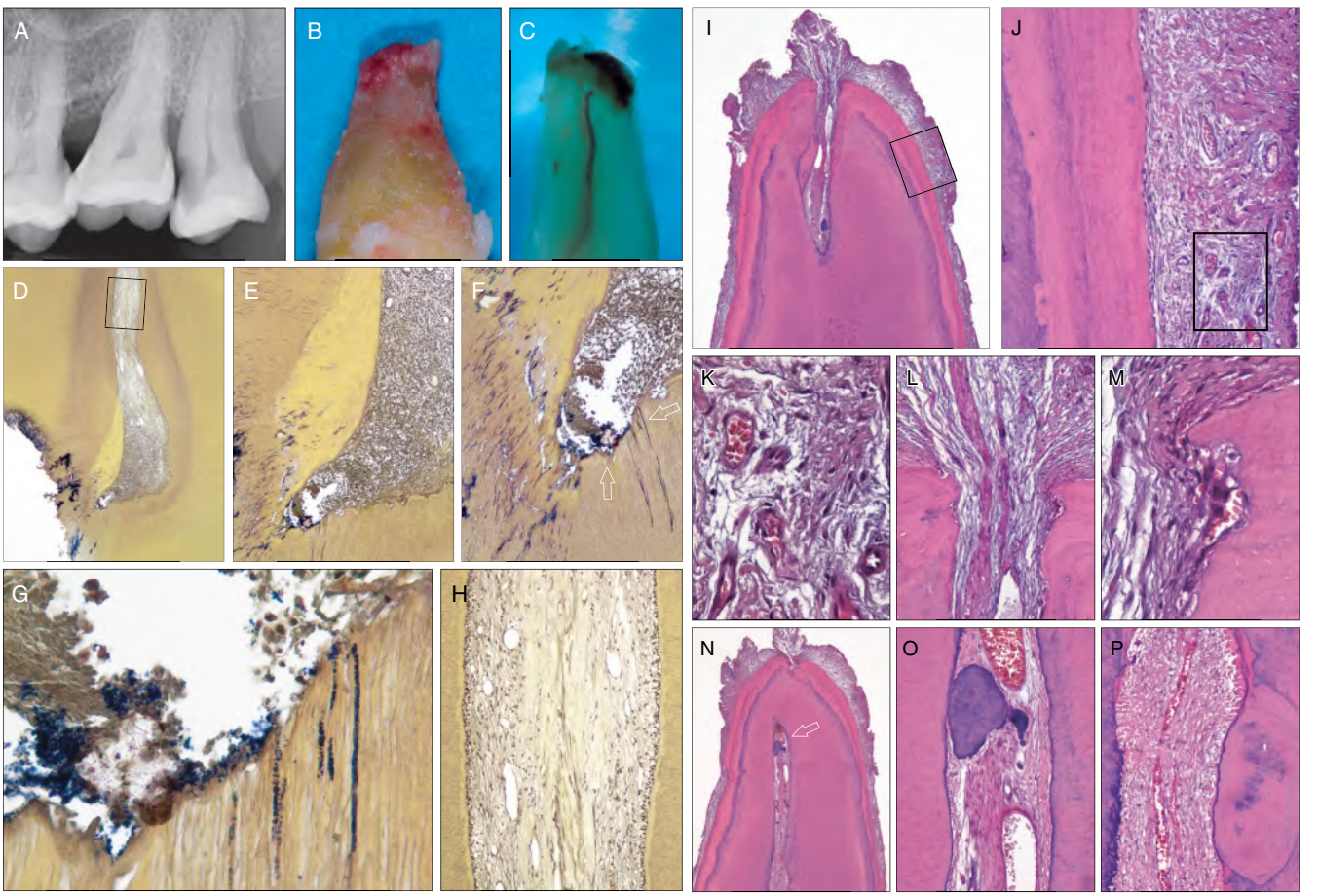


FIGURE 6-1 ***A***. Diagnostic radiograph. ***B***. Mesial view of the extracted tooth. ***C***. Photograph of the radicular portion at the end of the demineralization process, (buccal view). ***D***. Overview of the caries lesion and subjacent pulp chamber. No pulp exposure is evident (Taylor's modified Brown & Brenn, original magnification ×16). ***E***. Detail of the coronal pulp horn in D (×50). ***F***. Higher magnification of the pulp horn. The empty space is surrounded by a concentration of inflammatory cells, indicating a microabscess (×100). ***G***. High power view of the area of the pulp horn indicated by arrows in F. Necrotic debris heavily colonized by bacteria (×400). ***H***. Magnification of the area demarcated by the rectangle at the orifice in D. The pulp is not inflamed at this level (×100). ***I***. Section cut through the main foramen (Hematoxylin and eosin, original magnification ×16). ***J***. Magnification of the rectangular area in I. Connective tissue with abundance of collagen bundles and hyperemic vessels (×100). ***K***. High power view of the rectangular area in J (×400). ***L***. Detail of the foraminal area in I (×100). ***M***. High power view of the right foraminal wall in L. Resorption lacuna with a multinucleated giant cell (×400). ***N***. Section cut at some distance from that in I, not showing the foramen (original magnification ×16). ***O***. Magnification of the area indicated by arrow in N. A large pulp stone and dilated vessels can be observed (×100). ***P***. Pulpal area coronal to that shown in N. Pulp stone embedded in the dentin wall is present, facing an area with congested vessels (×100).

at a considerable distance from the necrotic area colonized by bacteria is possibly explained by the diffusion of bacterial virulence factors and/or chemical mediators of inflammation through the vital connective tissue up to the periradicular region. In other words the inflammatory response is not only restricted to the narrow area immediately in contact with the most advanced bacteria, but bacterial products and inflammatory mediators may involve a large tissue area, although with less intense reaction because of the dilution in tissue fluids.[9-12]

In later stages, increasing portions of the coronal pulp are affected by degeneration, and necrosis reaches the root pulp, moving slowly in an apical direction. The clinical consequence can be periapical radiolucency as in the case illustrated in Figure 6-2. This is the left mandibular first molar of a 19-year-old woman with destructive caries. The pulp did not respond to thermal and electric pulp tests, and the radiograph showed periapical radiolucencies on both roots (Figure 6-2A). The diagnosis of necrotic pulp was made. The patient did not accept any treatment aimed at conservation and the tooth was extracted and processed for light microscopy. Sections, cut on a mesio-distal plane, revealed necrotic in the coronal third in the mesial root the tissue. However, toward the middle third, there was a transition between necrotic and viable pulp tissue, characterized by the typical gradient of tissue reaction: infected/necrotic tissue—acute inflammation—chronic inflammation—uninflamed tissue (Figure 6-2B-G).[10] It is important to point out that this vital tissue lacks odontoblasts and predentin, which is replaced by a less tubular or atubular calcified tissue resembling cementum (Figure 6-2G). For this reason, this tissue cannot be properly referred to as "pulp tissue." The periapical pathologic tissue appears comprised a severe concentration of acute and chronic inflammatory cells and absence of bacteria (Figure 6-2H and I). In conclusion, an apparently illogical condition is observed, characterized by a portion of relatively uninflamed tissue between two areas of severe inflammation.

The histologic features observed in this case confirm that it is not necessary for the entire pulp to be necrotic and for the frontline of infection, established at the apical foramen, to develop apical periodontitis.[9–13] Lin et al.[14] conducted a clinical and histological study on 75 carious teeth with radiographic evidence of apical periodontitis. After access preparation, they carefully extirpated the pulp tissue with barbed broaches, and performed histologic and histobacteriologic analyses of the tissue fragments. They observed that in most cases, pulp necrosis was only partial, with inflamed connective tissue and intact nerve fibers. This observation was later confirmed by Ricucci et al.[12] in a histologic study of 50 extracted teeth with an apical periodontitis lesion attached. Vital tissue, with varying degree of inflammation, was observed in the apical canal in 18 teeth, which represents approximately one third of the cases.

The possible presence of structured tissue between the bacterial front and the apical foramen in teeth with apical periodontitis lesions was also observed in a scanning electron microscopy study.[15] Therefore, in some teeth with apical periodontitis, microbial infection has not reached the very apical portion of the canal. This condition is referred to as "partial necrosis" and helps explain why an unanesthetized patient may feel a sharp pain when a file is inserted in the canal of a tooth with apical periodontitis even before the instrument reaches the apical canal. This also helps to explain the high rate of success in the treatment of teeth with apical periodontitis with instrumentation limited to 1 mm short of the apex,[16] in which apical patency files were not used.

With time, additional tissue becomes involved with necrosis in an apical direction, with or without concomitant clinical symptoms. Microabscesses can be observed in the apical pulp, while the most apical canal may still harbor structured connective tissue in direct continuation with the apical periodontitis lesion (Figure 6-3). Bacterial biofilms are located at the borderline between infection and inflamed tissue (Figure 6-3D and G). In more advanced stages, necrosis can reach the apical foramina. It is not uncommon to observe ingrowth of granulation tissue into the foramen, in an extreme attempt to halt the advance of the frontline of bacterial infection (Figures 6-4 and 6-5). Figure 6-6 illustrates the condition in which microbial infection has finally reached the end of the canal that appears clogged with a thick biofilm. It is evident that this was a condition of longstanding pulp necrosis, where microorganisms had time to organize in a complex biofilm structure that occupied the entire apical canal. At this point, host defenses are generally located outside the root canal in the apical PDL.

In cases with severe carious breakdown of the crown and direct exposure of the root canal space to the oral environment, food or other foreign bodies may gain access to the canals and be packed in an apical direction by mastication forces. Figure 6-7 illustrates a maxillary central incisor whose apical canal appeared to be clogged with various food remnants (including meat fibers), hair, and a toothpick fragment protruding through the foramen into the periapical area. These structures were enmeshed with amorphous debris heavily colonized by microorganisms (Figure 6-7G-L). Note that biofilms are formed on the external surface of hair (Figure 6-7H) and microorganisms also filled the large cellulose cells of the wood fragment (Figure 6-7J and K).

Intensity of Periradicular Tissue Response

The intensity of PDL response to the canal infection is directly proportional to the intensity of microbial aggression. The host-pathogen relationship will result in an acute inflammatory response (symptomatic apical periodontitis or

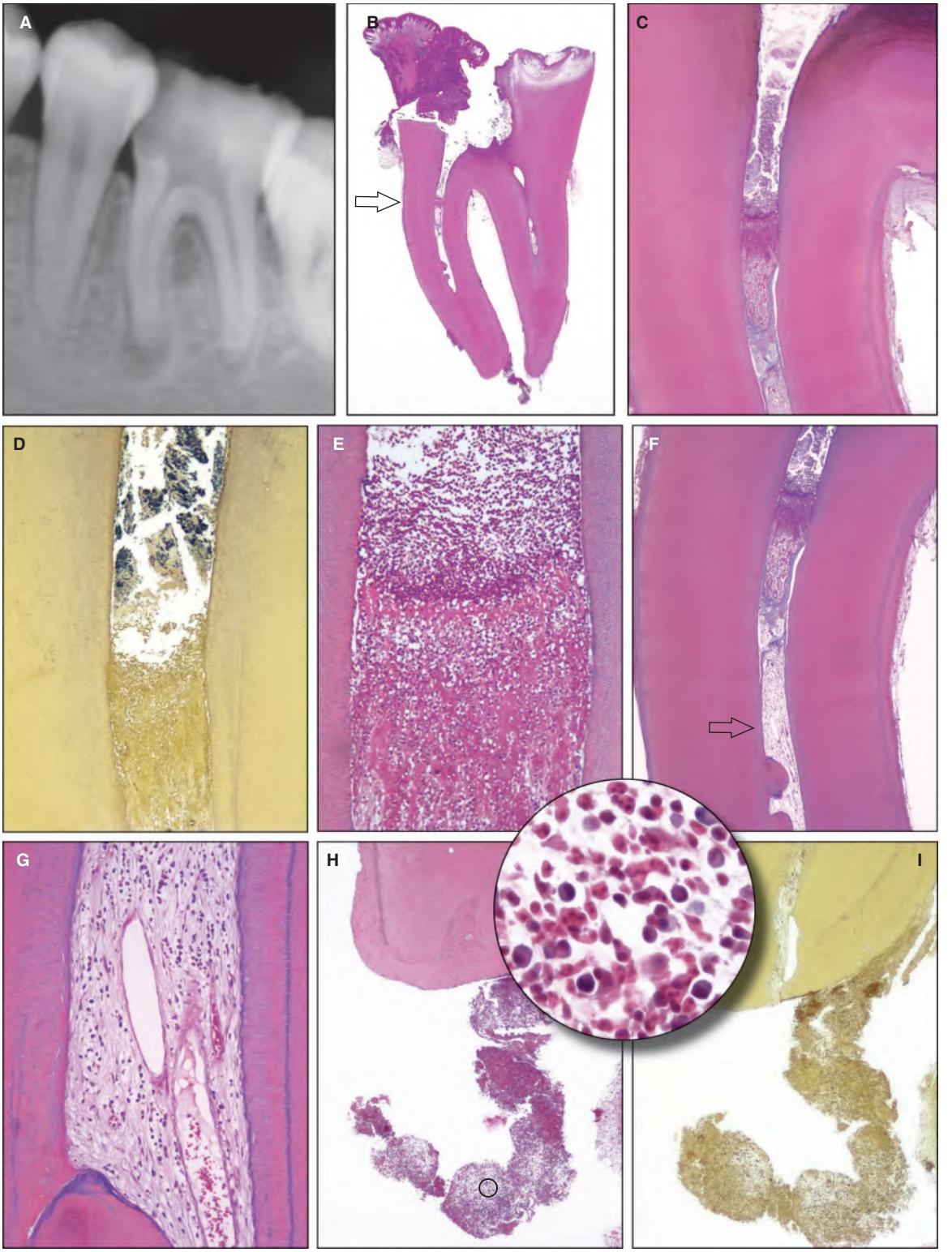


FIGURE 6-2 ***A***. Diagnostic radiograph. Apical periodontitis lesions are present on both roots. ***B***. The tooth was extracted and processed for light microscopy (Hematoxylin and eosin, original magnification ×2). ***C***. Area of the mesial root indicated by the arrow in B. A transition from necrotic to vital pulp can be appreciated (×16). ***D***. Section proximal to that in C. Microbial aggregations can be seen in the necrotic portion of the pulp. Apically, transition to a structured tissue with no infection can be seen (Taylor's modified Brown and Brenn, original magnification ×50). ***E***. Detail from the transitional area. Polymorphonuclear leukocyte concentration on top, facing a structured connective tissue with chronic inflammatory cells (×100). ***F***. Middle third of the root. Large pulp stones adhering to the root canal wall (×16). ***G***. Magnification of the area indicated by arrow in F. Moderate concentration of chronic inflammatory cells and vasodilation (×100). ***H***. Root tip with the periapical pathologic tissue that was partly detached from the apex during extraction (×16). Inset. High power view from the lesion. Acute and chronic inflammatory cells (×400). ***I***. Section proximal to that in H. Non-infected vital tissue in the apical canal (×16).

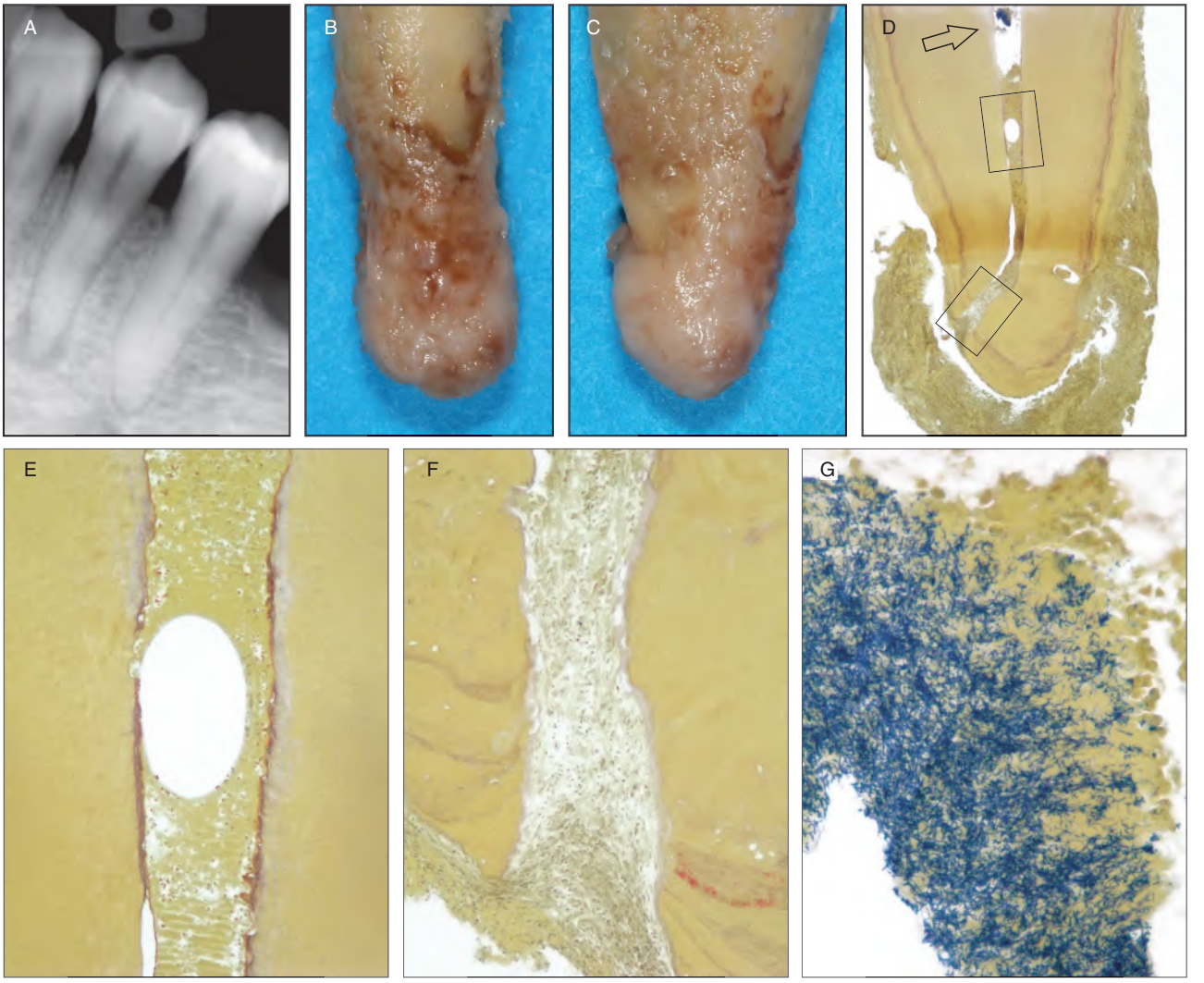


FIGURE 6-3 Mandibular first premolar of a 52-year-old woman with cervical caries and clinically necrotic pulp. ***A***. Radiograph showing apical radiolucency associated with the tooth. Despite the tooth being treatable, the patient requested extraction. ***B*** and ***C***. The apical periodontitis lesion remained attached to the apex at extraction. Buccal and mesial views. ***D***. Section encompassing the root canal and one apical ramification. A second foramen can be seen on the opposite side of the apex. Note that the tissue in the most apical part of the left ramification is not necrotic. The apparently empty space between the pathologic tissue and the apex is a surgical artifact (Taylor's modified Brown and Brenn, original magnification ×16). ***E***. Magnification of the area demarcated by the upper rectangle in D. A microabscess is present in a disintegrated tissue (×100). ***F***. Magnification of the area demarcated by the lower rectangle in D. Non-infected vital tissue is present in the ramification (×100). ***G***. High power view of the area indicated by the arrow in D. Necrotic tissue heavily infected (×400).

acute apical abscess) or a chronic response (asymptomatic apical periodontitis or chronic apical abscess), resulting in bone destruction. Inflammation is by and large effective in confining the infection to the root canal space, but cannot eliminate the infection therein. Therefore, microorganisms are located in a strategic position within the necrotic root canal, because the lack of an active circulation in the necrotic tissue prevents phagocytes to have a direct access to the source of infection. Additionally, the presence of biofilm structures protects bacteria from the host defense attacks. For more details, see Chapter 3.

Although the source of infection is not effectively eliminated, an equilibrium between aggression and defense is usually achieved. This equilibrium is often characterized by chronic inflammation, clinically referred to as asymptomatic (or chronic) apical periodontitis. As the tooth remains untreated and infection persists in the root canal system, the chronic process gives rise to bone resorption to the point of being radiographically detected. By adequately treating the root canal, the clinician promotes an imbalance favoring the host. Consequently, healing of the periradicular tissues is initiated. If the microbial aggression is significant, due to high bacterial counts and/or virulence, the host can mount an acute inflammatory response to curb the advance of infection and re-establish equilibrium. This gives rise to a condition clinically characterized as symptomatic (or acute) apical

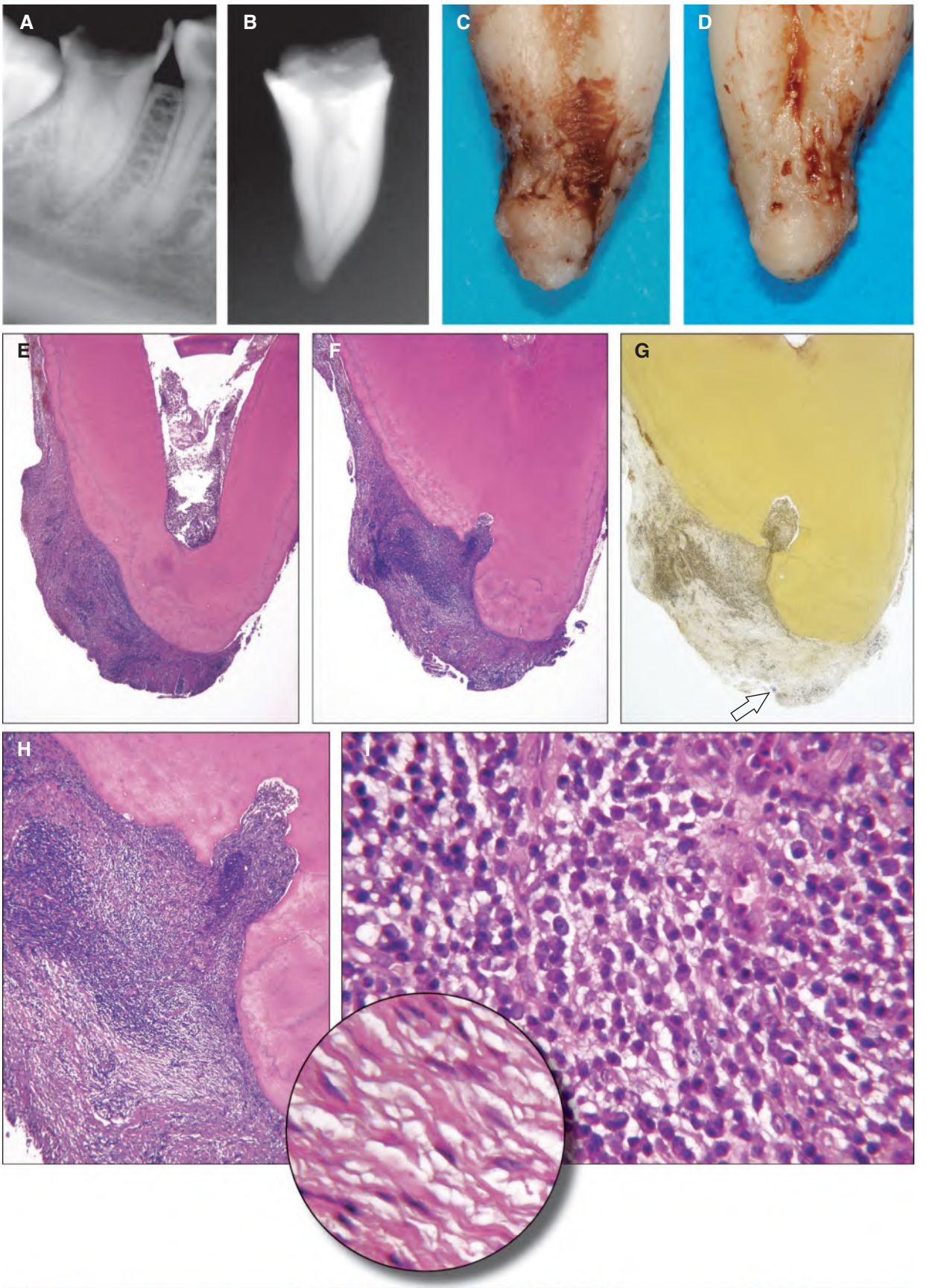


FIGURE 6-4 (*Continued on facing page*)

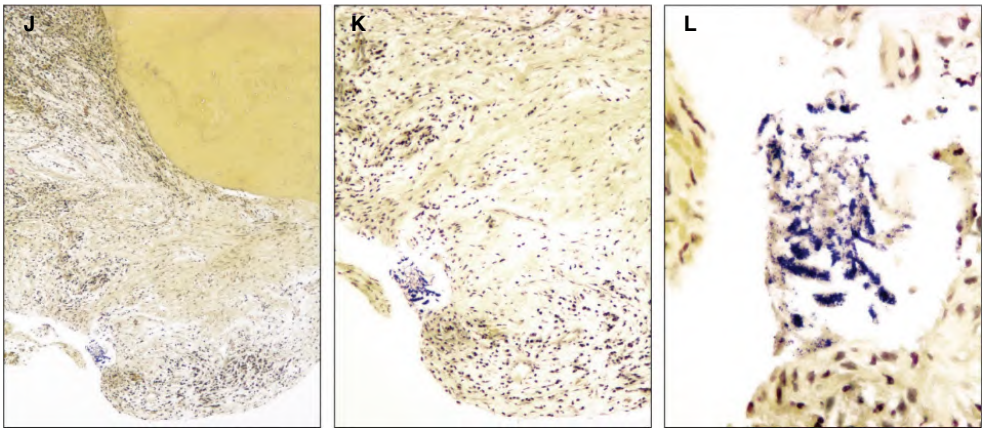


FIGURE 6-4 Mandibular second molar with a crown, totally destroyed by caries. The pulp was necrotic. No symptoms were present. ***A***. Radiograph showing periapical radiolucency associated with the tooth. ***B***. Radiograph of the extracted tooth. ***C*** and ***D***. The lesion remained attached to the apex after extraction. Buccal and lingual views. ***E***. Section cut through the canal, not encompassing the foramen (Hematoxylin and eosin, original magnification ×16). ***F***. Section cut through the foramen that emerges laterally to the root apex and contains tissue in direct continuation with the periapical lesion (×16). ***G***. Section taken not far from that in F (Taylor's modified Brown and Brenn, original magnification ×16). ***H***. Detail of the periapical pathologic tissue in F. Concentration of inflammatory cells in the area surrounding the foramen. Fibrous tissue with predominance of collagen bundles more peripherally (×50). ***I***. High power view from the center of the lesion. Abundance of chronic (mononuclear) inflammatory cells, mainly plasma cells (×400). Inset. High power view from the peripheral area. Fibroblasts and collagen bundles. Absence of inflammatory cells (×400). ***J–L***. Progressive magnifications of peripheral portion of the lesion indicated by arrow in G. Microbial aggregation can be seen (×50, ×100, and ×400).

periodontitis. The inflammatory response can become exacerbated, resulting in purulent exudate formation that characterizes an *acute apical abscess*.

Extraradicular Extension of Root Canal Infection

Infection is usually confined to the root canal, but occasionally can advance to the periradicular tissues. For more details, see Chapter 3. Acute apical abscess is the most common form of extraradicular infection. There is, however, another form of extraradicular infection that, unlike acute abscess, is usually characterized by absence of overt symptoms. This condition encompasses the establishment of infection in the periradicular tissues, either by forming cohesive actinomycotic colonies within the body of the inflammatory lesion (a condition referred to as apical actinomycosis),[17] or adhering to the apical external root surface in the form of biofilms.[18,19]

The diagnosis of apical actinomycosis by light microscopy is possible only in the presence of the typical structure of a "ray fungus" or actinomycotic rosettes in tissue sections.[20] An actinomycotic colony typically consists of a central area more or less intensely stained, containing intertwining and branching filaments, held together by an extracellular matrix with peripheral filaments in the form of clubs or rods.[20-22] The colony is usually surrounded by several layers of polymorphonuclear neutrophils (PMNs).

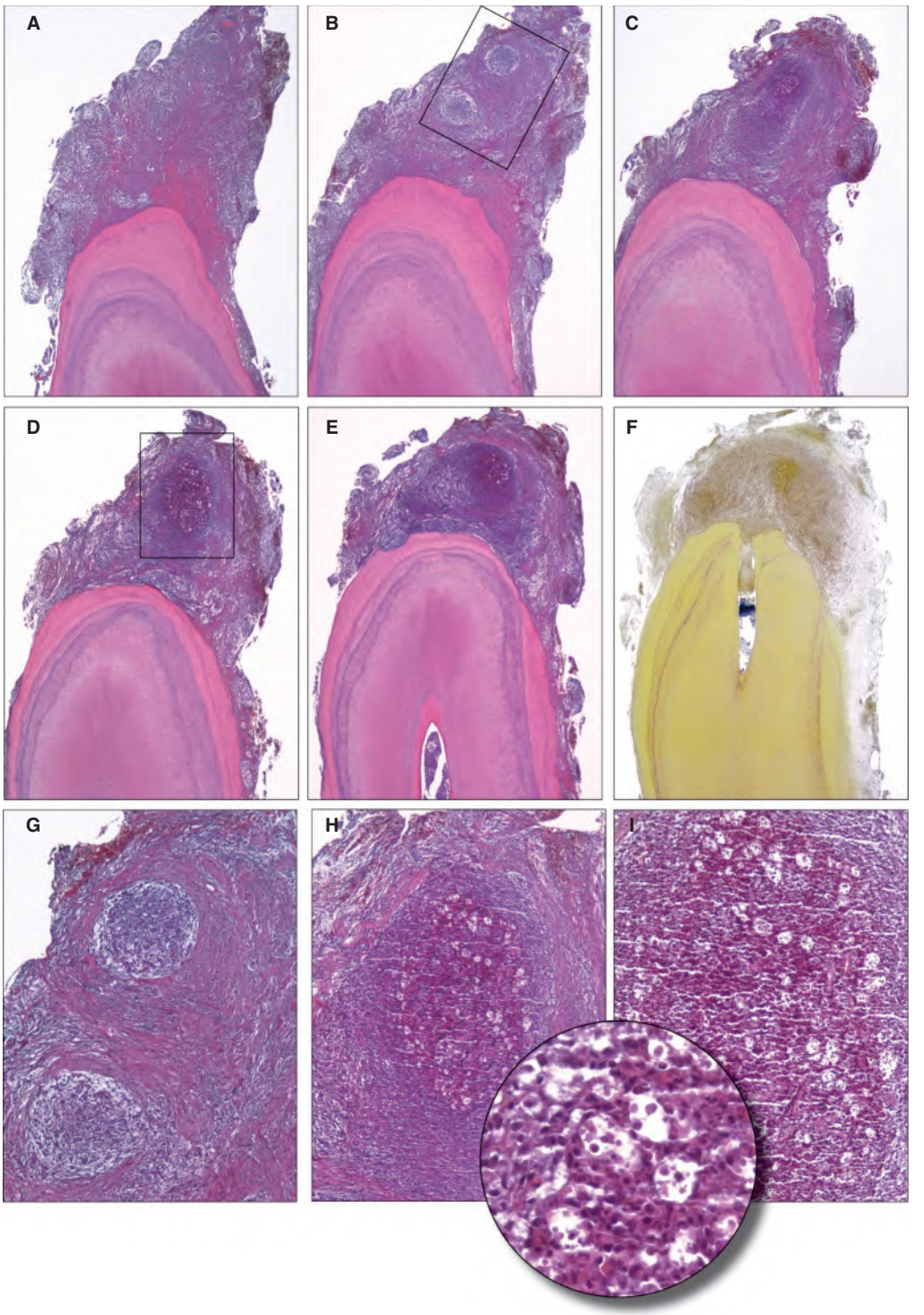
Another form of extraradicular infection is the spread of bacterial biofilms from the apical canal to the outer root surface, around the apical foramen/foramina. Biofilm-like structures have been observed on the external surface of the apical portion of teeth with infected canals. Tronstad et al.[18] reported that in some teeth with posttreatment disease, the external apical surface was covered by a continuous and smooth layer, without a defined structure, containing various bacterial morphotypes. Bacteria held together by extracellular material were present in resorption lacunae and other irregularities of the root surface. Lomçali et al.[23] observed chains or layers of bacteria enclosed in a dense matrix on the surface of teeth with asymptomatic apical periodontitis lesions. In 26 similar biopsies from untreated teeth, Siqueira and Lopes[24] reported one case in which dense bacterial deposits were seen beyond the apical foramen, including forming a corncob-like structure.

Extraradicular infection is a rare phenomenon. Based on the few cases described in the literature, it is possible to infer that extraradicular infections occur in teeth with massive infection of the root canal space. Such massive infection may result from prolonged exposure of the canal to the oral environment due to gross caries, inadequately restored teeth or sinus tracts.

The early phases of biofilm formation and adhesion to the external apical surface are illustrated in Figure 6-8. It is a maxillary second premolar of a 58-year-old patient with the crown destroyed by caries, and the canals exposed to the oral environment for a long period. The tooth was extracted with an apical periodontitis lesion attached, and bacterial staining demonstrated some small aggregates of filamentous bacteria just outside the buccal canal (Figure 6-8E,F). The cementum

layer was detached from dentin. The presence of debris in these spaces indicates that this condition is not histological artifact (Figure 6-8E). The clinical relevance of this observation is clear, as these spaces can be colonized by microorganisms consequently not responding to nonsurgical root canal treatment procedures.[25]

Sometimes, parts of the biofilm structure may even become mineralized and calculus formation is observed. This may be associated with a longstanding sinus tract. Ricucci et al.[26] described two cases in which calculi were present on the external root surface of teeth with posttreatment apical periodontitis.



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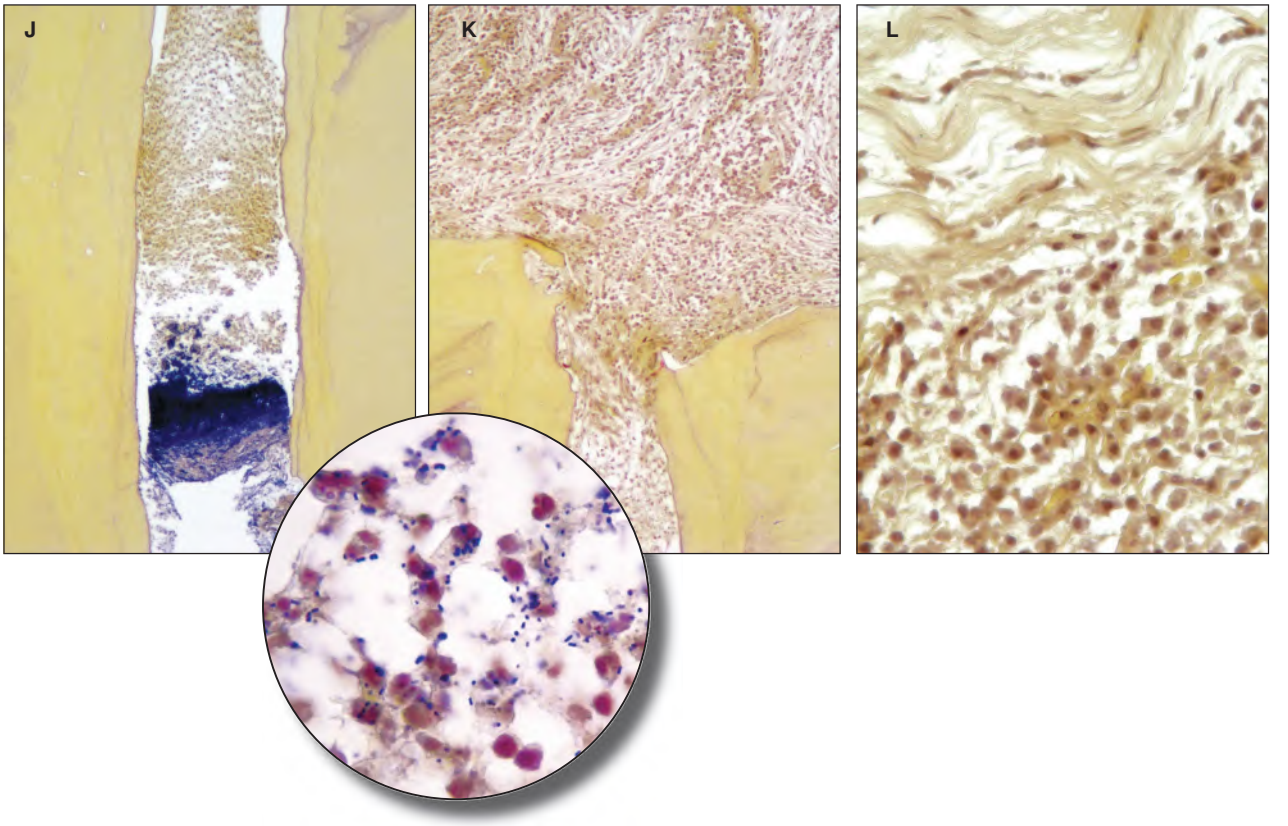


FIGURE 6-5 Mesio-buccal root apex of a maxillary first molar in a 54-year-old man. ***A–F***. Series of sections taken approximately 30 sections from each other (Hematoxylin and eosin and Taylor's modified Brown and Brenn, original magnification ×16). ***G***. Magnification of the area demarcated by the rectangle in B. Two areas of severe inflammation surrounded by fibrous connective tissue (×50). ***H***. Central portion of the lesion demarcated by the rectangle in D (×50). ***I***. Further magnification showing severe concentration of PMNs and microcavities, leading to diagnosis of apical abscess (×100; inset ×400). ***J***, Detail from the apical canal in F. A dense microbial biofilm filling the canal lumen, faced with inflammatory tissue (×100). Inset. High power view from the interface between the bacterial biofilm and the inflammatory tissue. PMNs engaged in an intense phagocytic activity. Microbial fragments can be seen in their cytoplasms (×1000). ***K***. Magnification of the apical canal in F. The inflammatory tissue filling the apical canal is in direct continuation with the periapical pathologic tissue. Resorption of the apex can be appreciated (×100). ***L***. Magnification from the periphery of the lesion in F. The inflammatory tissue is limited externally by dense collagen bundles with few scattered inflammatory cells (×400).

Figure 6-9 shows an advanced condition in which the apical third of the mesial root of a mandibular first molar of a 35-year-old man is completely covered by calculus. The patient stated that the tooth had been treated endodontically 4 years previously and abscess symptoms had been occasionally present. No sinus tracts were present at the time of visit. A radiograph could not disclose the root tips, but showed inadequate endodontic treatment and a large radiolucency (Figure 6-9A). The patient was in pain and requested tooth extraction. The tooth was sectioned and the roots were extracted separately. The mesial root showed apical resorption and the apex was completely enveloped by a dark calculus (Figure 6-9B, C). Cross-cut sections showed that the untreated apical canals were filled with necrotic debris, dentin spicules, and bacteria (Figure 6-9D and E). Calculus exhibited masses of bacteria interspersed in an abundant extracellular matrix (Figure 6-9F).

The calculus deposits are conceivably originating from mineralization of an extraradicular biofilm of endodontic origin, present on the external surface of the root. Saliva is the main source of mineralization of supragingival calculus, whereas serum transudate of gingival crevicular or pocket fluid provides the minerals for subgingival calculus development.[27] As for periradicular diseases, a longstanding sinus tract may function as a possible route of communication between the periradicular area and the external environment. In addition to minerals available from tissue fluids, the passage of minerals and salts from the oral fluids into the apical periodontitis lesion, via sinus tract, may help explain calcification of the biofilm present on the apical root surface. However, an extraradicular bacterial biofilm can become mineralized even in the absence of a sinus tract, and the source may be the inflammatory exudate and periradicular tissue fluids saturated with calcium and phosphate from the solubilization of both bone and tooth.

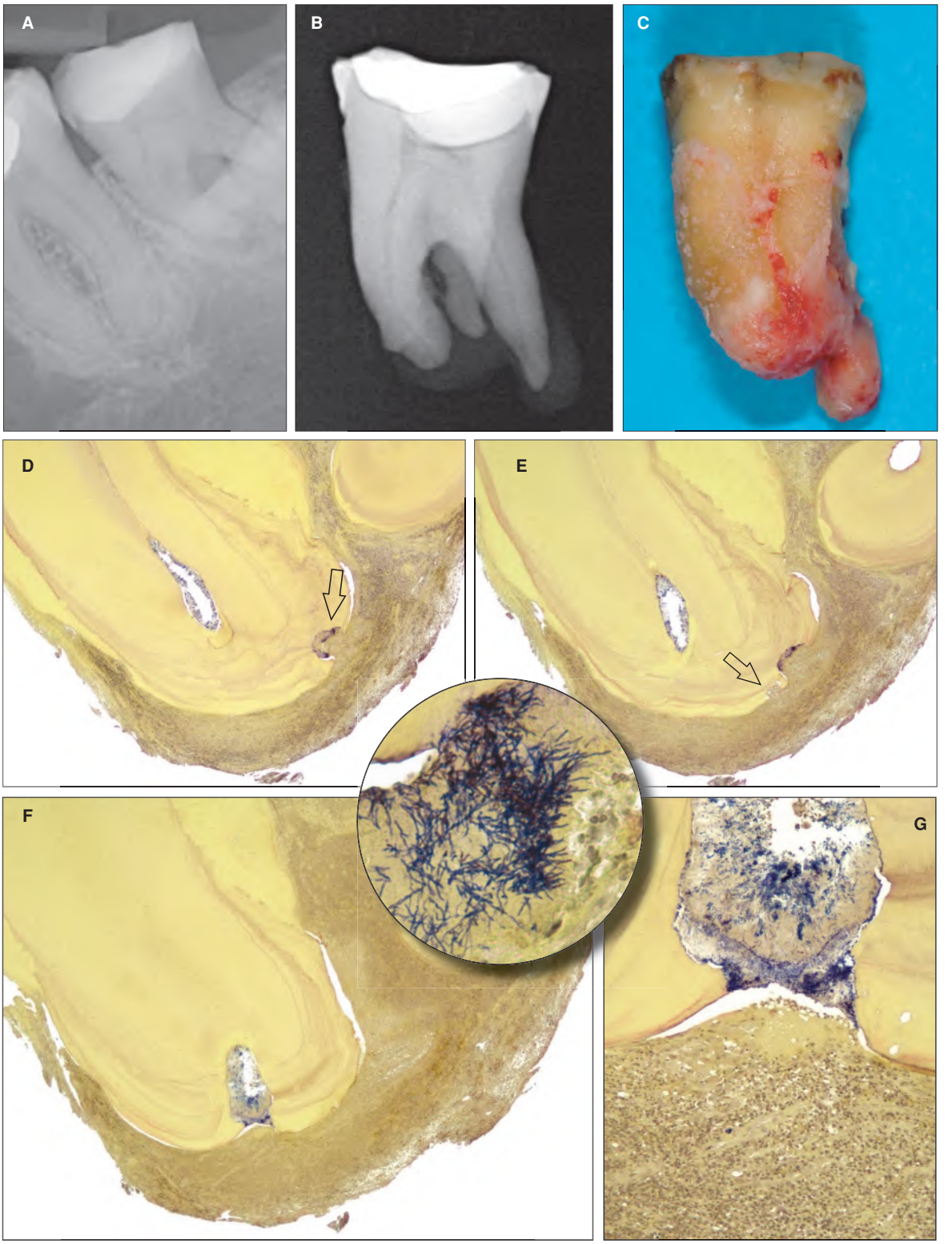
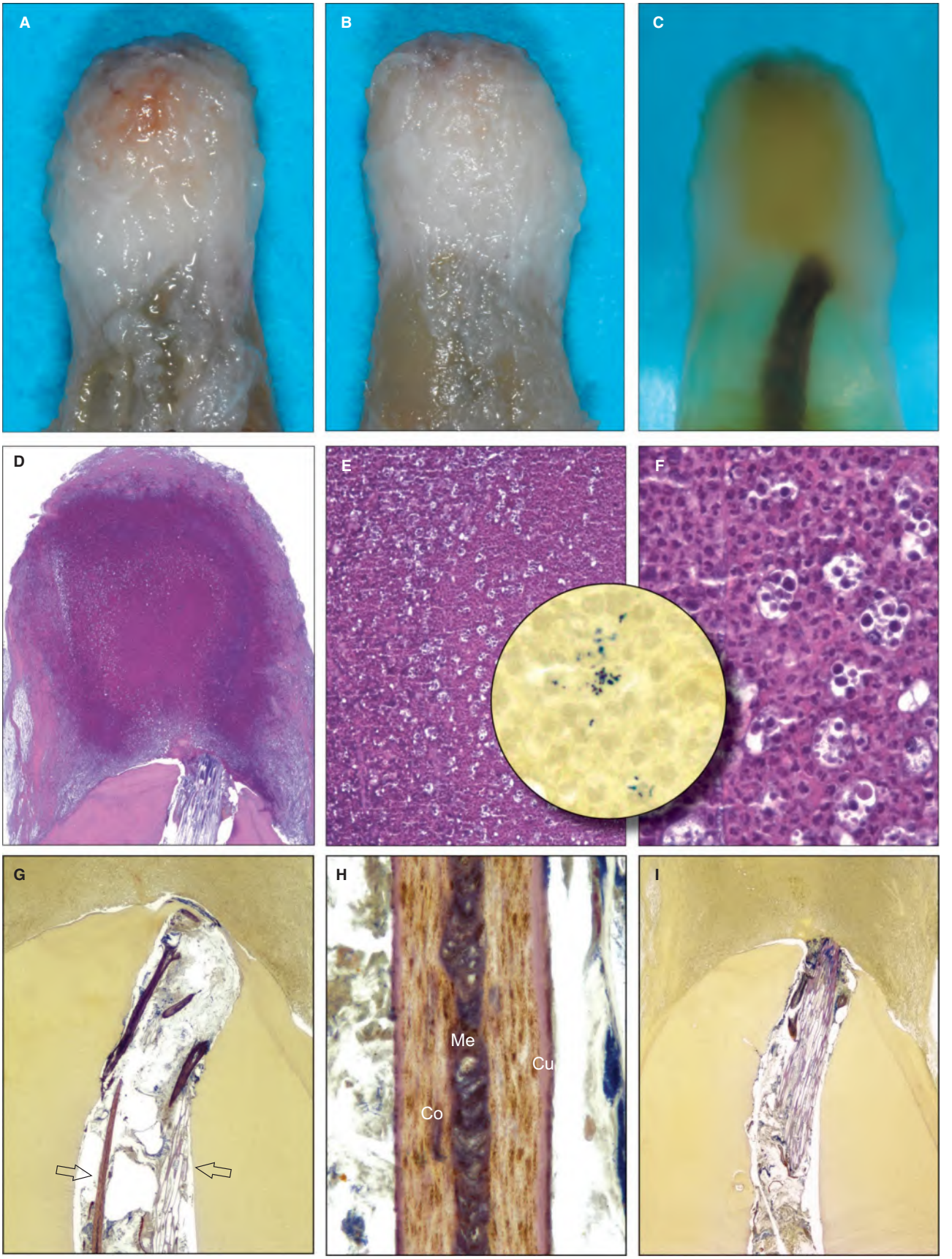


FIGURE 6-6 A. Symptomatic mandibular third molar with necrotic pulp in a 77-year-old woman. ***A***. Diagnostic radiograph. ***B***. Radiograph of the extracted tooth shows apical resorption of the mesial root. ***C***. Photograph of the extracted tooth. Pathologic tissue remained attached to all roots at extraction. ***D***. Section passing through one mesial canal, encompassing one apical ramification. The foramen is filled with a thick microbial biofilm (Taylor's modified Brown and Brenn, original magnification ×16). Inset. High power view of the area of the foramen indicated by arrow in D. The biofilm is mainly composed of filamentous forms (×400). ***E***. Section taken 30 sections after that in D. The exit of another ramification appears mesially (arrow) (×16). ***F***. Section taken 50 sections away. The exit of another ramification, filled with a dense biofilm, appears at the center of the root tip (×16). ***G***. Detail from the foramen in F (×100).



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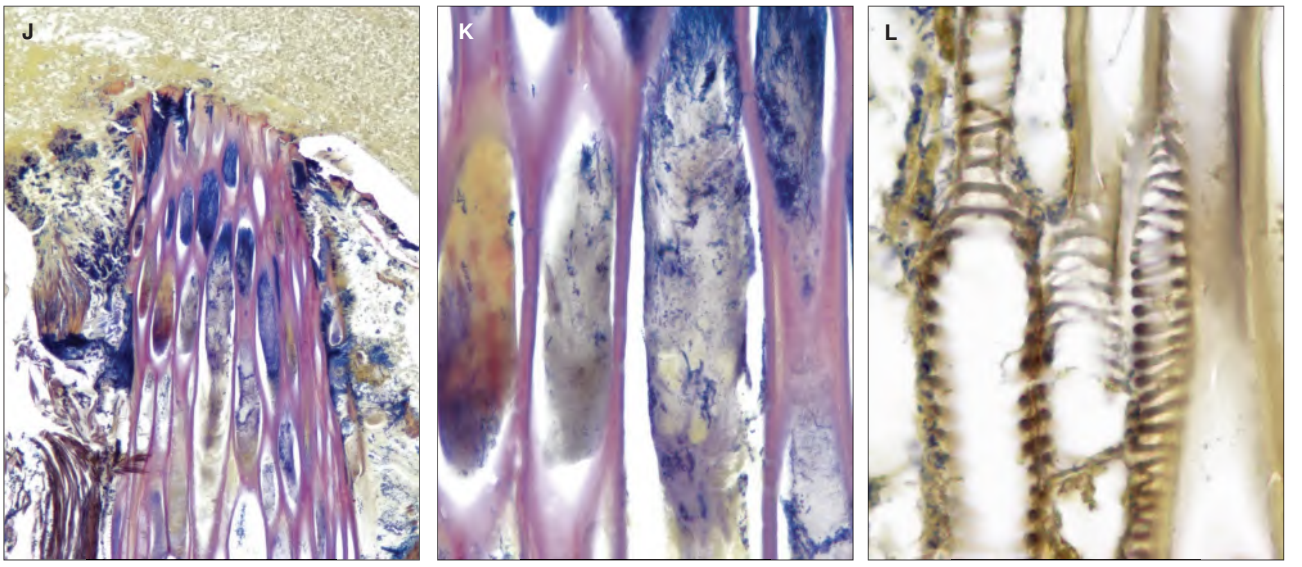

FIGURE 6-7 Symptomatic maxillary central incisor extracted with an apical periodontitis lesion firmly attached to the root tip. ***A*** and ***B***. Buccal and lingual views. ***C***. Photograph of the cleared specimen before embedding in paraffin. ***D***. Longitudinal section cut at the center of the foramen. Overview of the root tip with the apical periodontitis lesion (Hematoxylin and eosin, original magnification ×16). ***E***. Severe accumulation of PMNs with microcavities at the center of the lesion (×100). ***F***. High power view from E (×400). Inset. Small microorganisms aggregations are spread through the periapical inflammatory tissue (Taylor's modified Brown and Brenn, original magnification ×400). ***G***. Other section. Necrotic material and several elongated unusual bodies are present in the apical canal (×25). ***H***. High power view of the left body indicated by left arrow in G. The structure of a hair can be recognized (Me: medulla. Co: cortex. Cu: cuticle) (×400). ***I***. Other section. An elongated body fills the apical canal protruding into the periapical tissue (×16). ***J***. High magnification reveals cellulose fibers that can be recognized by the typical large vegetal cells. This is likely a fragment of a toothpick. These cells and the surrounding necrotic tissue are heavily colonized by microorganisms (×100). ***K***. High power view of the cellulose cells filled with microorganisms (×400). ***L***. High power view of the area indicated by right arrow in G. Food remnants resembling meat fibers (×400).

CLINICAL CLASSIFICATION OF PERIRADICULAR DISEASES

Periradicular diseases of endodontic origin have been classified in many ways. In this chapter, terminology for clinical classification of these diseases follows the reports of the American Association of Endodontists Consensus Conference on diagnostic terminology.[28] Accordingly, periradicular lesions are basically divided into three main clinical groups: asymptomatic and symptomatic apical periodontitis; acute and chronic apical abscesses; and condensing osteitis.

Normal Periradicular Tissues

A tooth with normal healthy periradicular tissues shows no sensitivity to periradicular (percussion or palpation) tests. Radiographically, it presents with intact lamina dura surrounding the root and a uniform PDL space.

Asymptomatic Apical Periodontitis

Asymptomatic apical periodontitis is characterized by inflammation and destruction of the apical periradicular tissues and appears as a radiolucent area that is in the large majority of cases located around the root apex, but may occasionally develop laterally to the root or in the furcal region.

As the name implies, it presents with no symptoms, but the patient may report a previous painful episode. On clinical examination, the cause of pulp necrosis is evident, including the presence of extensive caries or coronal restoration. The tooth crown may be darkened due to pulp necrosis.

The results of pulp sensitivity tests are negative because the pulp is necrotic. Periradicular tests (percussion and palpation) yield negative results. In some cases, the lesion may cause bone fenestration at the level of the apical part of the root, so that palpation may reveal a slight increase in volume, as the inflammatory proliferative lesion is located in the submucosal tissue.

In the initial phase of asymptomatic apical periodontitis, radiographs show a thickened PDL space. In more advanced stages, a radiolucent area around the root apex (or sometimes laterally to it) is seen. The lesion usually has well-defined borders and exhibits loss of the integrity of the lamina dura.

Asymptomatic apical periodontitis is classified histopathologically as granuloma or cyst (see next section). These two histopathologic entities are radiographically indistinguishable. Attempts have been made to differentiate between the two conditions by means other than biopsy, including electrophoresis of the fluid sampled from the lesion,[29,30] computerized tomography,[31] cone-beam computed tomography,[32] ultrasound,[33] and ultrasound real-time imaging technique (echography).[34-36] However, the results were

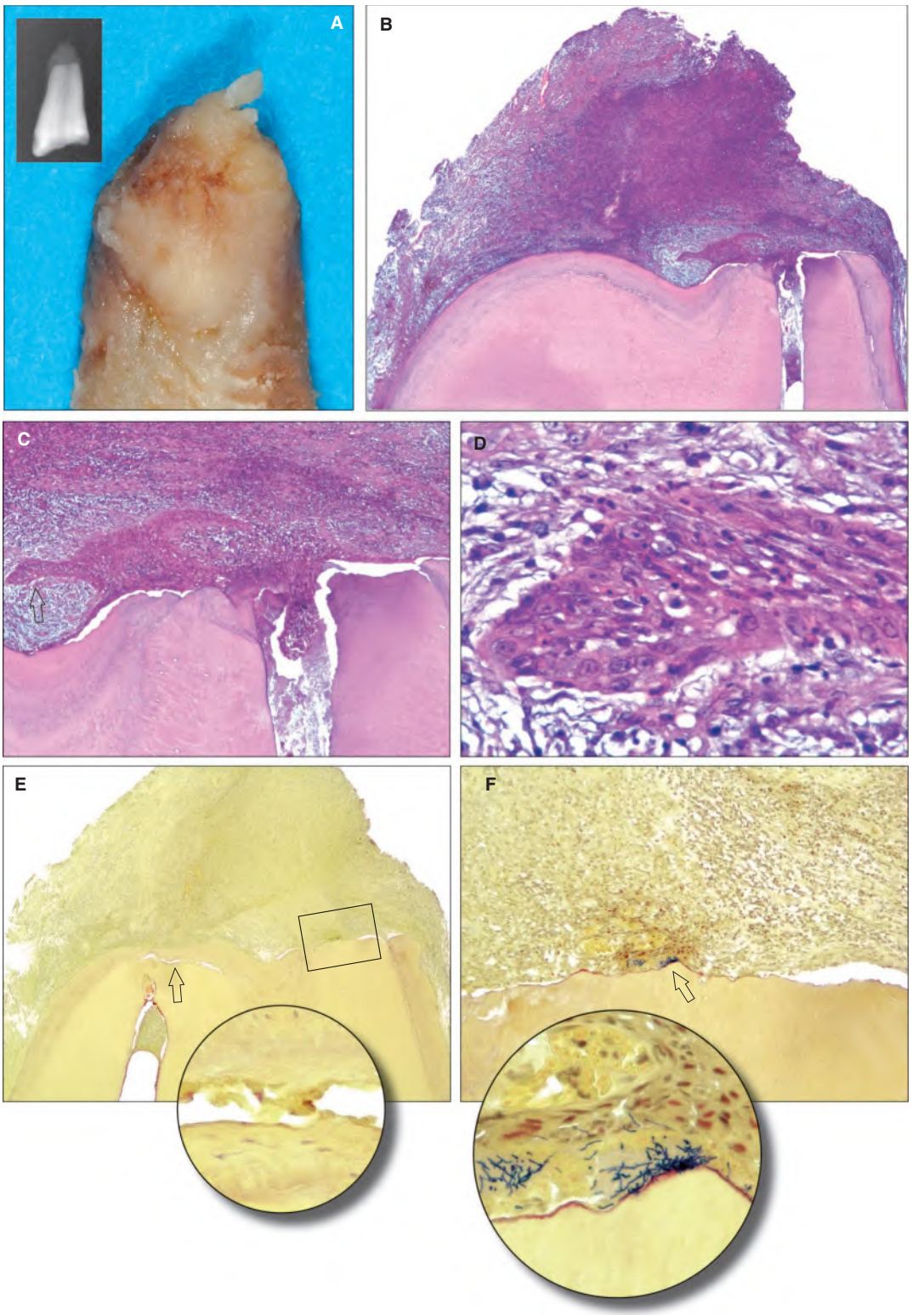

FIGURE 6-8 Maxillary second premolar extracted with an apical periodontitis lesion attached. ***A***. Mesial view. ***B***. Section passing through the palatal canal. Necrotic tissue is present in the apical canal. An epithelial strand can be seen in the periapical tissue, close to the canal opening (Hematoxylin and eosin, original magnification ×16). ***C***. Detail of the apical canal in B (×50). ***D***. High power view of the portion of the epithelial strand indicated by arrow in C (×400). ***E***. Section cut through the buccal canal. Note detachment of the cementum from underlying dentin (Taylor's modified Brown and Brenn, original magnification ×16). Inset. High power view of the area indicated by arrow in E. Necrotic debris and some cells can be seen (×400). ***F***. Magnification of the area demarcated by the rectangle in E (×100). Inset. High power view of the area indicated by arrow in F. Small microbial aggregation, some of which adhering to the apical surface (×400).

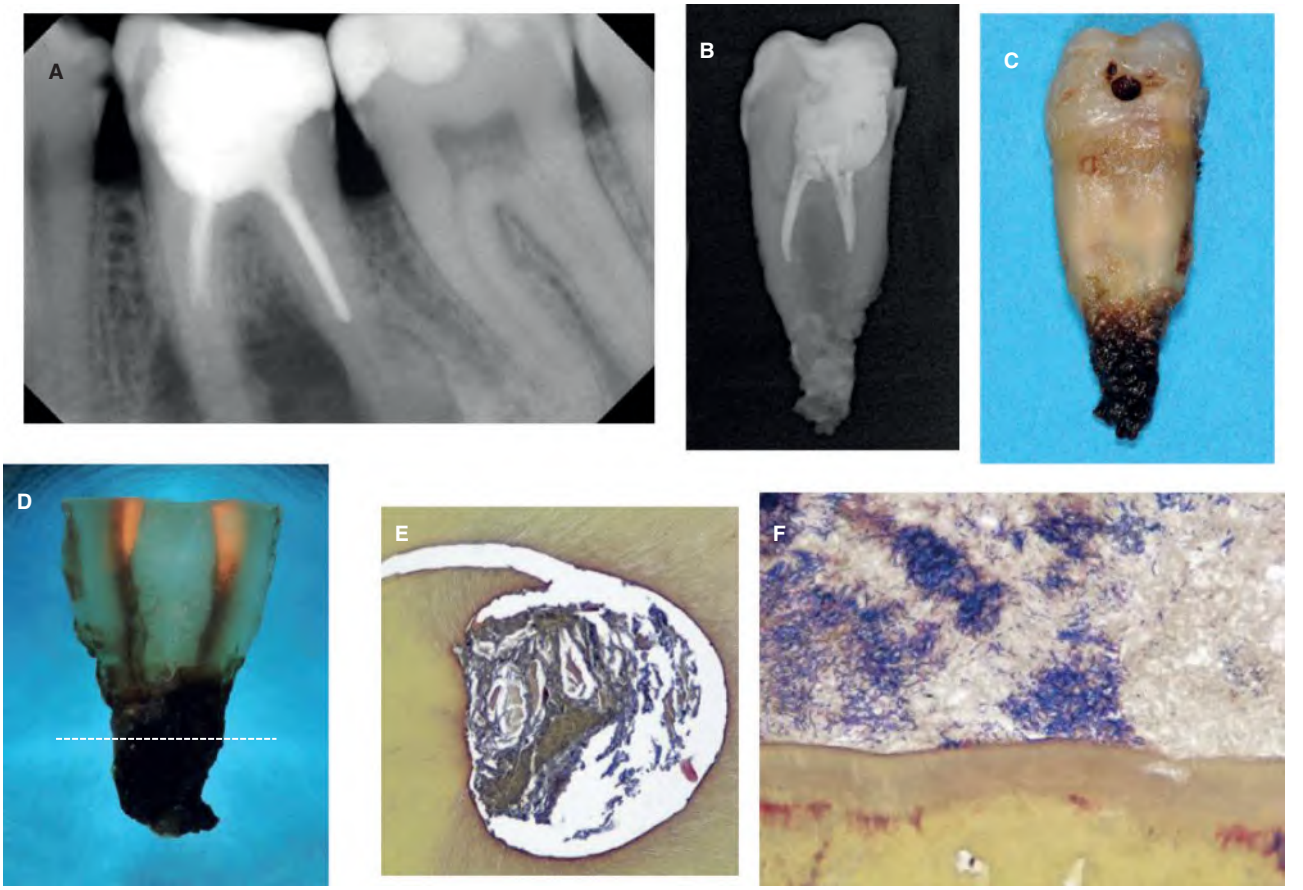

FIGURE 6-9 Symptomatic root canal-treated mandibular first molar with a large periradicular radiolucency. ***A***. The apices cannot be seen on the radiograph, but an unusual indistinct radiopacity is appreciated around the mesial root, as well as a net radiopacity around the distal root tip. ***B***. Radiograph of the mesial root in a mesio-distal projection. ***C***. Photograph of the mesial root. The root tip is completely covered by dark calculus and considerably resorbed. ***D***. Apical half of the mesial root immersed in the clearing agent before paraffin infiltration. ***E***. Cross-cut section at the level of the line in D. The buccal canal is completely filled with necrotic debris and dentin chips enmeshed with microorganisms (Taylor's modified Brown and Brenn, original magnification ×100). ***F***. Same section. Distal aspect. Microbial biofilm with abundant extracellular matrix adhering to cementum (×400).

inconclusive. In addition, the need for distinguishing the two conditions is questionable, as the treatment for both is the same. Although controversial, it seems that the outcome of the treatment for both conditions is also the same.

Symptomatic Apical Periodontitis

Symptomatic apical periodontitis is characterized by acute inflammation in the PDL, generating clinical symptoms. The patient usually complains of intense, pounding, and localized pain. The tooth is also tender to percussion. The patient may report that the tooth seems to have "grown or pushed out" of the alveolus. This is because the tooth is slightly extruded to accommodate the edema formed in the apical PDL. Chewing usually provokes or exacerbates pain.

This condition develops when the aggression caused by microorganisms and their byproducts leaching via the apical foramen is of high intensity. Pain is a result of the increase in vascular permeability associated with acute inflammation, producing an increase in tissue hydrostatic pressure and compression of sensory nerve fibers. Bacterial products, such as lipopolysaccharides (LPS), and chemical mediators of inflammation, such as bradykinin, can also cause pain by directly affecting the nerve fibers. However, because there is limited room for expansion of the PDL during acute inflammation, compression of nerve endings seems to be more significant in causing pain than the direct effects of mediators.

The results of pulp sensitivity tests are negative, because symptomatic apical periodontitis is most often associated with pulp necrosis. Percussion test is always positive and is often exquisitely painful to the patient. Therefore, it is strongly recommended that whenever symptomatic apical periodontitis is suspected, the percussion test should be performed by gently percussing the tooth with the tip of the index finger instead of using the handle of a mirror, just to confirm the involved tooth. In case the patient cannot even touch the tooth, the percussion test should be avoided. Palpation test is usually positive.

A tooth with symptomatic apical periodontitis may exhibit a normal or thickened apical PDL space. The latter

is due to the slight extrusion of the tooth in the socket to house the edema. However, there are cases in which a frank apical bone resorption is observed; this means that the symptomatic apical periodontitis developed from exacerbation of an asymptomatic lesion.

Acute Apical Abscess

Acute apical abscess is characterized by a severe inflammatory reaction to root canal infection. It has a rapid onset and is associated with pain, pus formation, and swelling of the associated soft tissues. Pain is usually spontaneous, throbbing and localized, exacerbated by chewing. Systemic involvement may occur, including fever, malaise, and regional lymphadenitis. Pain becomes severe when the purulent collection is still intraosseous or has reached the subperiosteal space. A dramatic relief in pain occurs as the purulent exudate breaks through the periosteum and reaches the supraperiosteal soft tissues.

The acute abscess is a sequel to symptomatic apical periodontitis, and is characterized by formation of a purulent inflammatory exudate in response to bacteria egressing from the apical foramen. An extraradicular infection is established in these cases. Bacterial proteolytic enzymes, especially lysosomal enzymes and oxygen radicals released by PMNs, promote tissue liquefaction and pus formation. In some cases, the host defenses do not succeed in confining the infection to the periradicular tissues and the abscess can spread to anatomical spaces of the head and neck and even give rise to life-threatening conditions. For more details, see Chapter 30.

Depending on the stage of the abscess process, intra-oral or extra-oral swelling, fluctuating or not, can be observed. Swelling is usually localized, but in some cases can become diffuse and spread to other head and neck sites to form a cellulitis. Localization of the swelling is determined by the relationship of the root apex to muscle attachments. In initial phases, no swelling is present and the clinical features resemble those of symptomatic apical periodontitis.

The results of pulp sensitivity tests are negative. Percussion yields positive results and, similar to symptomatic apical periodontitis, should be performed with caution or even avoided, as it can cause extreme pain.

If the abscess develops as an exacerbation of a longstanding asymptomatic apical periodontitis (the so-called "phoenix" abscess), radiographs reveal a radiolucent area around the root apex. If the abscess develops as a direct extension of pulp necrosis and infection, the PDL space may appear only thickened.

Chronic Apical Abscess

Chronic apical abscess is characterized by a mixed inflammatory reaction to root canal infection, with gradual onset, and associated with little or no discomfort. It may be related either with areas of exacerbation in an asymptomatic lesion or with a chronicization of an acute apical abscess.

Clinically, this condition is very similar to asymptomatic apical periodontitis, except for the presence of a sinus tract that promotes an intermittent or continuous discharge of pus. The sinus tract is usually located intra-orally, but in some cases, it can be found extraorally and even be confused with other facial skin lesions. The tract can be traced by introducing a gutta-percha point via its trajectory and then taking a radiograph, in order to determine the source of the sinus tract.

Pulp and periradicular tests yield negative results. A few cases, however, may present a mild sensitivity to percussion or palpation.

Radiographic findings for chronic apical abscess are very similar to asymptomatic apical periodontitis. However, the borders of the radiolucent lesion may not be so well defined.

Condensing Osteitis

Condensing osteitis is a diffuse radiopaque lesion, usually surrounding the root apex, and characterized by a localized reaction of the bone to low-grade inflammatory stimuli egressing from the root canal. In contrast to the other periradicular lesions, condensing osteitis is associated with a predominant active bone formation. The reason why the periradicular response is different in these cases is unknown. This condition appears radiopaque because the localized excessive production of bone around the root apex results in narrowing of the medullar bone spaces (and not an increase in calcification). Condensing osteitis is more commonly found in association with mandibular molars with pulp necrosis or asymptomatic pulpitis. Bone can be remodeled and return to radiographic normalcy following successful root canal treatment.

HISTOPATHOLOGIC CLASSIFICATION OF PERIRADICULAR LESIONS

The study of periradicular lesions using conventional light microscopy, according to contemporary agreed directives and defined criteria,[10] provides valuable information on the overall morphology of the periradicular pathologic tissue and its relationship with the root apex and apical root canal system. The presence and prevalence of the various types of inflammatory cells, identified by their classical morphology, as well as the occurrence of epithelium and collagen bundles can be ascertained, together with the structures originated from the interaction between these different tissues. This information is of great value because it provides spatial location, for example, of the epithelial structures and bacteria. However, this approach does not allow the study of the immunobiology of the periradicular host response. Consequently, immunohistochemical techniques have been employed to characterize the various immunocompetent cells present (T and B lymphocytes, macrophages, dendritic cells, plasma cells, and their subsets) and inflammatory mediators in periradicular lesions.[37-45]

When dealing with the histomorphological aspects of apical periodontitis lesions, one should have in mind the cross-sectional nature of the analysis, that is, we are observing a

specific moment of the lesion evolution. Depending on the microbial challenge and other factors, periradicular lesions may exhibit different cellular and molecular composition. This may change over time, in a dynamic host-pathogen interaction.

The methodology adopted in histological studies of periradicular lesions deserves special consideration. Many studies published in the past were based on inadequate biopsies and less than ideal histologic methodologies.[46,47] It is currently recognized that the correct histopathologic diagnosis of periradicular lesions can only be made when two conditions are met: *(1)* the biopsy sample includes the lesion and the root apex in their original spatial relationship and *(2)* a serial sectioning approach is undertaken.[11-12,48]

This may imply that adequate biopsies are only obtained from tooth extraction, in which the lesion remained attached to the root apex, or from periradicular surgery, in which block sections containing the apex, lesion, and possibly the surrounding bone are available. Fragments of periradicular pathologic tissue or even the entire lesion, removed by curettage from the alveolus, are not considered appropriate for a precise diagnosis of the type of apical periodontitis. Moreover, sections taken randomly through the biopsy specimen are not acceptable. The specimen should be sectioned completely from pole to pole. Serial sectioning approach is currently considered essential for proper histopathologic diagnosis.[10,48] This is because sections taken at different points of the lesion may differ considerably in terms of histologic patterns. For example, the section shown in Figure 6-5A would lead to a diagnosis of granuloma, while the sections taken at some distance and shown in Figure 6-5C and D show an abscess with microcavities and a massive concentration of PMNs in the center of the lesion (Figure 6-5H and I).

In one of the first reports on extracted teeth with periradicular lesions attached, Thoma[49] studied the microscopic appearance of periradicular lesions and described the possible presence of epithelium (including cyst formation), and suppuration in dental granulomas. His classification has not changed significantly over the years. The histopathologic classification used in this chapter is based on the presence or absence of epithelial cells and the distribution and type of inflammatory cells in the periradicular lesion.

Apical Abscess

Abscess is a collection of pus in a cavity formed by tissue liquefaction. Acute apical abscess can develop without previous chronic inflammation or may result from exacerbation of a previously chronic asymptomatic lesion. Abscess lesions are formed by an accumulation of PMNs, usually delimited by a granulomatous tissue composed mainly of lymphocytes, plasma cells, and macrophages. This pattern is mainly observed when the abscess develops as an exacerbation of granuloma (Figure 6-5A-I). Based on the presence or absence of epithelial strands, abscesses can further be categorized as epithelialized (Figure 6-10) or non-epithelialized (Figures 6-5 and 6-7). The epithelialized abscess could have originated from an exacerbation of an epithelialized granuloma (described below). In cases with severe clinical symptoms, histomicrobial analysis usually reveals microbial cells in the periradicular tissues that can be present both in the extracellular compartment and engulfed by PMNs (Figure 6-7F). The latter represents frank phagocytic activity. Several microcavities containing cellular debris and PMNs can be observed throughout the lesion body (Figures 6-5H, I and 6-7D-F), sometimes affecting the epithelial strands (Figure 6-10D, E). The peripheral area of the lesion may be composed of bundles of collagen fibers, indicating exacerbation of a previously chronic lesion (Figures 6-5E, H, L and 6-7D).

Apical Granuloma

Granuloma is characterized by chronic inflammation, and consists of elements of granulation tissue infiltrated by immune defense cells. Chronic inflammatory cells are observed in varying numbers and distribution throughout the granuloma tissue. They include macrophages, lymphocytes, plasma cells, foam cells, and multinucleated foreign-body cells (Figure 6-4I). At the periphery of the lesion, the inflammatory tissue usually gives way to a fibrous tissue, formed essentially by collagen, with few, if any, inflammatory cells (Figure 6-4I).

Apical granuloma can be epithelialized (Figure 6-8) and non-epithelialized (Figures 6-4 and 6-6). When present, epithelial cells can form strands that are randomly scattered (Figure 6-8C & D) and may be infiltrated by PMNs and chronic inflammatory cells (Figures 6-10E and 6-11A-C). This is a peculiarity of the apical granuloma, as other granulomas elsewhere in the body rarely contains PMNs in the extravascular location. The epithelium may exhibit extensive proliferation at the area adjacent to the apical foramen and even invaginate into the apical canal (Figures 6-8B & C and 6-10D). The epithelium may also form ramified structures that enclose islands of granulomatous tissue containing lymphocytes, plasma cells, and a rich circulatory network (Figures 6-11A-C and 6-12A, B).

Granuloma is the most common histopathologic form of apical periodontitis. Bhaskar[50] examined 2,308 specimens of periradicular lesions and found that 48% were classified as granuloma, 42% as cyst, and 10% represented other pathologic forms. Nair et al.[48] evaluated 256 apical periodontitis lesions and reported that 50% were granulomas, 35% abscesses, and 15% cysts.

Apical Cyst

Apical cyst is an inflammatory lesion of endodontic origin that is characterized by the presence of a cavity lined by epithelial cells. The epithelial lining is usually of the stratified squamous type, but in a few cases, a ciliated columnar epithelium can be observed (see below). According to the

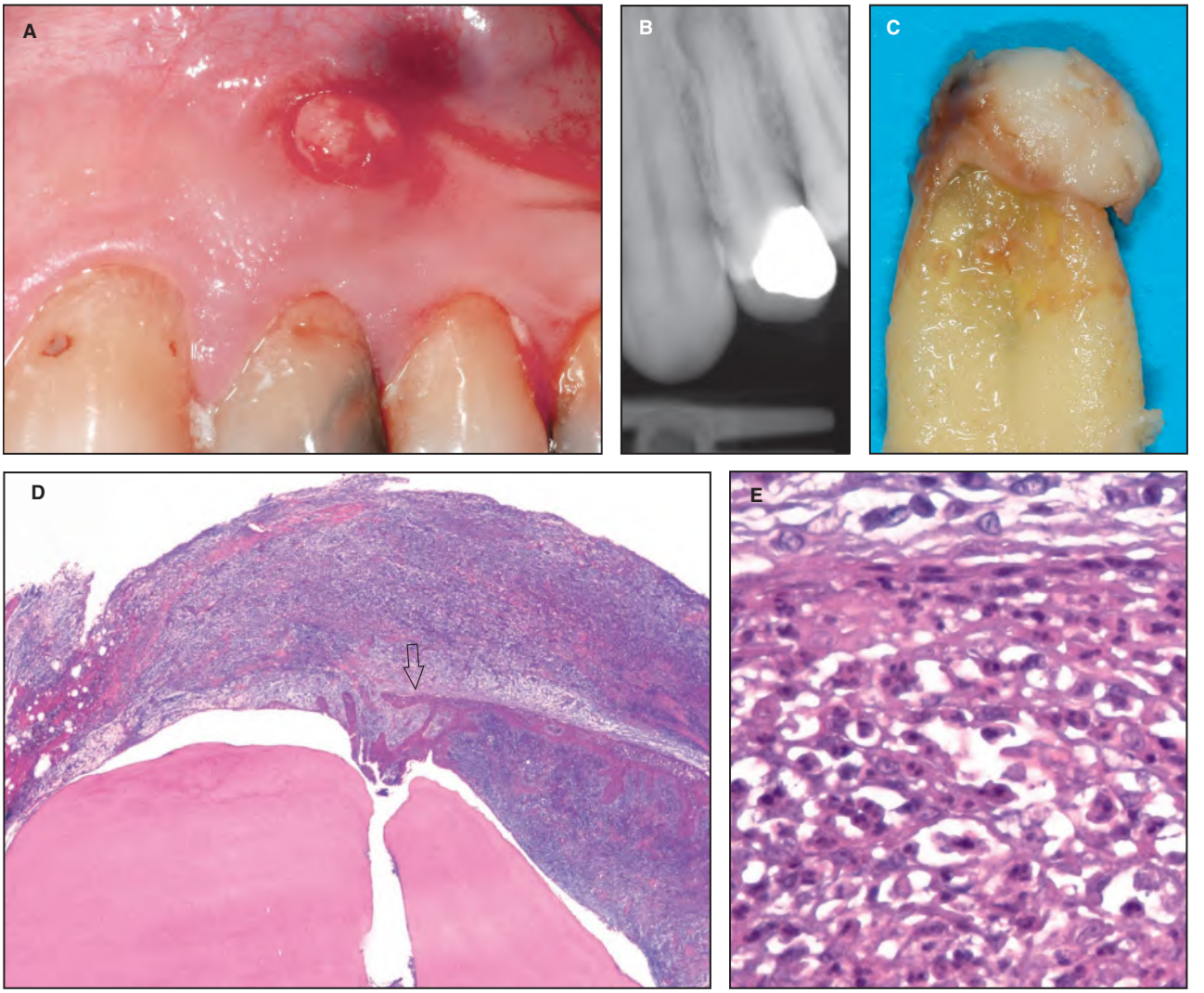

FIGURE 6-10 ***A***. Sinus tract in the buccal gingiva apically to the maxillary first premolar. ***B***. Radiograph of the tooth showing apical radiolucency. ***C***. Following extraction, a periapical lesion remained attached to the root tip. ***D***. Section cut on a bucco-lingual plane encompassing the apical foramen. Proliferating epithelial strands forming an epithelial "plug" at the foramen (Hematoxylin and eosin, original magnification ×25). ***E***. High power view from the area of the epithelium indicated by arrow in D. The epithelial tissue is heavily infiltrated by PMNs (×400). The histologic diagnosis was "epithelialized abscess."

spatial location of the cavity, the cyst can be classified as true or pocket (bay).[48,51]

The true apical cyst is a periradicular lesion with a distinct pathologic cavity completely enclosed by the epithelial lining, without any communication with the root canal system (Figures 6-13 and 6-14). The pocket apical cyst is a sack-shaped apical inflammatory lesion, with the epithelium-lined cystic cavity in direct contact with the root canal system via apical foramen or lateral foramina. For a precise classification of a cyst as true or pocket, serial sectioning analysis of the lesion attached to the root apex is mandatory. For instance, according to the cavity morphology in Figure 6-15A, the lesion would have been diagnosed as a true cyst. However, hundreds of sections away revealed a clear communication between the cyst lumen and the root canal (Figure 6-15D) that is compatible with diagnosis of pocket cyst.

Histologically, the true cyst presents an epithelium-lined cavity separated from the root canal system. The cavity appears half-empty or containing necrotic tissue and clusters of cells in various stages of degradation (Figure 6-13A-D). In some instances, cholesterol crystals are detected (Figure 6-14). These crystals are usually dissolved by chemicals used in histologic processing. Therefore, they appear as void spaces with a typical pointed appearance of "broken glass" or "pine needles" (Figure 6-14A, B). Cholesterol crystals are often surrounded by a dense accumulation of PMNs (Figure 6-14C, D). Varying portions of the cyst lumen may be occupied by macrophages with foamy cytoplasm (foam cells) intermixed with PMNs (Figure 6-14E). Depending on the location, the epithelial lining of the cyst cavity may vary significantly in thickness and is often infiltrated by PMNs (Figure 6-13C, D). The epithelial lining may show signs of

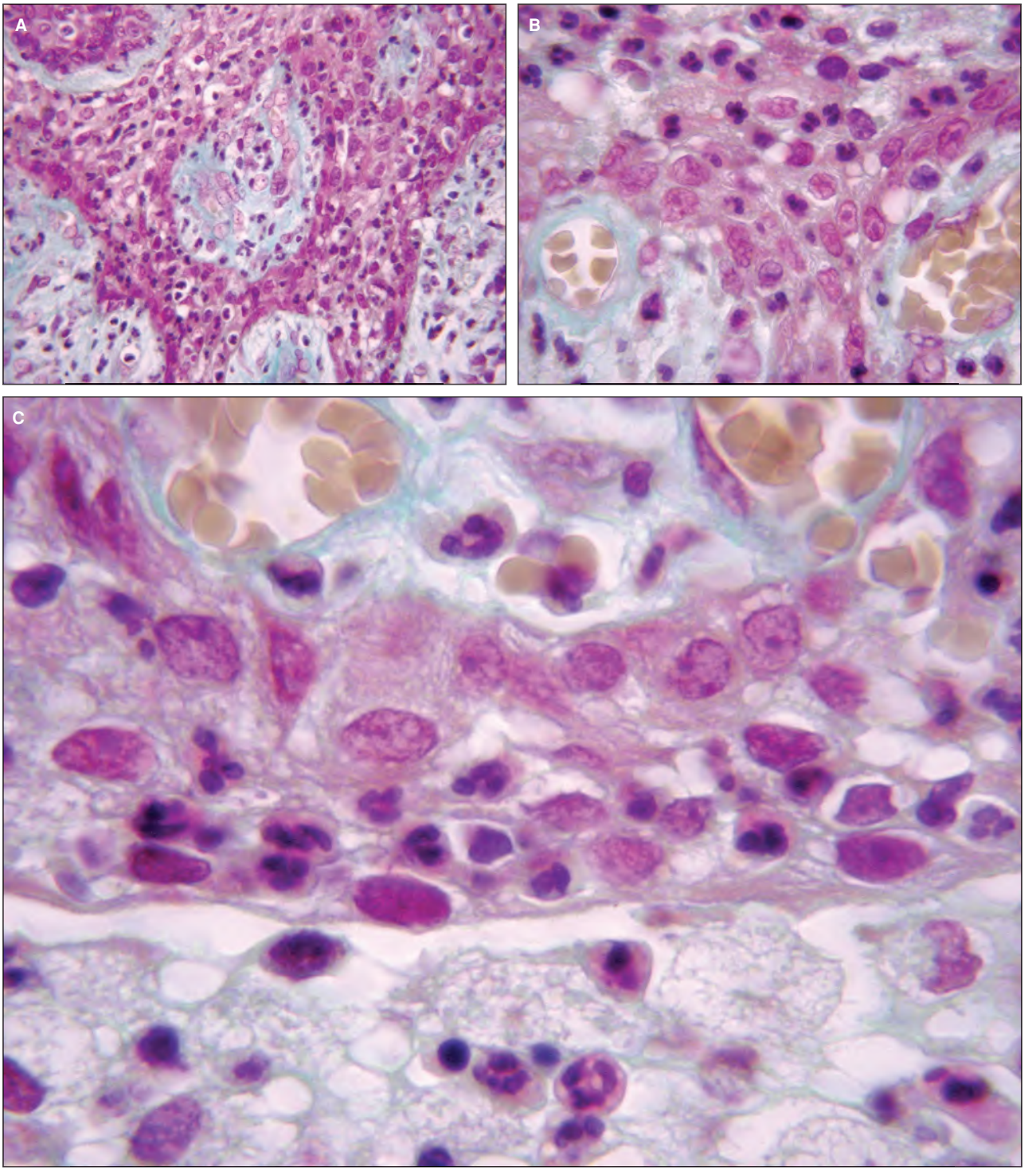

FIGURE 6-11 High power views of epithelial strands present in an epithelialized granuloma. ***A***. The epithelium surrounds cores of connective tissue infiltrated with inflammatory cells (Masson trichrome, original magnification ×400). ***B*** and ***C***. The strands appear infiltrated by PMNs. Note the hyperaemic cross cut vessels in the adjacent connective tissue (×1000).

exfoliation near the cyst lumen, with cells assuming a blurred appearance and indistinct nuclei. In the periphery of the true cyst, bundles of collagen fibers form a pseudo-capsular structure, separating the lesion from bone (Figure 6-13A). Chronic inflammatory cells may be observed, in moderate concentrations, between the bundles of collagen fibers. More than one cyst cavity may be present in the same lesion.[12]

The lesions classified as pocket cysts show a cystic cavity lined by epithelial cells forming a sac-like structure, separating the foramen from the remainder of the lesion

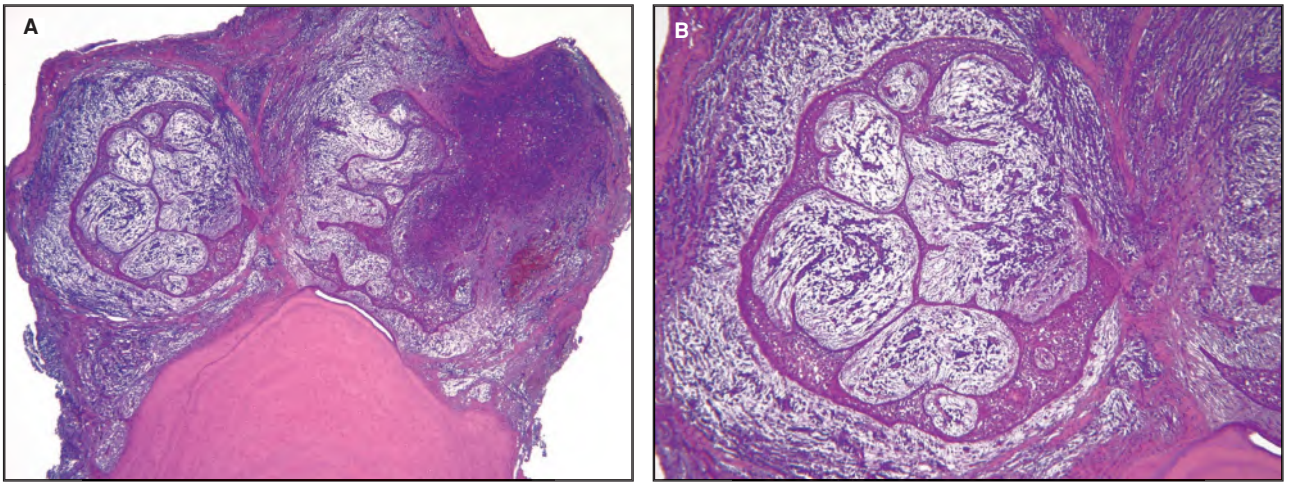

FIGURE 6-12 Maxillary premolar extracted with a large apical periodontitis lesion attached. ***A***. The lesion appears composed of two portions, separated by connective tissue. Both parts show epithelial strands surrounding islands of inflamed connective tissue, but no cavities can be seen. Despite the advanced proliferation of the epithelium, in the absence of cavities, the diagnosis is "epithelialized granuloma" (Hematoxylin and eosin, original magnification ×16). ***B***. Magnification of the left portion of the lesion (×50).

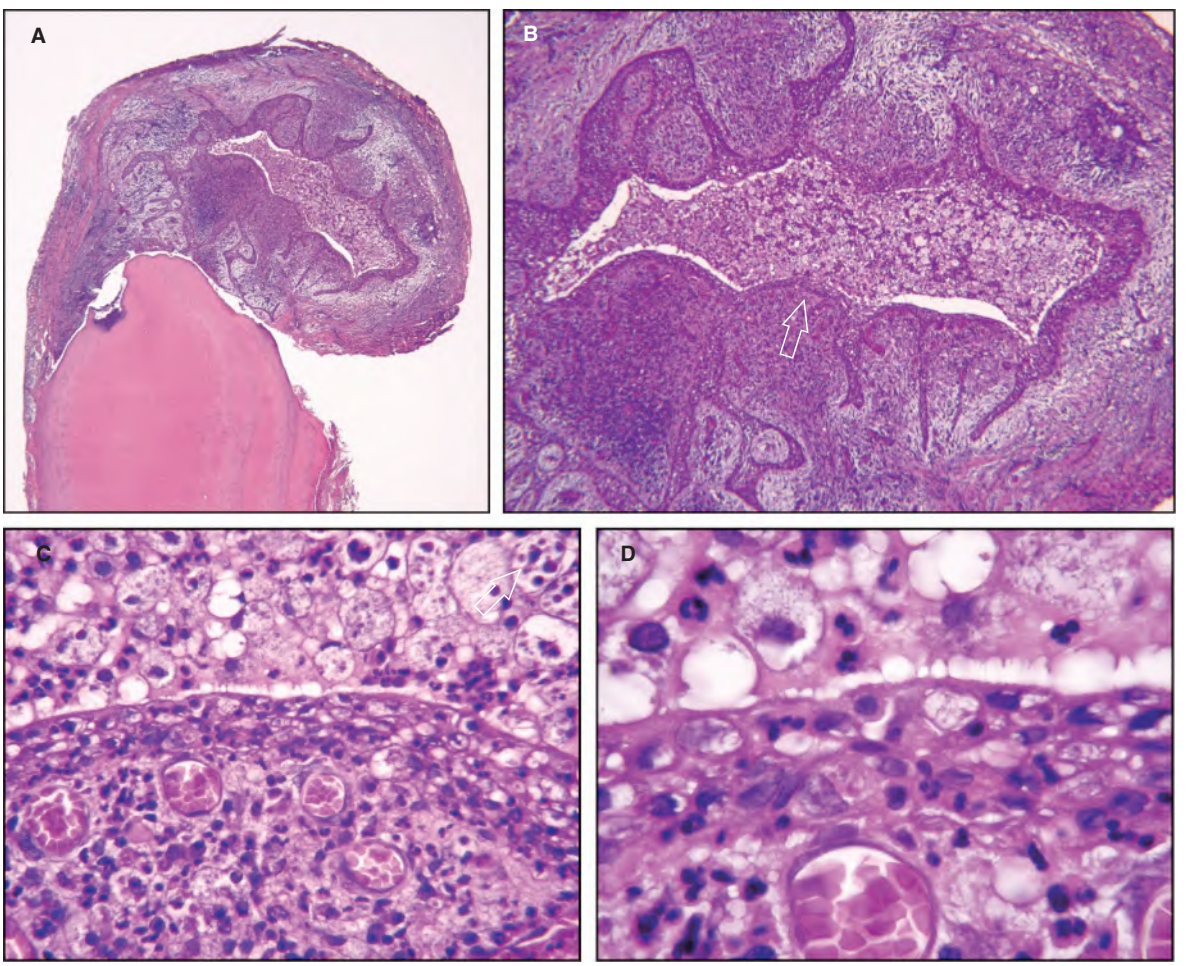

FIGURE 6-13 Distobuccal root of a maxillary molar. ***A***. The lesion shows a central cavity completely lined by epithelium, not in communication with the apical foramen. Serial sections confirmed the absence of communication between the cyst lumen and the root canal space, leading to the diagnosis of "true cyst" (Hematoxylin and eosin, original magnification ×25). ***B***. Magnification of the cyst cavity. Its lumen contains necrotic debris, with microcavities, foam cells, and PMNs (×50). ***C*** and ***D***. Progressive magnifications of the area of the cyst wall indicated by arrow in B. Necrotic debris, foam cells and PMNs in the lumen; hyperaemic vessels and PMNs in the underlying connective tissue (×400 and ×1000).

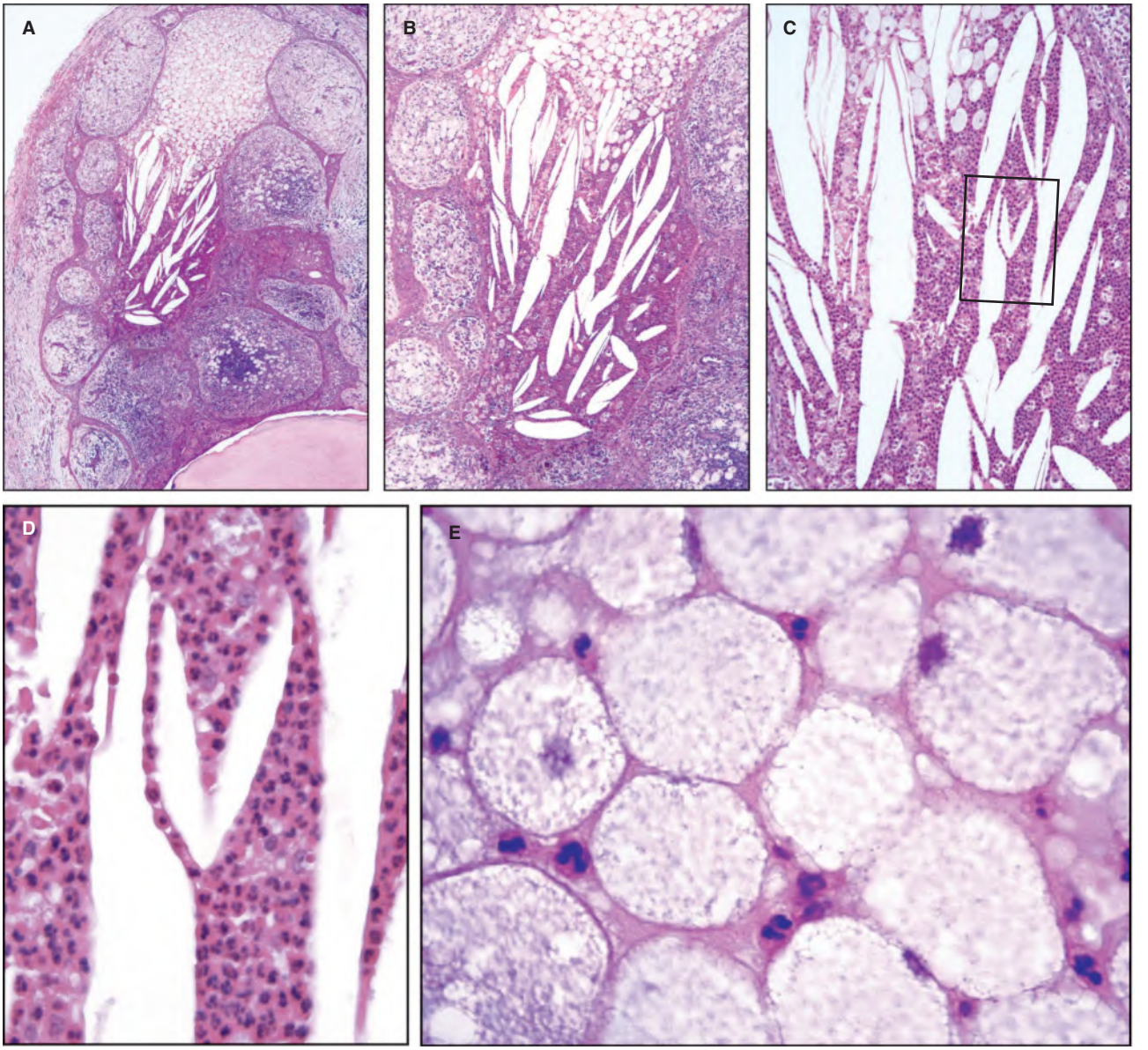

FIGURE 6-14 Maxillary premolar extracted with a lesion attached. ***A***. Serial sections demonstrated that the lesion was a "true cyst." In the upper portion, the cyst cavity appears filled with foam cells, while empty spaces with the appearance of "broken glasses," typical of cholesterol crystals, are present in the lower part (Hematoxylin and eosin, original magnification ×25). ***B***. Detail from the lower part of the cyst content (×100). ***C*** and ***D***. Progressive magnifications show empty spaces surrounded by a severe concentration of PMNs (×100 and ×400). E. High power view from the upper portion of the cyst cavity. Dead or dying lipid-filled foamy macrophages and PMNs (×1000).

(Figure 6-15). The epithelium forming the cystic wall may show signs of desquamation and is infiltrated by PMNs. Abundant debris are commonly seen in the cyst lumen. Similar to other chronic lesions, collagen bundles are seen at the periphery.

Hypothetical Mechanisms to Explain Cyst Formation

The epithelium forming cysts of endodontic origin is believed to be derived from the Hertwig's epithelial root sheath.[52] This sheath originates from the fusion of the external and internal dental epithelium to form the cervical loop that invaginates into the underlying connective tissue of the dental organ. The Hertwig's root sheath guides the deposition of root dentin, after which it disintegrates, leaving behind nests of epithelial cells in the PDL. These are known as epithelial cell rests of Malassez (ERM), and considered to remain in a resting stage.[53,54] The exact function of ERM in normal PDL is unknown.[55] However, if stimulated by specific extracellular signals, they have the potential to undergo cell division. In apical periodontitis lesions, epithelial cell rests proliferate in three dimensions and forming strands that have the tendency to form a three-dimensional network.

Structurally, ERM are surrounded by the basal lamina and appear as small agglomerates of cells with large nuclei and distinct nucleoli, closer to the cementum than to the bone

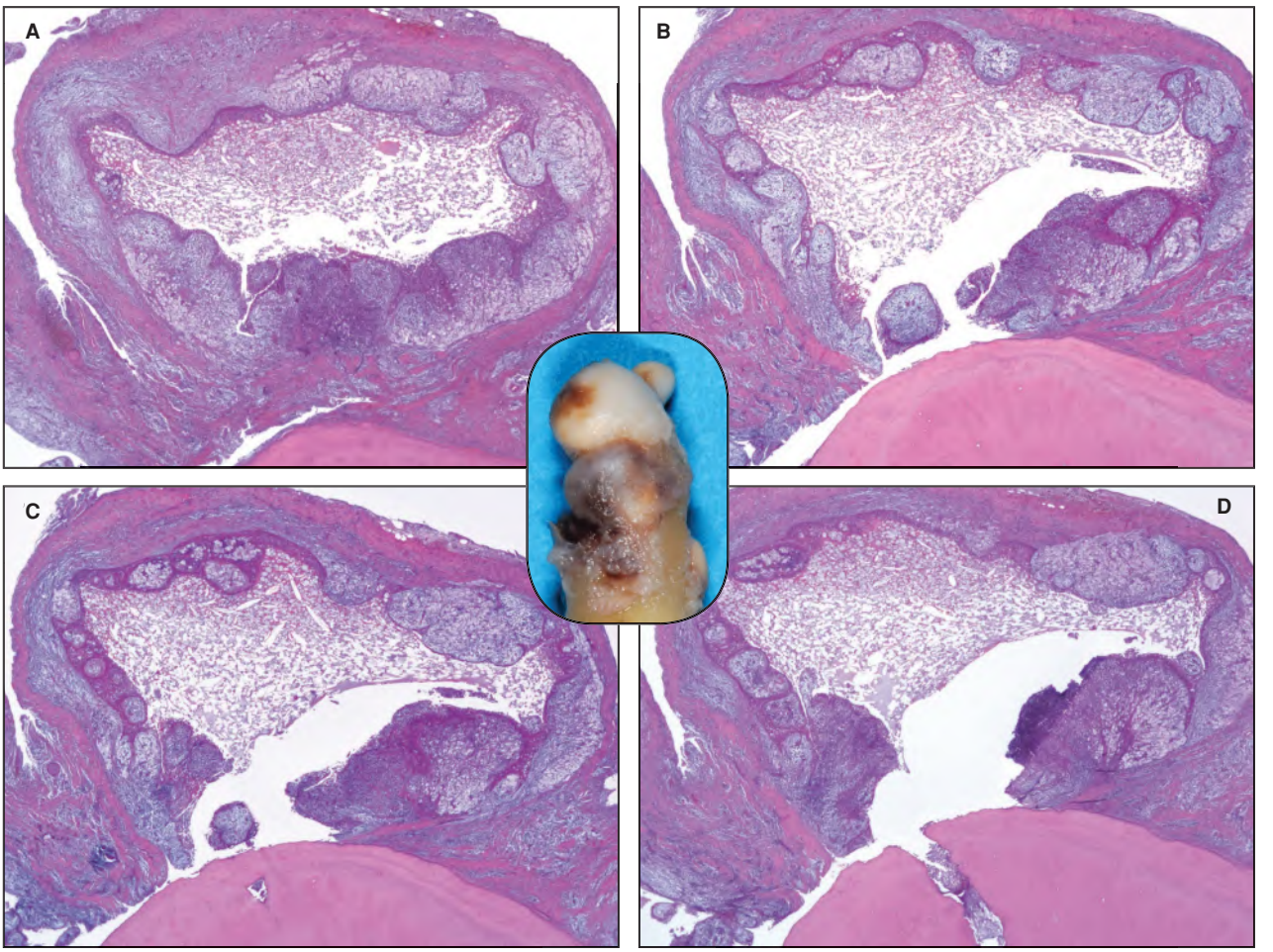

FIGURE 6-15 Series of sections taken from a maxillary third molar extracted with an apical periodontitis lesion attached. Section were distant approximately 50 sections from each other. ***A***. A cavity, completely surrounded by epithelium, may lead to a histologic diagnosis of a "true cyst." ***B–D***. Serial section procedure demonstrates a clear relationship between the cyst lumen and the root canal space, confirming a diagnosis of a "pocket cyst" (Hematoxylin and eosin, original magnification ×16).

surface (Figure 6-16A–C). Sometimes, they can be seen as large agglomerates of cells (Figure 6-17A, B).

The development of apical cysts can be regarded as a late stage of the host defense against root canal infection. Every apical cyst is supposed to have its origin in an apical granuloma, but there are no supporting data that every granuloma will invariably be transformed into a cyst. One might think of the epithelialized granuloma as an intermediary stage in the granuloma-to-cyst. However, there is no evidence that epithelial strands proliferating in the inflamed periradicular tissues will always form an apical cyst. Nair et al.[48] found that 52% of the periradicular lesions examined showed epithelial proliferation, but only 15% of them were classified as cysts.

Several theories have been suggested to explain cyst formation[55]:

1. ***Breakdown theory***. This theory states that epithelial islands continue to grow in a three-dimensional spherical mass, responding to inflammatory stimuli. The central cells of the epithelial mass will then drift away from their original source of nutrients and undergo necrosis and liquefaction degeneration. The microcavities would coalesce to form a cystic cavity.[52] This theory has been questioned because the epithelial cells form strands in the apical granuloma that are frequently surrounded by a vascularized connective tissue.
2. ***Abscess theory***. According to this theory, a cyst is formed after epithelial cells started to proliferate to cover a preexisting abscess cavity formed in the connective tissue.[56,57] This is due to the intrinsic tendency of epithelial cells to cover exposed surfaces of connective tissue.[58,59] To test the hypothesis that radicular cysts develop via the "abscess pathway," Nair et al.[60] attempted to experimentally induce inflammatory cysts in rats. Inoculations of epithelial cells, followed by a *Fusobacterium nucleatum* injection into tissue cages implanted in rats' backs, 7 days later, resulted in development of inflammatory cysts in two of the 16 cages. These two cages contained a total of four cystic sites. They concluded that inflammatory cysts were induced by acute inflammatory foci initiated by bacterial injection that were further enclosed by a proliferating epithelium.

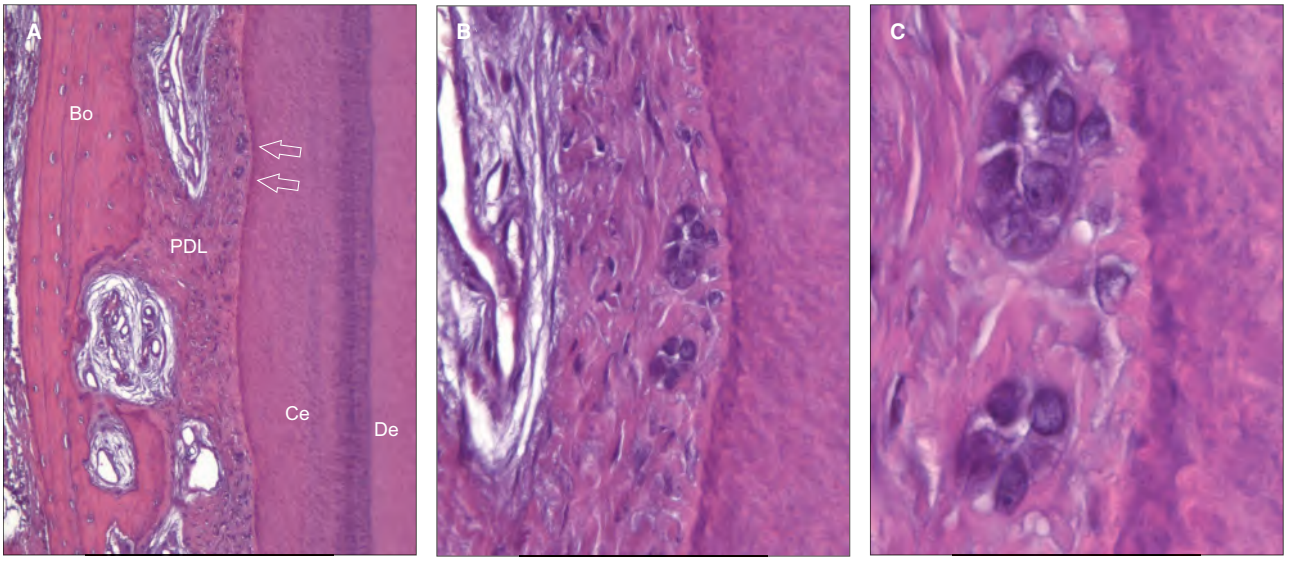

FIGURE 6-16 Histology of normal periodontal ligament. ***A***. From right to left are dentin (De), cementum (Ce), periodontal ligament (PDL), and alveolar bone (Bo). Arrows indicate two small groups of cells in the PDL, close to cementum (Hematoxylin and eosin, original magnification ×100). ***B*** and ***C***. Progressive magnifications show that these structures are nests of epithelial cell rests of Malassez surrounded by basement membrane (×400 and ×1000).

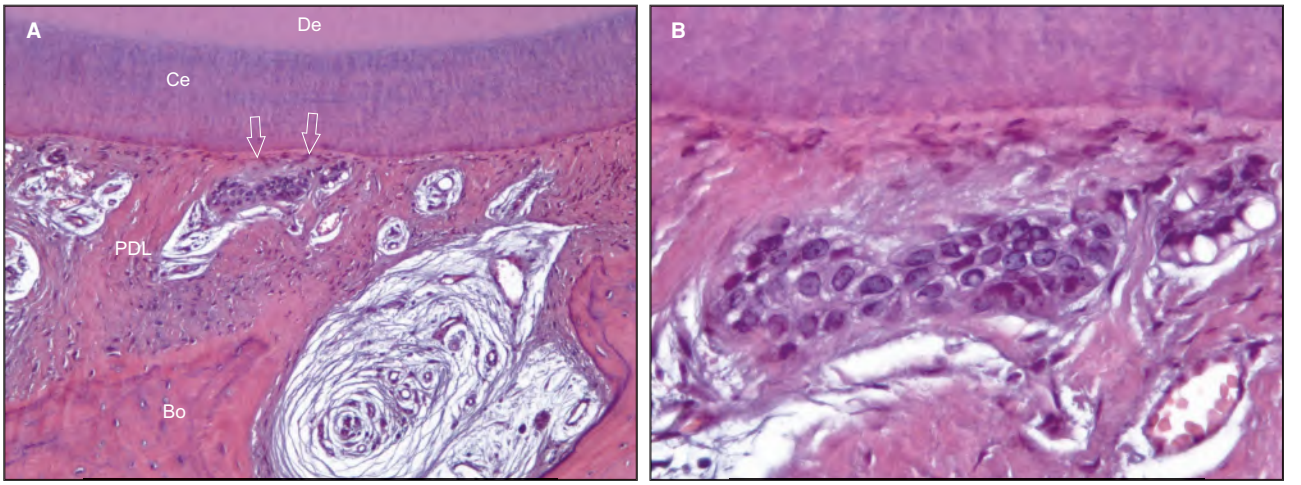

FIGURE 6-17 ***A*** and ***B***. Histology of normal periodontal ligament displaying a large aggregate of epithelial cells of Malassez (De, dentin; Ce, cementum; PDL, periodontal ligament; Bo, alveolar bone) (Hematoxylin and eosin, original magnification ×100 and ×400).

3. ***Immunologic theory***. This theory assumes that the development of cystic cavities in the proliferating epithelium is mediated by an immune response against activated epithelial cell rests of Malassez that become antigenic as a result of the abnormal growth.[61]
4. ***Fusion theory***. This theory[55] suggests that formation of the inflammatory apical cyst is caused by fusion of proliferating epithelial strands in all directions, to form a three-dimensional ball mass. The connective tissue trapped in the epithelial mass would undergo gradual degeneration as a result of the decrease in vascular support, giving rise to a cystic cavity.

Whatever be the process leading to development of cyst cavities, the phases for apical cyst formation are chronologically divided into three stages[62]:

1. Stage 1: The "dormant" cell-rests of Malassez start to proliferate stimulated by chemical inflammatory mediators produced and released in the lesion.
2. Stage 2: The cavity covered by epithelium is formed via one of the mechanisms discussed above.
3. Stage 3: The cyst expands at the expense of bone. Expansion of the cystic lesion may be related to an increase in the osmotic pressure generated by the decomposition of

inflammatory cells accumulating in the cyst lumen. This would substantially increase the number of macromolecules within the cyst lumen. Supporting this hypothesis is the fact that decompression (marsupialization) procedures usually succeed in reducing the lesion size. On the other hand, the observation that pocket cysts opened to the canal lumen also show a tendency to increase in volume might question the possibility that increase in osmotic pressure is responsible for lesion growth. Production of pro-resorptive inflammatory mediators in the periradicular tissues is the main cause of formation and growth of periradicular lesions, including granulomas. Therefore, it could be the main factor involved in cyst expansion.

Can Cystic Lesions Heal after Conventional Root Canal Treatment?

Some clinicians claim that cysts do not heal following root canal treatment. Based on morphological observations, some authors[63-65] speculated that true cysts are refractory to treatment because they may behave like a self-sustaining pathologic entity, independent of the influence of endodontic infection.

Lin et al.[66] argued against this notion. Inflammatory cells are always seen in the epithelium lining and/or fibrous connective tissue capsule of apical true cysts (Figures 6-8D, 6-10E, 6-11A-C, 6-13C, D).[12,48] This is a strong indication that irritants are present either in the root canal system or in the cyst lumen. It is also highly unlikely that the epithelial cells of true cysts, but not pocket cysts, can suddenly change the patterns of gene expression and start behaving like self-sustaining neoplastic cells.[66] However, this issue has not been fully elucidated yet.

To prove this theory, one should be able to distinguish between the types of cysts before nonsurgical endodontic treatment is performed. So far, no clinical or imaging diagnostic method can reliably differentiate granulomas from cysts, let alone true cysts from pocket cysts. The gold standard for diagnosis of periradicular lesions is still histologic examination, and this can only be performed in a cross-sectional approach.

A disease that persists, after cessation of stimuli that evoked its development, is regarded as a neoplasm.[67] Common examples are tobacco chewing and oral cancer, and smoking and lung cancer. Neoplasm is related to genetic damage of cells, including mutation of proto-oncogenes or loss of tumor suppressor genes to restrain cell growth and proliferation.[68,69] Neoplastic cells undergo spontaneous division, usually supported by autocrine secretion of growth factors, and are self-sustaining and immortal.[68,69] There is no evidence that epithelial cells, initially stimulated to grow by inflammatory stimuli in apical true cysts, become immortal and self-sustaining.

A disease caused by microbial infection, including true and/or pocket cysts, is expected to heal as long as the causative agents are eliminated or controlled. Exceptions include cases of chronic bacterial infections contributing to the development of malignancies, such as *Helicobacter pylori* infection and gastric cancer.[70-72] However, endodontic bacteria have not been shown to make epithelial cells behave like self-sustaining neoplastic cells.

Considering that epithelial cells from pocket cysts can undergo apoptosis in response to removal of the growth and survival stimuli following proper root canal treatment,[55] there is no reason to believe that the epithelial cells from true cysts would behave differently. One study[72] reported results of root canal treatment done in 42 teeth with large periradicular lesions regarded as cysts on radiographic basis. The canals were instrumented, dressed with calcium hydroxide, and filled. Complete healing was observed in about 75% of the cases. It must be pointed out that the examined cases may not have necessarily represented cystic lesions, since the study design does not permit histologic examination to confirm the diagnosis. Considering that all lesions were indeed cysts, one can conclude that most cysts heal after treatment. Non-healing lesions, irrespective of their histologic diagnosis, are virtually always a result of persistent intraradicular infection or extraradicular infection. For more details, see Chapter 3.

Based on current knowledge, there is no reason to believe that the two morphological varieties of cysts respond differently to root canal treatment. If one has a greater potential to present a significant problem, it will be the pocket cyst. Because the lumen of pocket cysts is in direct contact with the root canal system, it is possible that these cysts are more prone to become infected after direct advance of microorganisms from the root canal to the cyst cavity. Therein, microorganisms are beyond the effects of non-surgical root canal treatment and may be difficult to eliminate by the host defenses.[25] Thus, pocket cysts would have more plausible reasons to become recalcitrant than true cysts, because bacteria in the cyst lumen may provide continuous stimuli for growth and survival of epithelial cells.

Another problem related with pocket cysts is that the fluid present in the cyst lumen may seep into the canal after access opening, or during instrumentation, rendering conclusion of treatment difficult. In some cases with persistent drainage of the cyst fluid into the canal, application of a calcium hydroxide dressing or an apical plug of calcium hydroxide may help achieve a dry canal.

In addition to true cysts, cholesterol crystals have been suggested as another possible non-microbial cause of post-treatment apical periodontitis.[65,73,74] Cholesterol crystals can be formed locally in areas of tissue damage or necrosis caused by release of cholesterol from disintegrated cell membranes.[68] Because cholesterol is insoluble in water, it crystallizes into thin, flat rhomboid plates.[75] Cholesterol crystals occur in the cyst lumen, lining epithelium, and/or fibrous connective tissue capsule. Histologically, macrophages and giant cells are always found in association with cholesterol crystals in apical periodontitis lesions.

A study in guinea pigs evaluated the subcutaneous tissue response to implantation of pure cholesterol crystals and reported that macrophages and multinucleated giant

cells were unable to eliminate the cholesterol crystals over a period of 8 months.[74] It was extrapolated that accumulation of cholesterol crystals in periradicular lesions may prevent healing following endodontic treatment, and given the extraradicular location of the crystals, even retreatment would not succeed. However, it is unknown whether the same response occurs in humans. Actually, the medical literature provides evidence that activated macrophages can deal with cholesterol crystals.[66] Similar to true cysts, there is no evidence showing that cholesterol crystals can prevent periradicular wound healing after nonsurgical root canal therapy. Because the prevalence of cholesterol crystals in periradicular lesions has been shown to range from 18% to 44%,[75,76] some might infer that the same proportion of lesions would not heal following root canal treatment, if the cholesterol crystals had the ability to sustain inflammation by themselves. However, this does not match the high success rate of endodontic treatment[77] and the reported frequent causes of treatment failure.[78] Nevertheless, cholesterol crystals are the product and not the cause of apical periodontitis. However, it is conceivable that the occurrence of these crystals may delay the healing process following nonsurgical endodontic treatment.

Ciliated Epithelium in Apical Cyst Lesions

The epithelium commonly present in apical cysts is of the stratified squamous type. However, in some cases, a ciliated pluriserial (or pseudostratified) columnar epithelium, with features of the epithelium covering the respiratory airways, is observed.[79-84] The term "pseudostratified" indicates an epithelium consisting of a single layer of cells of varying heights and disposition; these epithelial cells rest on the basal membrane, but only some of them reach the free surface. This epithelium type presents with cilia that are moving filaments covering the cell surface. In the respiratory system, the cilia move regularly and rhythmically to induce currents in the fluid that bathes the cell surfaces, facilitating the removal of mucus and harmful particles.

The presence of the goblet cell is another feature of the respiratory epithelium. This cell is the only example of single-cell gland in the human body and is found inserted between the epithelial cells. Its main function is to secrete mucin, a protein-polysaccharide that, along with water, forming the mucus.

Occurrence of respiratory epithelium in apical cysts is not rare. Nair et al.[83] observed three ciliated epithelium-lined cysts among 39 cysts. These three cases were associated with maxillary premolars. Ricucci et al.[84] found four ciliated epithelium-lined cysts among 49 cysts. These four cases were all associated with maxillary molars. The proximity of the affected roots to the maxillary sinus floor was radiographically evident in three of them. These studies indicated that approximately 8% of the apical cysts exhibited ciliated epithelium.

Although this type of epithelium has been reported to occur also in cysts of the mandible,[79,82] findings from these two studies indicate that ciliated epithelium is most frequently associated with cysts affecting maxillary posterior teeth. In the posterior maxilla, presence of ciliated epithelium may be due to pathologic exposure of the maxillary sinus by the apical cyst. Radiographically, the apical cyst is seen in close proximity to, or extending into, the sinus. In some cases, a sinus tract may establish a contact between the cyst lumen and the maxillary sinus. This may cause the ciliated epithelium to merge with the squamous epithelial lining of the cyst. Indeed, an evident sinus tract crossing the cyst wall and connecting the cyst lumen with the external surface of the lesion (likely the maxillary sinus) was shown by Ricucci et al.[84] To our knowledge, evidence for this had been previously provided only by Kronfeld in 1939.[85]

An example of ciliated epithelium in an apical cyst is illustrated in Figure 6-18. It is a maxillary right first molar of a 50-year-old woman with the diagnosis of necrotic pulp. A radiolucency with indistinct margins was present around the mesiobuccal root apex proximal to the maxillary sinus floor (Figure 6-18A). To the patient's request, the tooth was extracted and the lesion remained attached to the mesiobuccal root. Histologic analysis revealed a cavity in the tissue mass and an apical extension with a sinus tract completely lined by ciliated epithelium (Figure 6-18B–E).

Prosoplasia is a pathologic phenomenon that can take place in the cyst lining epithelium and may help explain the rare occurrence of ciliated epithelium in cysts of the mandible and in maxillary teeth not in close proximity to the sinus. Prosoplasia is a progressive transformation of cells to either a higher level of function or organization. Accordingly, a simple epithelium would upgrade its differentiation from simple and non-functional epithelium to a higher or functional epithelium, such as those that contain secretory "mucus-cells."[86] Therefore, the presence of ciliated epithelium in apical cysts can be explained by either sinus involvement (in the posterior maxilla) or prosoplasia (other regions).

MORPHOLOGIC CHANGES OF THE APICAL ROOT STRUCTURE IN APICAL PERIODONTITIS LESIONS

Morphologic changes of the apical root structure may be associated with pulp and periradicular inflammation. The most common phenomenon is resorption. Although much less frequent, hypercementosis can also be observed.

Root Resorption in Apical Periodontitis

Root resorption can be defined as the process of removal of cementum and dentin by multinucleated clastic cells often referred to as odontoclasts.[87] This process is considered physiologic in shedding of deciduous teeth, but it is pathologic when it manifests as a sequel to microbial infection of the root canal space and consequent periapical inflammation. Pathologic root resorption is also caused by traumatic injuries to the teeth, neoplastic processes in the jaws and dental

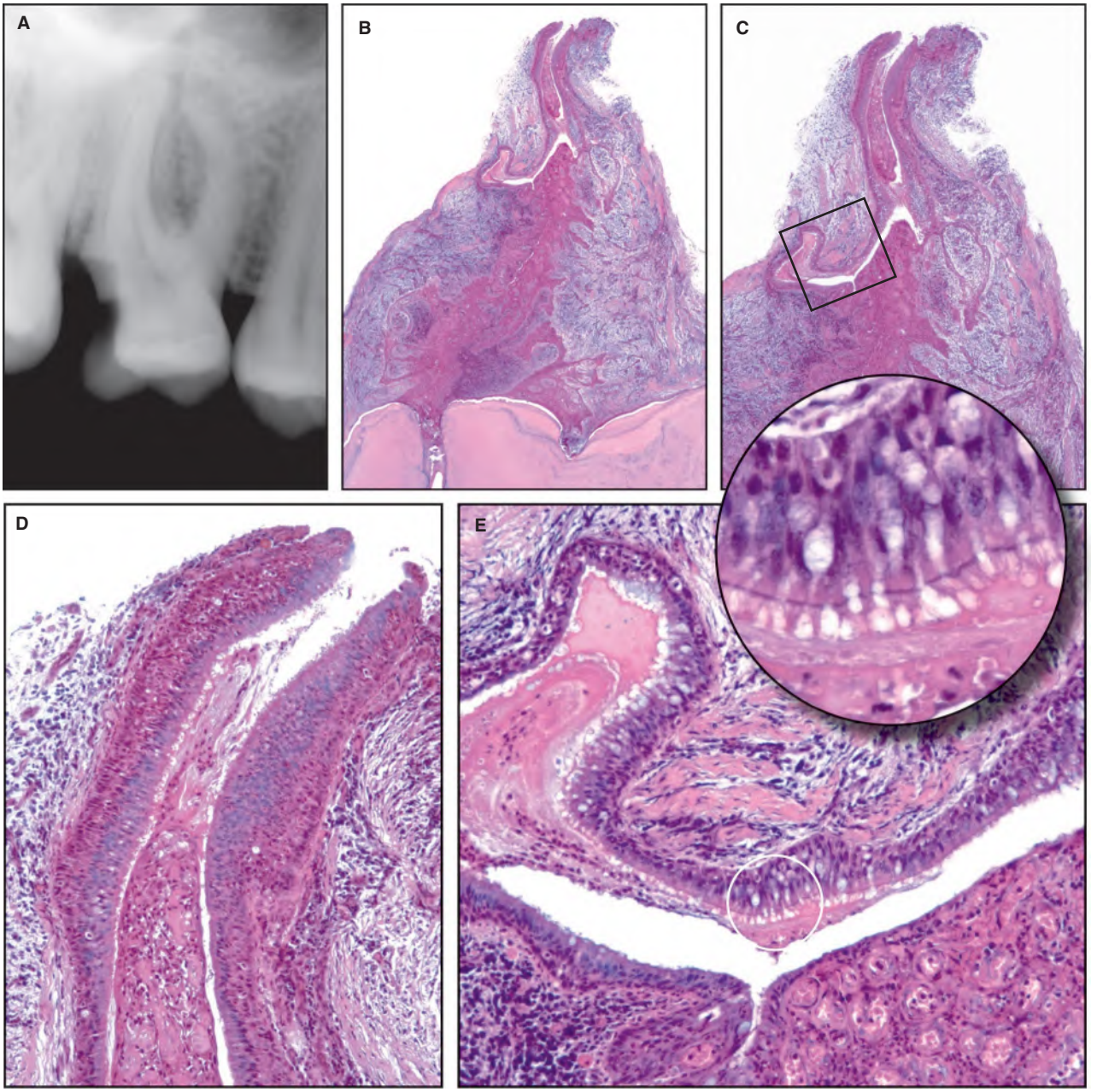

FIGURE 6-18 ***A***. Diagnostic radiograph. The radiolucency around the mesiobuccal root apex is proximal to the maxillary sinus floor. ***B*** and ***C***. Progressive magnifications of the periapical pathologic tissue that remained adhered to the apex of the mesiobuccal root at extraction. A cavity is present in the tissue mass (Hematoxylin and eosin, original magnification ×16 and ×25). ***D***. Detail of the apical extension of the lesion in C. A sinus tract completely lined by ciliated epithelium, containing debris and blood cells, can be observed (×100). ***E***. Detail of the area of the cyst cavity demarcated by the square in C. The epithelial lining consists of ciliated columnar epithelium with goblet cells, and stratified squamous epithelium in some parts. The cyst cavity is partly occupied by debris and inflammatory cells (original magnification ×100, inset ×400). Reprinted with permission from Ricucci D, Loghin S, Siqueira JF Jr, Abdelsayed RA. Prevalence of ciliated epithelium in apical periodontitis lesions. *J Endod*. 2014;40:476–483.

treatment procedures, among which orthodontic forces and intentional replantation are most common. For more details, see Chapter 15.

Depending on the amount of calcified tissue lost, root resorption can be detected by periapical radiographs. However, in many cases, the condition can be disclosed only by histologic sections. Laux et al.,[87] examined histologically 114 teeth and found that only 19% were diagnosed as having apical resorption on the basis of radiographs, whereas histologically, 81% of the teeth exhibited inflammatory apical root resorption. Vier and Figueiredo[88] examined 74 root apices associated with apical periodontitis lesions using light microscopy and scanning electron microscopy (SEM); 75% of the roots showed internal apical resorption.

Odontoclasts are the cells that resorb dental hard tissues. They are morphologically similar to osteoclasts[89]; the main difference is their smaller size and fewer nuclei. Osteoclasts and odontoclasts possess similar enzymatic properties[90] and resorb their target tissues in a similar manner. Both create resorption depressions termed Howship's lacunae on the surface of the mineralized tissues (Figure 6-19A–D).[91] These cells attach to their substratum via binding of integrins in the clear zone of the clastic cell to the amino acid motif Arg-Gly-Asp (RGD), present in proteins of the bone or tooth matrix.[92] After binding of the clastic cell to the bone/tooth, an intracellular signal cascade is generated that promotes re-organization of the clastic cell cytoskeleton and induces acidic vesicles to migrate to the region of the ruffled border. The ruffled border, also called brush border, corresponds to a number of cytoplasmic folds. These originate a network of channels that establish a communication between the attacked surface of the target mineralized tissue and the cell cytoplasm (Figure 6-19D). After adhesion, the bone surface beneath the clastic cell becomes segregated from the environment. Consequently, acids and enzymes released by the clastic cell concentrate on the surface of the subjacent mineralized tissue, leading to resorption.

Similar to what happens in the control of clastic functions during bone remodelling,[93] the OPG/RANKL/RANK system seems to be implicated in root resorption (see next section).[94] This system is responsible for the differentiation of clastic cells from their precursors via complex cell–cell interactions with osteoblasts and marrow stromal cells.

The precise events involved in triggering apical root resorption have not been completely elucidated, but it is evident that pulpal/periradicular inflammation and infection are responsible for the onset and the advancement of the resorptive process. Microbial stimulation, direct or indirect, of clastic cells is necessary for hard tissue resorption.[95]

Odontoclasts do not normally adhere to nonmineralized layers of the precementum covering the apical root surface. Similar to what happens in internal root resorption, where a breach in integrity of the predentin and odontoblast layer is necessary for resorption to occur, the precementum and

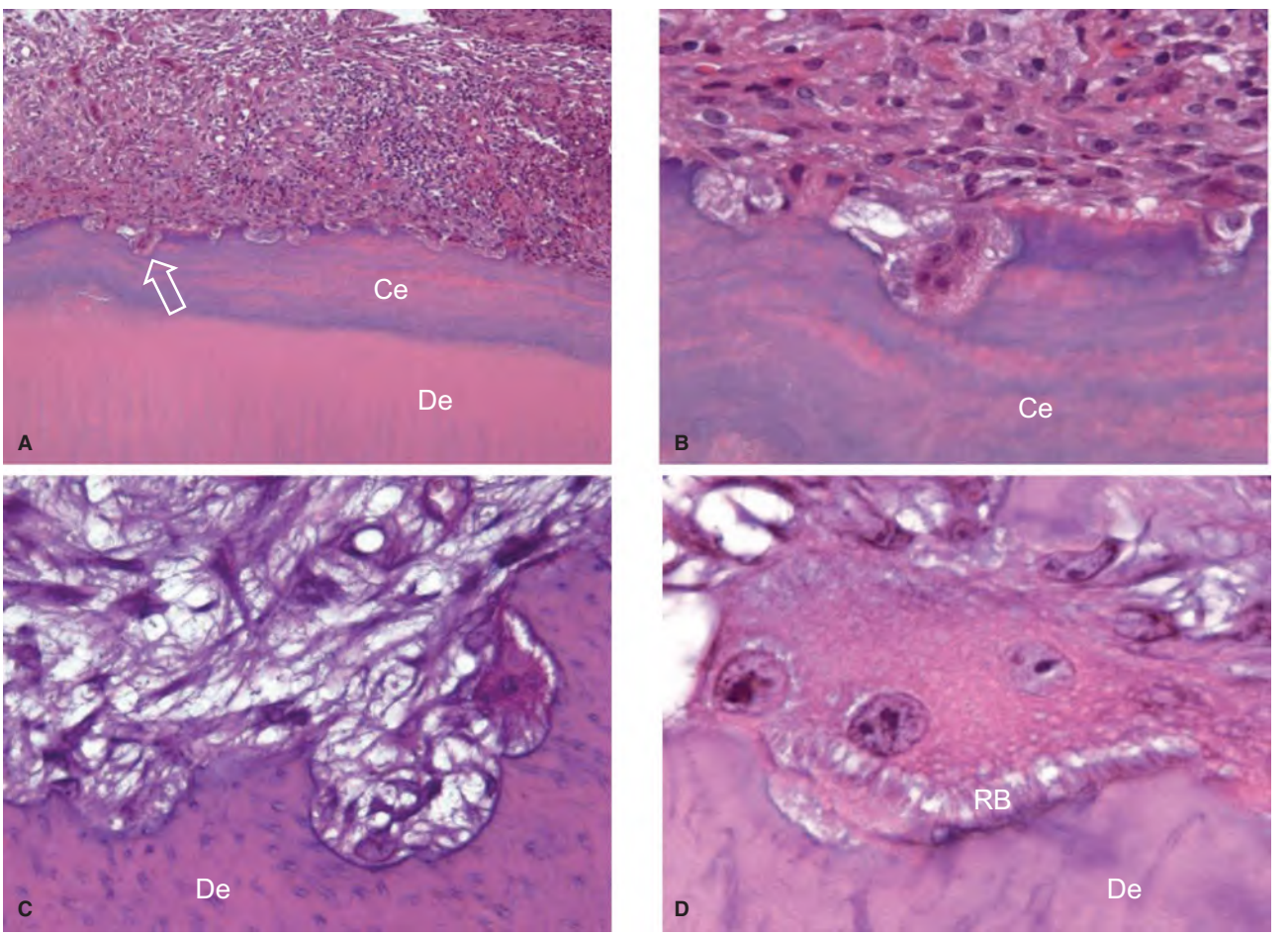

FIGURE 6-19 Apical external root surface of a tooth with periradicular inflammatory disease. ***A***. Cementum resorption is evident, as demonstrated by the presence of numerous Howship´s lacunae (Hematoxylin and eosin, original magnification ×100). ***B***. Magnification of the lacuna indicated by arrow in A showing an odontoclast (×400). ***C***. Another root in which dentin is denuded from cementum. Resorption lacunae are present. An odontoclast can be discerned in the major lacuna, adhered to dentin (×400). ***D***. High power view from a neighboring area, showing a large odontoclast with three nuclei containing several nucleoli. The ruffled border can be seen all along the cell/dentin interface (×1000) (De, dentin. Ce, cementum. RB, ruffled border).

cementoblast layers need to be lost in order to stimulate cementum resorption.[95,96] Other than traumatic dental injuries or orthodontic forces, periradicular inflammatory changes due to endodontic infection may be responsible for the damage to precementum and cementoblast layer.

Microbial infection of dentinal tubules contributes significantly to maintain root resorption. Dentinal tubules, underneath endodontic biofilm communities, are usually invaded by bacterial cells from the bottom of the biofilm structure.[97,98] If the cementum layer is lost for any reason (e.g., superficial root resorption due to apical periodontitis lesion or trauma), dentinal tubule infection or bacterial mediators passing through tubules may sustain inflammation and the root resorption process.[99–101]

One example of extensive apical resorption caused by periradicular inflammation leading to considerable shortening of the apical structure is illustrated in Figure 6-20. It is a mandibular second premolar of a 58-year-old man extracted with an apical periodontitis lesion firmly attached to the root tip by a sleeve of dense collagen bundles (Figure 6-20C). The resorptive process destroyed the apical constriction and an embedded pulp stone was formed on the apical right wall (Figure 6-20C). The clinical relevance of these morphologic changes is evident.

Another example of apical root resorption associated with periradicular inflammation that altered considerably the apical structure is shown in Figure 6-21. It is a palatal root of a maxillary molar of a 45-year-old woman. It is interesting to note that, other than the apical top and the foraminal area, resorption affected the periforaminal root surfaces, with resorption lacunae in the cementum layer (Figure 6-21A, B).

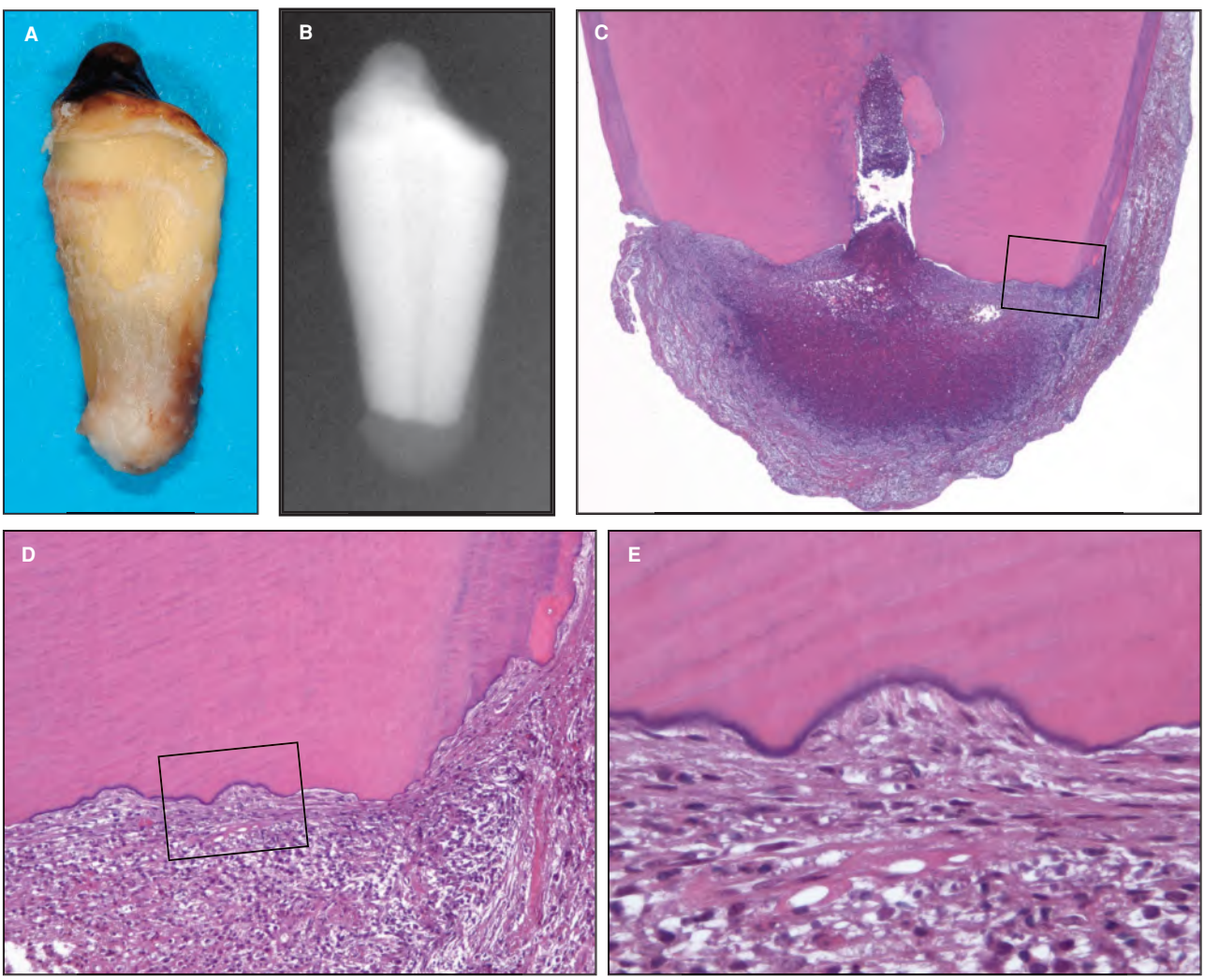

FIGURE 6-20 Mandibular second premolar with necrotic pulp, extracted with an apical periodontitis lesion attached. ***A***. Mesial view. ***B***. Radiograph of the specimen. ***C***. Section cut through the main apical foramen. Extensive resorption of the apical structure. Inflammatory tissue is visible in the apical foramen, as well as a pulp stone embedded in the root canal wall (Hematoxylin and eosin, original magnification ×16). ***D*** and ***E***. Progressive magnifications from the right apical portion showing resorbed dentin surface denuded from cementum (×100 and ×400).

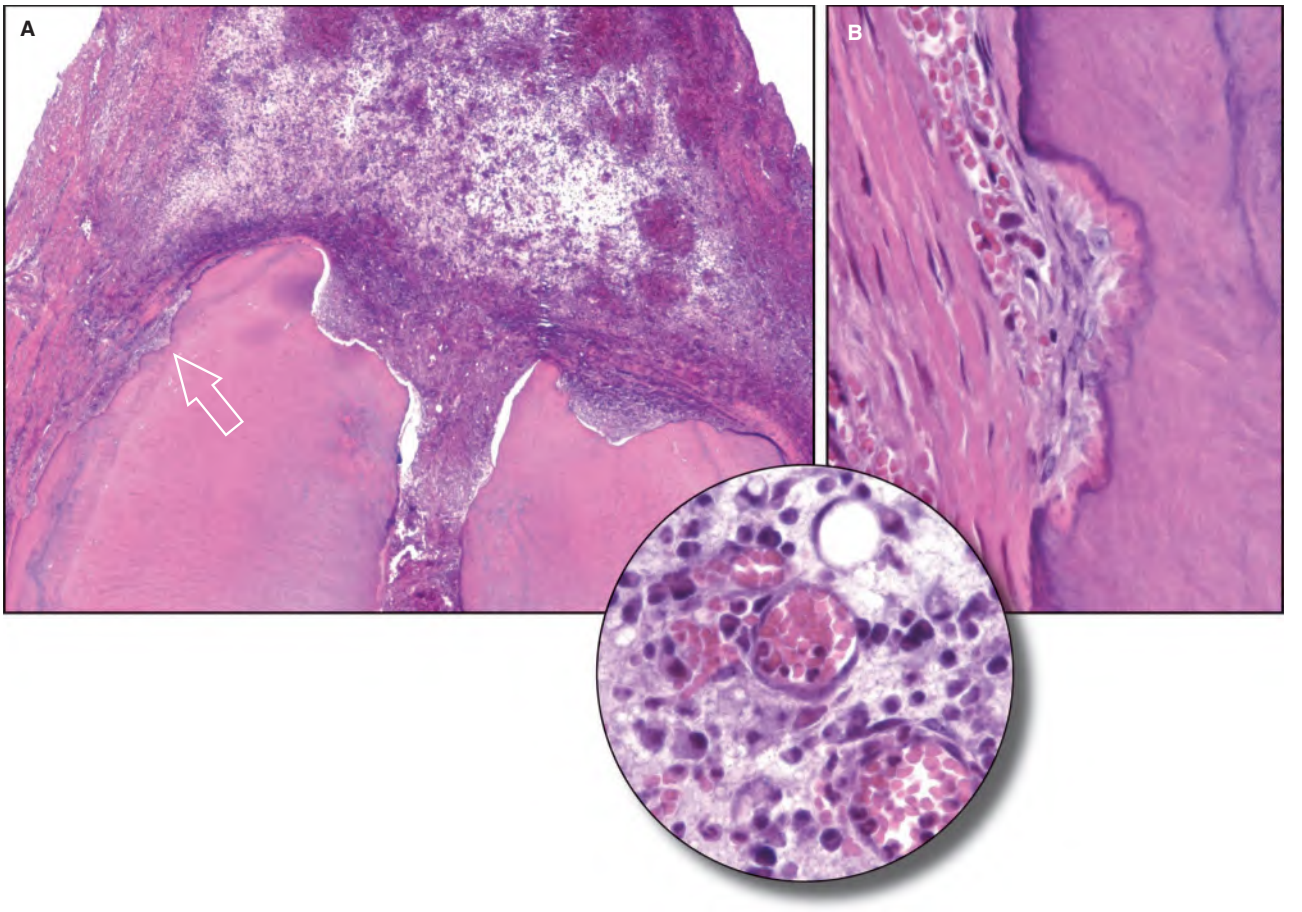

FIGURE 6-21 Palatal root of a maxillary first molar with necrotic pulp. ***A***. Section cut approximately at the center of the apical canal. Massive resorption of the apical structure can be appreciated (Hematoxylin and eosin, original magnification ×25). ***B***. High power view of the area of external radicular surface indicated by arrow in A (×400). Inset. High power view from the center of the apical periodontitis lesion. Concentration of acute and chronic inflammatory cells. Congested blood vessels in cross section (×1000).

Hypercementosis

Less frequent than resorption, the pathologic deposition of several layers of cementum around the apical structure can be observed in association with some periradicular lesions. One example is illustrated in Figure 6-22. In histologic sections, dentin appears surrounded by several layers of alternating cellular and acellular cementum, giving the appearance of the so-called "mixed stratified cementum."

IMMUNOLOGICAL ASPECTS OF PERIRADICULAR DISEASES

In response to irritants (usually microbial) egressing from the root canal system via apical, lateral, and furcal foramina, the host mobilizes its immune defense apparatus and concentrate cells and molecules in the tissue compartment near these foramina in an attempt to confine the infection to the canal. The immune response to tissue injury is a double-edged sword. On one hand, it has a protective function against infection, preventing dissemination to other areas, and preparing the injured area for repair. On the other hand, the immune reaction itself can lead to undesirable effects, such as tissue destruction resulting in pain, liquefaction, and bone resorption.

According to the intensity of aggression and host susceptibility state, the response may be acute or chronic.

Acute Periradicular Response

Microorganisms reaching the periradicular tissues from root canals are initially combated by local host defenses represented by resident macrophages and the complement system. Microbial antigens and virulence factors accumulate in the intraradicular biofilm and diffuse through tissues to reach the periradicular tissues and activate the immune defenses. For instance, bacterial cell structural constituents can activate macrophages as well as the complement system via the alternative[102] and lectin pathways.[103] Bacterial structural components and metabolic end-products are cytotoxic and can induce release of pro-inflammatory cytokines.[104,105] Bacterial peptides and metabolites are chemoattractants to PMNs.[106]

Macrophages and other defense cells possess surface receptors that recognize microbial substances and give rise

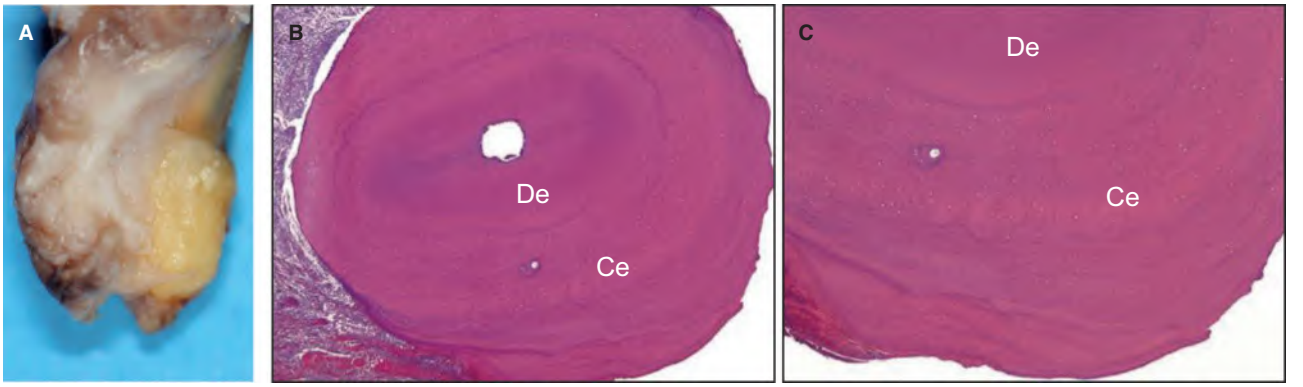

FIGURE 6-22 Distal root from the mandibular molar in Figure 6-9A. ***A***. Hypercementosis is associated with the periapical pathologic tissue. ***B*** and ***C***. Cross-cut section taken at the level of the line in A. Dentin (De) is surrounded by overgrowing layers of cementum with different morphologic characteristics, the so-called "mixed stratified cementum" (Hematoxylin and eosin, original magnification ×16 and ×25).

to signals that activate antimicrobial and pro-inflammatory functions. Once activated by microbial structural components, macrophages exhibit stronger phagocytic and antigen-presenting capabilities. Activated macrophages increase the production of pro-inflammatory mediators, including interleukin-1 (IL-1), IL-6, and tumor necrosis factor (TNF)-a the chemokine IL-8/CXCL8, prostaglandins, leukotrienes, lisosomal enzymes, oxygen metabolites, nitric oxide, and platelet-activating factor.[107-111]

Activation of the complement system leads to increased antimicrobial activity of the serum by inducing cytolysis of microbial cells and promotes opsonization for improved performance of phagocytes. Some products of complement activation, such as C3a and C5a, can stimulate mast cells to degranulate and release histamine,[112,113] an important mediator of acute inflammation, by causing arteriolar dilation and increased venular permeability.[114] C5a is also a powerful chemotactic agent for phagocytes.

Activation of macrophages and the complement system results in the production and release of several molecules that stimulate the next step in host defense against infection (acute inflammation). It involves several vascular and cellular changes in the affected tissues. Increased vascular permeability is the hallmark of acute inflammation. The fluid that leaves the vessels (exudate) accumulates in the extravascular space to form edema that causes an increase in tissue hydrostatic pressure, with consequent swelling and pain.

Because microbial infection in the root canal cannot be eliminated by the host defenses alone, more phagocytic cells are recruited to the injury area. Consequently, the host ability to cope with the infection is increased, but excessive recruitment of PMNs coupled with intense damage to the periradicular tissues can result in abscess formation.

Cytokines, such as IL-1 and TNF, released by macrophages in response to microorganisms, stimulate endothelial cells to sequentially express different molecules that mediate the preferential attachment of different types of leukocytes.[115] Microbial structural components may also act directly on endothelial cells to promote the same effects.[116] Slowing of blood flow, as a result of increase in vascular permeability and activation of endothelial cells by mediators such as IL-1, TNF, and LPS, generates conditions that are conducive to leukocyte margination, rolling and adhesion to the endothelium. As the leukocyte rolls on the surface and then adheres to the endothelium, it may be activated by chemokines displayed on endothelial cells. In response to chemokines, leukocytes rearrange their cytoskeletons and become more motile.[117] Afterward, they leave the vessel by crossing the endothelial lining and, once in the extravascular space, they are attracted to the site of injury by chemotaxis induced by microbial products and host-derived factors (particularly chemokines, C5a, and LTB4).[115,118]

PMNs are the first leukocytes to migrate to the injured area. Once exposed to IL-8 and TNF-α, PMNs are activated and increase the production of oxygen radicals and release of granule contents, improving the bacterial killing ability. However, as a side effect, activated PMNs can also cause damage to the host tissues due to release of oxygen radicals and lisosomal enzymes. Abscess formation is the extreme form of tissue injury caused by PMNs in response to bacterial infection.

CHRONIC PERIRADICULAR RESPONSE

Microbes have numerous distinct antigens. Since endodontic infections are characterized by multispecies microbial colonies, one can realize the myriad of antigens that accumulate in a biofilm mass. Ingredients from this "antigenic soup" constantly diffuse and contact the periradicular tissues. Asymptomatic apical periodontitis develops as an adaptive immune response to the chronic antigenic stimulation egressing from the infected root canal.

The adaptive immune response develops in a host already sensitized to the antigens that induced the response. Sensitization occurs following the first contact with the antigen and at the level of draining regional lymph nodes. Recirculating naive B and T cells have more chances to

encounter the antigen to which they are specific and then undergo all the processes necessary to become activated.

The initial acute inflammatory response to infection generates a large number of antigenic fragments as a result of microbial killing. These fragments accumulate in the inflammatory exudate and are captured by dendritic cells that direct them to the local lymph nodes via lymphatics. Lymphocytes are constantly circulating through the lymph nodes. Consequently, these cells have more chances to encounter the antigen to which they are specific, rather than in the periradicular tissues. After antigen recognition, lymphocytes are activated to proliferate and differentiate in effector cells. Immune cells rarely proliferate in the periradicular area.[119] Thus, for the initiation of the adaptive immune response against endodontic pathogens, antigens must be taken to regional lymph nodes where recognition and lymphocyte activation can occur.[120]

Effector immune cells include plasma cells, originated from B cells, which secrete antibodies, and effector T cells that can activate other immune cells (CD4+ T lymphocytes) or destroy target cells infected by virus (CD8+ cytolytic lymphocytes). Some cells do not enter the differentiation process and give rise to memory cells.

Effector cells leave the lymph node, enter the circulation, and are attracted by chemokines to the source of antigens, that is, the injury site (the periradicular area). Effector immune cells then become established and accumulate in the periradicular tissues to eliminate the source of antigens or at least curb the spread of infection.

In sensitized individuals, a secondary immune response is faster against microbial species involved in the root canal infection. Sensitization may have occurred after previous contact with antigens from the same bacterial species present in caries, gingivitis, periodontal disease, or apical periodontitis in another tooth. Memory T cells predominate over naive T cells in periradicular lesions.[121]

A chronic inflammatory response can start from the onset or be resultant of chronicization of a previous acute episode. In response to microbial infection, chemical mediators are released into the periradicular tissues by macrophages, T cells, and other cell types and may reach critical concentrations to stimulate osteoblasts and bone stromal cells to express mediators that activate osteoclasts and bone resorption.[122,123] Bone is resorbed and replaced by a granulomatous tissue basically composed of immunocompetent cells such as lymphocytes, plasma cells, macrophages, and PMNs, as well as components of granulation tissue, including fibroblasts and newly formed nerves and blood vessels. The bone resorptive process creates enough space to accommodate a large number of defense cells in the area adjacent to the apical foramen in an attempt to confine the infection in the root canal. In the periphery of the granulomatous tissue, collagen fibers encapsulate the lesion. At a certain point, bone resorption is sufficient to produce a radiolucent lesion that is discernible radiographically. A granuloma is formed.

The predominant inflammatory cells in chronic periradicular lesions are lymphocytes and plasma cells, followed by mononuclear phagocytes (macrophages and dendritic cells) and neutrophils.[44,121,124,125] Mast cells and Natural Killer (NK) cells can also be found.[37,121,126,127] Even though PMNs are typical of acute inflammation, long-lasting chronic inflammatory response to root canal infection can exhibit large numbers of PMNs.[115] These defense cells accumulate particularly in areas adjacent to where microorganisms are located (e.g., near the apical or lateral foramina and around actinomycotic colonies).[10]

A variety of chemical mediators are produced in the inflamed periradicular tissues in response to bacterial infection of the root canal. Cytokines, such as IL-1α and β, TNF-α, IL-6, and macrophage colony-stimulating factor (M-CSF), chemokines, and prostaglandins are all bioactive molecules with great relevance in inducing inflammation and bone resorption.[106,128-130] These proinflammatory and proresorptive mediators can play an important role in pathological bone resorption associated with apical periodontitis.[1,131]

Chemical mediators primarily involved in regulation of osteoclastogenesis and bone resorption include receptor activator of NF-κB ligand (RANKL), M-CSF, and osteoprotegerin (OPG).[132] Differentiation of the precursors into osteoclasts requires the presence of osteoblasts/marrow stromal cells, which produce both RANKL and M-CSF.[92,133] M-CSF provides essential signals for survival and proliferation of osteoclast precursors.[136] RANKL is a surface molecule that is essential for differentiation of precursors into osteoclasts through interaction with RANK expressed on the osteoclast precursor.[134] RANKL can also occur in soluble form. To summarize, RANKL binds to RANK to activate osteoclastogenesis in the presence of survival factor M-CSF.

OPG is also produced by osteoblasts/marrow stromal cells and serves as a soluble "decoy" receptor that competes with RANK for RANKL to inhibit osteoclastogenesis and osteoclast activation.[135] The balance between RANKL (stimulator) and OPG (inhibitor) expression determines the amount of bone to be resorbed. Inflammation influences this balance, so that excessive bone resorption may ensue. Pathologic bone resorption typical of apical periodontitis is associated with increased RANKL/OPG ratio.[136,137] Upregulation of RANKL expression in apical periodontitis lesions correlates with the phase of lesion expansion.[136,137] A higher expression of RANKL than OPG has been reported in granulomas in comparison with cysts, resulting in a greater RANKL/OPG ratio and consequent increased bone resorption activity in granulomas.[138]

Immune cells participate in the differentiation and activation of osteoclasts in chronic inflammatory processes through the release of proinflammatory cytokines, chemokines, and other chemical mediators. These mediators can reach critical concentrations that indirectly stimulate

osteoclastogenesis by inducing osteoblasts/stromal cells to express RANKL and decrease OPG production.[122,123] Direct effects on osteoclastogenesis may also be induced by immune cells, since some cell types other than osteoblasts/stromal cells, including fibroblasts and T and B cells, can also produce RANKL.

Proinflammatory mechanisms must be tightly controlled in order to prevent excessive tissue destruction. The balance between proinflammatory and anti-inflammatory mediators controls the extent of host responses to antigen stimulation within chronic inflammatory processes. It has been suggested that T_H1 immune response, mostly mediated by proinflammatory cytokines such as IL-1, TNF-α and IL-6, is involved in the phases of lesion expansion as a result of bone destruction. Conversely, T_H2 response, mostly mediated by cytokines IL-4, IL-5, IL-5, IL-10, and transforming growth factor (TGF)-β, exert an important role in the healing process and restriction of the immune response.[139,140] T_H2 response can also exert an important protective role given its participation in the humoral response.[141] Colić et al.[121] proposed that T_H1 immune response is important for all stages of apical periodontitis development, while T_H2 and immunoregulatory cytokines are more significant in the process of stabilization of the lesion size.

T-regulatory cells (Tregs) can also be involved in regulation of destructive immune responses. These cells have been detected in apical periodontitis lesions.[142] Both T_H1 and T_H2 responses can be suppressed by Treg cells.[142] Treg infiltration in apical periodontitis may be delayed in comparison with other innate or adaptive immune cells, possibly to control excessive lesion expansion.[143]

T_H17 is a subset of T_H cells that seem to play a dominant role in exacerbating inflammation.[144] These cells produce IL-17, which stimulate IL-8/CXCL8 production and attraction of PMNs to the site. T_H17 cells and IL-17 have been detected in apical periodontitis lesions.[38-39,45,145]

Factors that may stimulate epithelial proliferation include the epidermal growth factor and the keratinocyte growth factor produced by macrophages and fibroblasts, respectively. Both factors have been detected in apical periodontitis lesions.[146,147] Other mediators produced by cells in the granuloma can also stimulate epithelial proliferation, including TNF, IL-1, IL-6, prostaglandins, and insulin-like growth factor.[55] Microbial components, such as LPS, can also stimulate the epithelial proliferation.[148]

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Chapter 7

Dental Innervations and Pain of Pulpal Origin

Nikita B. Ruparel, Asma A. Khan

The dental pulp is a highly innervated tissue in the body. It primarily consists of two types of innervations: sensory and autonomic. While sensory nerves are the primary conductors of noxious information within the dental pulp, sympathetic and parasympathetic nerves play a role in regulating the vasculature, with both types serving to maintain pulpal homeostasis. Evidence of parasympathetic fibers in the dental pulp is controversial.

Sensory innervation in the entire orofacial region, including teeth, comes primarily from the trigeminal system with cell bodies in the trigeminal ganglion. The maxillary component of the dental sensory innervation is derived from V2 (maxillary division), while the mandibular component is derived from V3 (mandibular division). On the other hand, the autonomic innervation comes from the superior cervical ganglion.

CLASSIFICATION

Anatomical and electrophysiologic studies have demonstrated the presence of A-type and C-type fibers within the dental pulp. The subtypes of A-fiber type are Aα, Aβ, Aγ, and Aδ. Each subtype has distinct functions (Table 7-1) with Aδ functioning to detect noxious stimuli. Based on the conduction velocity, all fiber types are classified as either fast-conducting A-fibers or slow-conducting C-fibers. A-fibers are myelinated fibers, whereas C-fibers are unmyelinated; the presence or absence of the myelin sheath accounts for the conduction velocities of these fiber types by forming the nodes of Ranvier. Moreover, the thicker the myelin sheath, the larger is the fiber diameter and the greater is the distance between adjacent nodes (i.e., conferring faster conduction velocity). Conceivably, C-fibers are only 0.4–1.2 μm in diameter, whereas Aδ are 2–5 μm in diameter.

Primate studies have demonstrated that ~70%–80% of the pulpal terminals are unmyelinated.[1] These include postganglionic sympathetic fibers and sensory nerve fibers. Labeling studies[2–5] and some immunohistochemical studies,[6] evaluating sensory nerve terminals, indicate that despite most sensory fibers being unmyelinated within the dental pulp, the majority have a myelinated origin (Figure 7-1). Large diameter myelinated fibers lose some, or all, of their myelin sheath at the port of entry into the tooth and appear as unmyelinated or thinly myelinated. Therefore, the dull aching pain, which is popularly attributed solely to the slow conducting C-fibers, may also actually be due to the slower conducting Aδ-fibers that have now lost their myelin sheath. Of course, the sharp shooting pain is still primarily attributed to the relatively faster conducting Aδ-fibers, as their C-fiber counterparts completely lack any myelination. Moreover, high-threshold electrical stimulation is also mediated by Aδ-fibers. Other types of pain sensations, such as prepain, are mediated by Aβ-fibers.[7–9]

Interestingly, another school of thought attributes dentinal pain to low-threshold mechanoreceptors (LTMs).[10] Stimuli such as air-puffs and water spray, when applied to gingival tissues, evoked a sensation of touch but the same stimuli evoked a sensation of pain when applied to exposed dentin.[10] Both air-puffs and water spray do not evoke sufficient mechanical force within the dental tubules to activate high-threshold nociceptors. Therefore, LTMs (or "algoneurons"[10]) may be the ones mediating the painful sensation when activated by low-threshold stimuli within a tooth. Possible mechanisms contributing to LTM-induced dentinal pain may be the projection of Aβ-fibers onto the same second-order neurons as nociceptors[10,11] as well as release neurotransmitter content that is typically present in nociceptor population, such as calcitonin-gene related peptide (CGRP), thereby activating projection neurons in the trigeminal brainstem complex.[10] Moreover, neonatal capsaicin treatment in rats that cause ablation of C-fiber nociceptors in the hindlimb do not show any loss in pulpal afferents,[10,12,13] suggesting that the dental pulp may primarily be innervated with A-fiber LTMs.

TABLE 7-1 Classification of Nerve Fibers in the Dental Pulp

Type of Fiber	Function	Diameter (μm)	Conduction Velocity (m/s)
Aα	Motor, proprioception	12–22	70–120
Aβ	Pressure, touch	5–12	30–70
Aγ	Motor	3–6	15–30
Aδ	Pain, temperature, touch	2–5	12–30
C	Pain	0.4–1.2	0.5–2
Sympathetic	Postganglionic sympathetic	0.3–1.3	0.7–2.3
Parasympathetic	Postganglionic parasympathetic	Unknown	Unknown

(Modified with permission from Kim S, Heyeraas KJ, Haug SR. Structure and function of the Dentin–pulp complex. In: Ingle JI, Bakland L, Baumgartner JC. *Ingle's Endodontics*. 6th ed. BC Decker Inc.; 2008:137.)

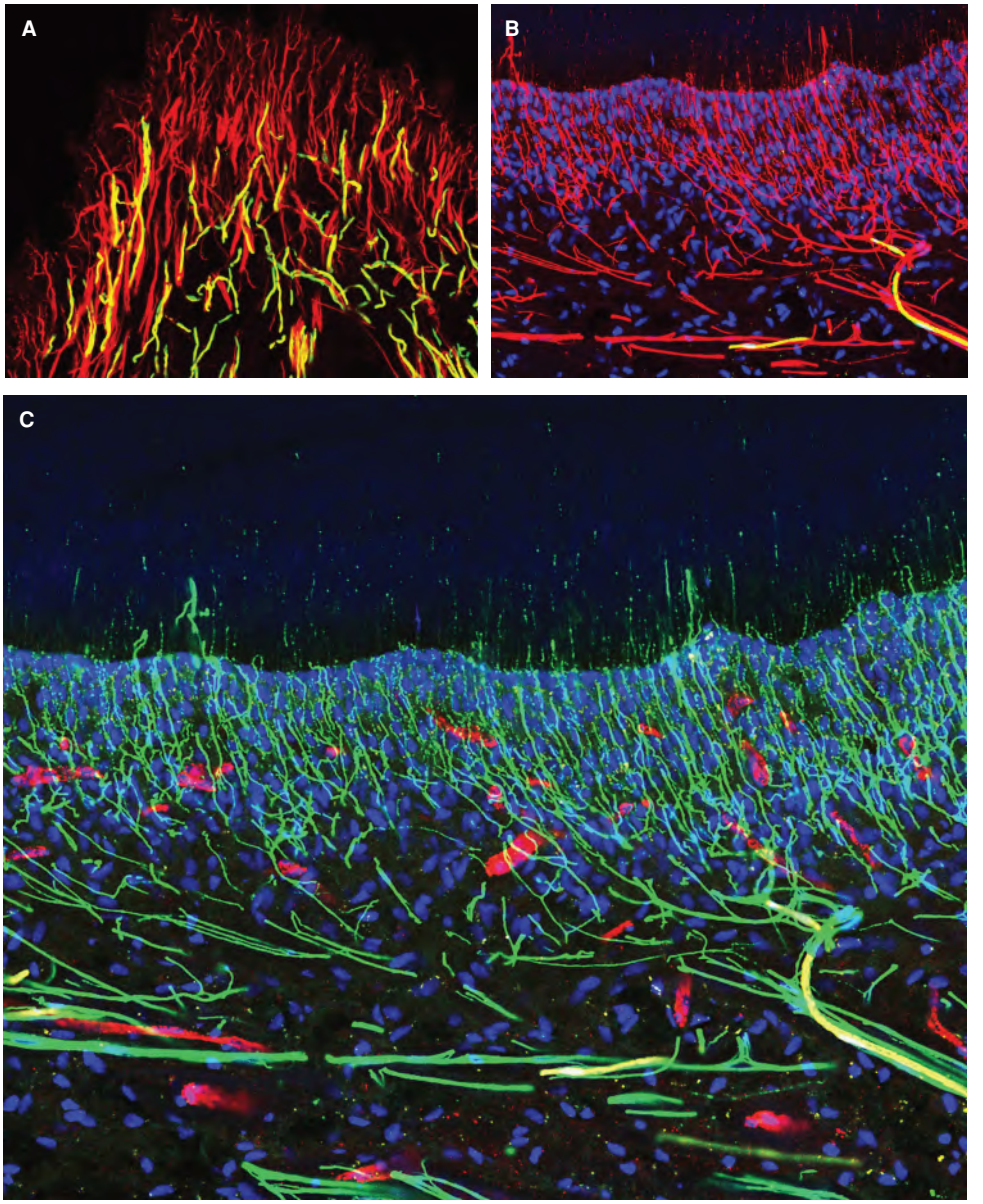

FIGURE 7-1 Colocalization (yellow) of N52 (red) is seen with myelin basic protein (MBP; green); however, axon bundles located in subodontoblastic plexus/odontoblast layer (SP/OL) lack MBP (arrowheads) as seen in the pulp horn of a pulp section ***A.*** and in SP/OL area in a decalcified specimen ***B.*** Confocal micrograph of the pulpodentin interface in a decalcified section from a tooth with a carious lesion and stained with N52 (nerve fibers; green), von Willebrand factor (blood vessels; red), myelin basic protein (myelin sheath; yellow arrow), and DAPI (nuclei; blue) shows that most fibers near the odontoblast layer lack a myelin sheath. Some of the nerve fibers extend into the dentin ***C.*** (Courtesy of Dr. Michael A. Henry, San Antonio, TX, U.S.A.)

Collectively, the dental pulp consists of some unique nerve fiber phenotypes that make it an intriguing tissue to study and understand pain mechanisms.

DISTRIBUTION

All primary afferent fibers make their entry into the pulp primarily via the major apical foramen. They are organized into bundles located in the center of the pulp. While many unmyelinated fibers terminate freely in the stromal of the pulp, some sensory fibers continue their path toward the subodontoblastic layer, forming the Plexus of Rashkow. From here, some even continue sending extensions into the dentinal tubules. This organization is key to understand the quick response of these nerve endings to various noxious stimuli and dentinal hypersensitivity.

The pattern of sensory innervation within the pulp follows a gradient in which regions, such as the pulp horn, are densely innervated with Aβ- and Aδ-fibers. However, the number of these fibers at the cervical region is low and is even lower in root dentin. Moreover, in the pulp proper, the primary fiber types found are the unmyelinated C-fibers. This distribution has implications on sensibility tests such as electric pulp tests (EPTs). According to many studies,

the optimal site for placement of the EPT electrode is the incisal, or incisal third, of an anterior tooth[14,15] or mesiobuccal cusp of a molar tooth,[16] both of which are regions with prominent pulp horns.

The pattern of distribution is attributed to the concentration and dependence of different nerve fibers on different neurotrophins.[17,18] A study using semaphorin3a knockout mice (Sema3a) has demonstrated that sema3a regulates the timing of navigation and the pattern of the terminals within the developing dental pulp.[19] Moreover, glial cell line-derived neurotrophic factor has been seen close to the coronal pulp with large diameter mechanosensitive A-fibers,[18] whereas nerve growth factor (NGF) is associated with the remainder of the fiber types.[17] Overall, the role of neurotrophins is critical for the spatial and temporal distribution of different nerve fibers in the dental pulp. This is not only prevalent during development but also during adulthood (see subsequently) and plays a strategic role in defending the pulp from tissue injury.

NEUROPEPTIDES

Nerve fibers innervating the dental pulp are classified as peptidergic or nonpeptidergic, based on the presence or absence of neuropeptides, such as CGRP[20–25] and Substance P (SP).[24–30] Aδ- and C-fibers are classically known to contain these two neuropeptides in their cell bodies. Under conditions of inflammation, they are able to package these peptides into vesicles, transport them to the terminals, and release them.[31]

Other neuropeptides present in the dental pulp and released by pulpal axons include neurokinin A (NKA),[24,30] neuropeptide Y (NPY),[32–35] vasoactive peptide (VIP),[25] cholecystokinin, somatostatin,[36,37] galanin,[38] cholecystokinin,[37] β-endorphin, and enkaphain.[37,39] Table 7-2 lists the commonly known neuropeptides, their source, and functions in the dental pulp.

Function

The dense innervation of the dental pulp with sympathetic and sensory nerve fibers indicates a potential role of these fibers in maintaining pulpal homeostasis and repair. The following subsections discuss three major functions of these fibers under normal and conditions of tissue injury.

Role in Regulation of Pulpal Blood Flow

In absence of tissue injury, the pulpal blood flow is maintained so that the pulp tissue is adequately perfused, delivering appropriate amounts of nutrients and oxygen to the cells residing within the pulp. Pulpal blood flow also maintains tissue pressure by keeping the capillary as well as colloidal tissue pressure and balance of proteins inside the capillaries and in the interstitium. On the other hand, under conditions of tissue injury and inflammation, two categories of nerve fibers are activated; sympathetic and sensory nerve fibers. The initial response of the pulp is reduction in pulpal blood flow. This effect is primarily mediated by neurotransmitters/peptides such as norepinephrine and NPY that are locally produced by sympathetic nerve fibers. Norepinephrine, in turn, activates α-adrenergic receptors expressed in smooth muscle cells of nearby arterioles to cause vasoconstriction.[40,41] Thus, the pulp undergoes a period of reduced blood flow upon sympathetic activation. Indeed, this effect is mimicked upon infiltration injection of local anesthetics containing "vasoconstrictors." Upon injury, however, primary afferent sensory nerve terminals are also activated.

External stimuli such as bacterial toxins, for example, lipopolysaccharides (LPSs) or inflammatory mediators, released by neighboring cells, act via respective receptors on these fibers and trigger vesicular release of many neuropeptides.[42–44] The two most extensively studied peptides are SP and CGRP. These peptides play a key role in mediating vasodilation and vascular permeability,[26] two essential components of "*neurogenic inflammation*." The effect of these neuropeptides is mediated via both Aδ- and C-fibers.

The neural regulation of pulpal blood flow is evident from anatomical studies showing the close proximity of peptidergic nerve fibers with pulpal microvasculature (Figure 7-2).[6] Moreover, sprouting of nerve fibers is also evident upon inflammation, suggesting an increase in the overall number and activation of fibers leading to neurogenic inflammation.[45] Immunohistochemical analysis demonstrates an increase in levels of SP, CGRP NPY, and NKA after irreversible pulpitis.[24,25,31,46,47] Transient changes in the pulpal vasculature are well tolerated by the pulp. However, under conditions of persistent injury or inflammation, the pulpal defenses are overwhelmed and unable to be sustained. For more details, see Chapter II.

TABLE 7-2 Tabulation of Dental Pulp Neuropeptides, Their Sources, and Functions

Neuropeptides	Source	Function
Substance P (SP)	Sensory nerve terminals	Vasodilation
Calcitonin-gene related peptide (CGRP)	Sensory nerve terminals	Vasodilation
Neurokinin A (NKA)	Sympathetic nerve terminals	Vasoconstriction
Vasoactive peptide (VIP)	Parasympathetic nerve terminals	Vasoconstriction
Neuropeptide Y (NPY)	Sensory nerve terminals	Vasoconstriction
β-endorphin	Sensory nerve terminals	Inhibition of SP release
Enkaphalin	Sensory nerve terminals	Inhibition of SP release
Somatostatin	Unknown	Inhibition of SP release
Galanin	Unknown	Unknown
Cholecystokinin	Unknown	Unknown

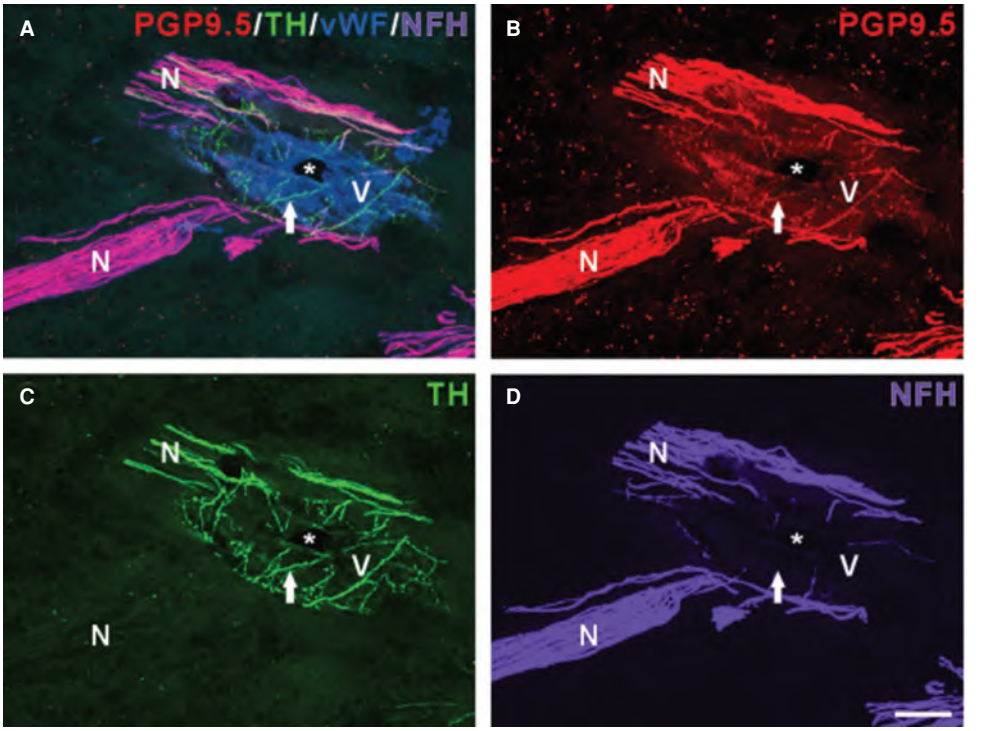

FIGURE 7-2 Confocal micrographs showing PGP9.5 ***B.*** tyrosine hydroxylase, a marker for sympathetic fibers ***C.*** and NFH ***D.*** identified nerve fibers closely associated with a blood vessel identified with von Willebrand factor (vWF), marker for blood vessels ***A.*** The *asterisk* indicates the lumen of the blood vessel. (Reproduced with permission from Henry et al.[6])

Role in Immune Regulation

Neuropeptides released from terminal nerve fibers are known to regulate the immune response. Studies evaluating SP have demonstrated several effects of this peptide in mediating proinflammatory effects in pulp tissue. These include recruitment and enhancement of macrophage-induced phagocytosis,[48,49] release of proinflammatory cytokines by macrophages,[50,51] and proliferation of T lymphocytes.[52,53] Similar effects on macrophage activation are also demonstrated by the neuropeptide, NKA.[54] CGRP, on the other hand, inhibits some of these effect while continuing to participate in neurogenic inflammation by triggering release of histamine from mast cells.[55]

In addition to their role in the dental pulp, neuropeptides have also been implicated in regulating periapical disease. Increased levels of VIP have been shown to limit the size of a periapical lesion.[54,56] This effect may be mediated by inhibition of macrophage-induced cytokine production thereby inhibiting osteoclastogenesis[57,58] and/or by $CD8^{+}$ lymphocyte apoptosis.[59] Similar effects have been observed with CGRP as well. Moreover, expression of SP on immune cells and endothelial cells indicates its potential key role in regulating the periapical response to infections from the root canal system.

Role in Transmission of Pain

The involvement of sensory nerve fibers or nociceptors in pain mechanisms is well studied. The pathway for pain transmission in the dental pulp starts with detection of noxious chemical, thermal, or mechanical stimuli by primary afferent nerve fibers (Figure 7-3). These belong to the trigeminal system, where the first-order neurons project their central terminals to the sensory nucleus, located in the brainstem. Three different regions of this nuclei namely, nucleus oralis, interpolaris, and caudalis receive projections from these terminals. However, the preponderance of evidence points to nucleus caudalis as being the primary nucleus receiving majority of the nociceptive input (Figure 7-4).[60,61] The signal is then relayed by second-order projection neurons to the thalamus that finally sends inputs to the somatosensory cortex. Therefore, the initiation of dental pain transmission occurs peripherally within the dental pulp for pulpal pain, such as pulpitis, and from the periodontium when periapical inflammation in ensued as in cases of symptomatic apical periodontitis or acute apical abscess.

Specialized fibers that can detect a variety of stimuli such as thermal, mechanical, and chemical stimuli are called polymodal nociceptors. These include both Aδ- and C-fibers. Detection of these noxious stimuli occurs via stimuli-sensing receptors located at peripheral termini. These include G-protein-coupled receptors (GPCRs) that upon activation couple/bind to one of three G proteins, namely $G\alpha_{s}$, $G\alpha_{i/o}$, $G\alpha_{q/11}$.[62] Depending on which G protein is bound, different cellular events occur. Several inflammatory mediators activate specific GPCRs. For example, prostaglandins are known to activate $G\alpha_{s}$; activation of a stimulatory G protein leads to activation of adenylate cyclase, that causes an increase in cyclic adenosine monophosphate (cAMP) and ultimately leading to cellular activation.[62] On the other hand, activation of $G\alpha_{i/o}$ by opioid and cannabinoid agonists leads to

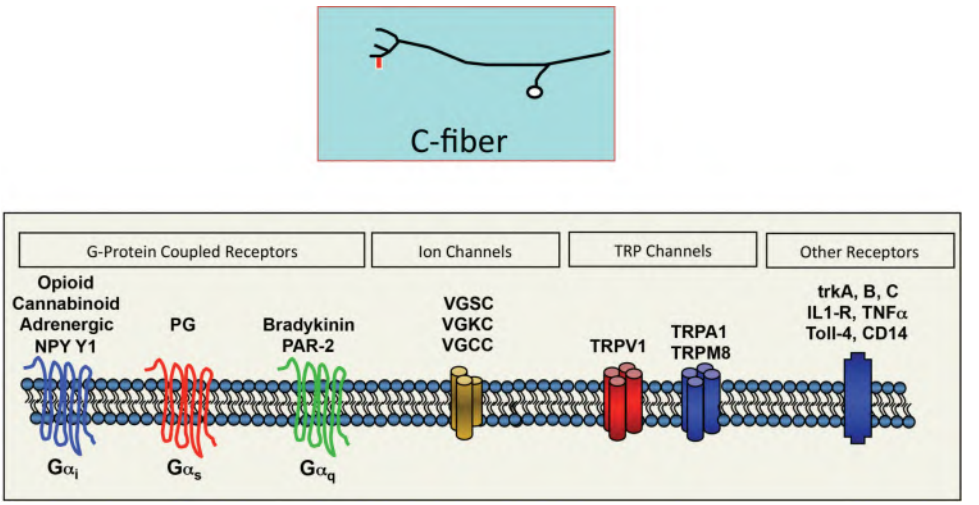

FIGURE 7-3 Cartoon depicting major classes of receptor or ion channels proposed to be present on peripheral terminals of sensory neurons that serve to transduce external stimuli into altered neuronal function. Not all receptors or ion channels are present on all neurons, and several have been shown to be altered during inflammation or nerve injury. PAR-2, protease-activated receptor subtype 2; PG, prostaglandin; TRPA1, transient receptor potential A1; TRPM8, transient receptor potential M8; TRPV1, transient receptor potential V1 (aka the capsaicin receptor); VGCC, voltage-gated calcium channel; VGKC, voltage-gated potassium channel; VGSC, voltage-gated sodium channel. (Reproduced with permission from Henry and Hargreaves.[62])

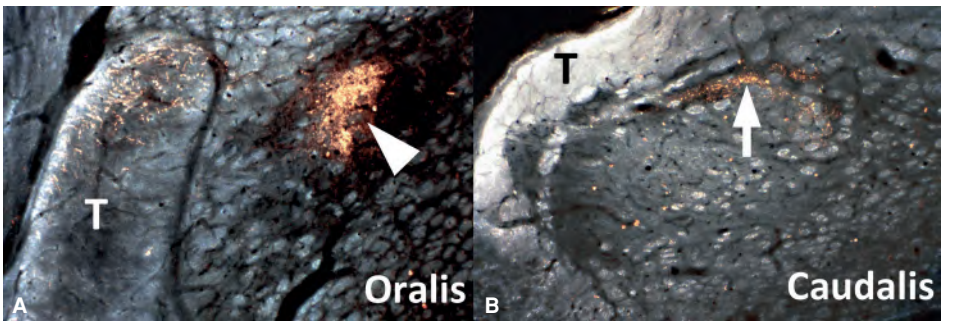

FIGURE 7-4 Dental afferents project broadly throughout the entire trigeminal sensory nuclear complex. Darkfield photomicrographs show the central projection of dental afferents (arrows) to oralis and caudalis following the transganglionic transport of horseradish peroxidase from the feline mandibular canine pulp to primary afferent terminals, located throughout the trigeminal sensory nuclei. Labeled fibers exit the trigeminal tract and terminate in rostral (oralis; arrowhead) and caudal levels of the complex including the superficial laminae of caudalis (arrow). (Courtesy of Dr. Michael A. Henry, San Antonio, TX, U.S.A.)

decease in cAMP levels leading to nociceptor inhibition.[62–64] Nociceptors also respond to inflammatory mediators by activating $G\alpha_{q/11}$; bradykinin and leukotriene receptors that activate their respective GPCRs bind to $G\alpha_{q/11}$ that activate a different signaling cascade involving phospholipase C ultimately leading to sensitization of nociceptors.[62,65,66]

Apart from metabotrophic GPCRs, ionotrophic receptors are also equally, if not more critical in nociceptor activation. One of the most extensively studied receptors is transient receptor potential vanilloid 1 (TRPV1) receptor.[62] It belongs to the ion channel family of receptors and is extensively studied for inflammatory pain. TRPV1 is capable of detecting chemical stimuli, such as capsaicin, the primary component in chili peppers, and temperatures >43°C.[67] Moreover, under conditions of inflammation, a variety of inflammatory mediators can reduce its threshold of activation by sensitizing the receptor, leading to a hyperalgesic state.[66,68] Interestingly, TRPV1 expression is evident in the dental pulp[3,69] and is increased in symptomatic and asymptomatic teeth, suggesting the critical role of TRPV1 in pain and inflammation.[70]

Other TRP channels such as TRP ankyrin 1 (TRPA1)[71–74] and TRP melastatin 8 (TRPM8)[72,75] have roles in detecting cold or cooling temperatures as well as inflammatory mediators. Overall, TRPs play a major role in being one of the first targets that detect external stimuli and mediate the transmission of pain.

Other types of receptors expressed on sensory nerve terminals include pattern recognition receptors, such as Toll-like receptors (TLRs). These recognize structurally conserved antigens produced by microorganisms, such as LPS from gram-negative and lipotechoic acid (LTA) from gram-positive microbes. Activation of TLR on nociceptors causes sensitization of receptors, such as TRPV1.[42,43] This in turn reduces the threshold of nociceptor activation and

therefore, promotes the process of spontaneous pain or lingering pain. Similar to LPS and LTA responses, local release of inflammatory meditators such as prostaglandins[76] and bradykinin[77,78] also sensitize the effects of nociceptors under conditions of inflammation.

Key to dental pain mechanisms are voltage-gated sodium channels (Na_v) that aid in the propagation of the signal through the length of the nerve. These channels have isoforms that are specially expressed on terminal nerve endings of sensory fibers.[79] The isoforms within the pulp are $Na_v1.7$ and $Na_v1.8$.[80] These isoforms are seen to be upregulated in painful teeth compared to normal pulps (Figure 7-5).[81] Their location along the nerve axon promotes faster conduction of an impulse from the termini to the central terminals thus sometimes mediating spontaneous and sharp shooting pain characteristic of pulpitis. Na_v can form clusters in the unmyelinated segments of myelinated axons, suggesting a possible role of these channels in mediating pain characteristic of irreversible pulpitis.[6] Moreover, inflammation-induced sensitization of Na_v channels by prostaglandins has also been implicated in pain from irreversible pulpitis as sensitization can allow for depolarization of nerve fibers at lower stimulus strengths than usual.[82]

Neural plasticity has been implicated in dental injury. Nerve fibers not only react by modifying gene expression but also change their cytochemical phenotype. Upon low intensity injury, primary afferent fibers increase in number within the dental pulp. This phenomenon has been termed a "sprouting."[83,84] The role of NGF in mediating this process has been implicated, as increase in pulpal NGF is seen following pulpal injury.[85] These effects appear to last as long as the stimulus is present. The neuronal architecture returns to normal soon after the stimulus is removed. However, under conditions of persistent inflammation or infection, sprouting can continue over long periods of time and may contribute to nociceptor sensitization. While this is conceivable, it is arguable. Wood et al.[86] showed preliminary data indicating that pulps collected from normal and cariously involved nonpainful teeth present significant differences in nerve density (Figure 7-6). Cariously involved nonpainful teeth

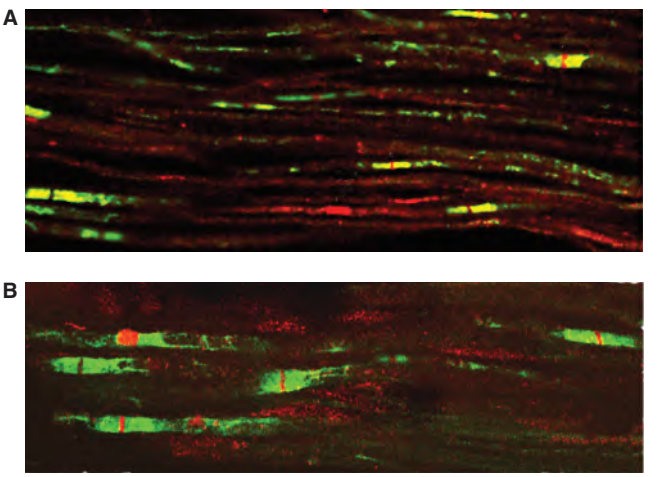

FIGURE 7-5 ***A*** and ***B.*** The $Na_v1.7$ and $Na_v1.8$ sodium channel isoforms (red) are prominently expressed within nerve fibers, located in the painful human dental pulp at both non-nodal and nodal locations. Nodes of Ranvier are identified by the paranodal staining of Caspr (green). (Courtesy of Dr. Michael A. Henry, San Antonio, TX, U.S.A.)

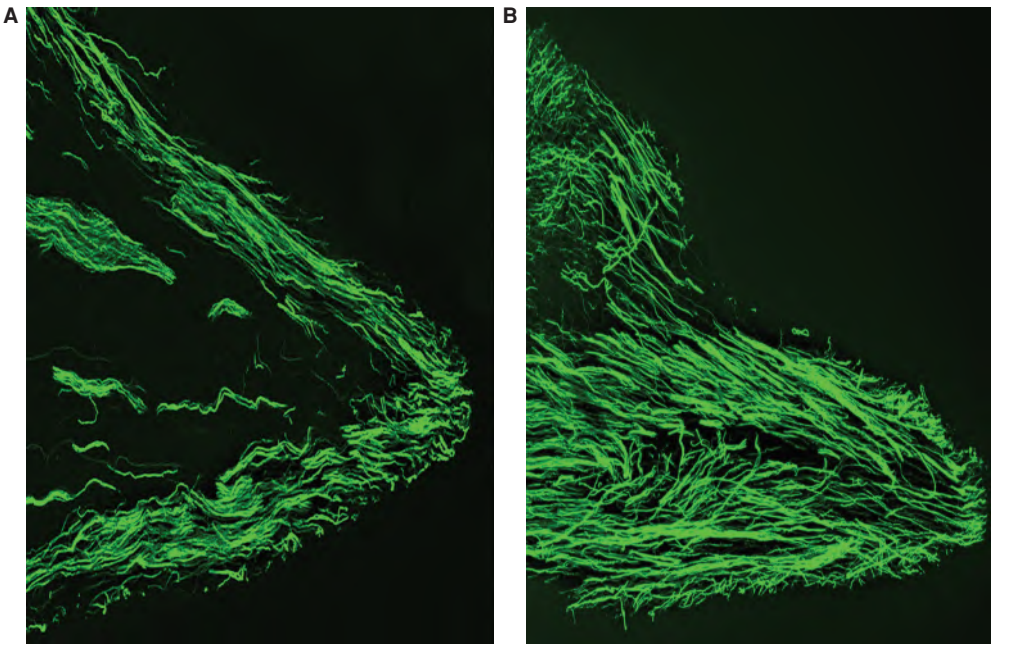

FIGURE 7-6 Nerve sprouting in carious nonpainful teeth. Confocal micrographs of nerve labeling (identified with N52/GAP43) within two different pulp horns, from the same human molar tooth, show a normal nerve distribution in the pulp horn lacking a carious lesion ***A.*** while extensive nerve sprouting is seen in the other pulp horn that was associated with an early carious lesion ***B.*** (Courtesy of Dr. Michael A. Henry, San Antonio, TX, U.S.A.)

have higher amounts of nerve sprouting compared to normal pulp, suggesting that sprouting may not directly correlate to increased pain. Nevertheless, sprouting may contribute to change in pulpal homeostasis, repair, and healing.

Central terminals of primary afferents, located in the dental pulp, are key in relaying peripherally detected stimuli to second-order neurons located within the medullary dorsal horn. Although the majority of peripheral tooth terminals terminate in nucleus caudalis, some also make connections on neurons located in nucleus oralis and interpolaris.[87–90] This may explain the inability of patients to localize pain originating from a tooth with symptomatic pulpitis. These second-order neurons or projection neurons are the primary centers of the majority of pain processing that occurs following peripheral injury.

Apart from the primary afferents, projection neurons also receive input from neighboring cells, such as interneurons, descending neurons, and glia. Upon peripheral tissue injury, central terminals of primary afferents release excitatory neurotransmitters, such as SP and glutamate. Glutamate has been studied extensively for its role in generation of hyperalgesia. Activation of NK1 for SP and NMDA and AMPA for glutamate on projection neurons causes excitation of these neurons that then send this signal via the trigeminothalamic tract to the thalamus from where the signal travels via the thalamocortical tract to the somatosensory cortex.

Although seemingly simple, the integration of input at the projection neuronal level, under conditions of inflammation, is quite complicated. Inflammation-evoked persistent activation of C-fiber input leads to sufficient intensity that renders the projection neurons in a state of constant activation. This establishes an area of synaptic plasticity. Other inputs from neighboring cells such as interneurons and glia can also modify this signal depending on the neurotransmitter they release. Moreover, larger fibers such as Aβ-fibers, which typically deliver information such as touch and proprioception from the periphery, are now primed under conditions of inflammation and contribute to amplification of this signal. Overall, these cellular pathways increase the receptive field, contribute to response mediated by non-noxious stimuli (allodynia), increase response to painful stimuli (hyperalgesia), and promote spontaneous pain. Together, the sensitization of the central projection neuron establishes the phenomenon of central sensitization.

Central sensitization is key to understanding some of the pain experienced by patients post treatment. Approximately 11% of patients continue to have pain seven days after endodontic treatment,[91] while 1.9% continue to have pain that remains unresolved for three to five years.[92] This suggests that despite achieving successful endodontic treatment as seen by clinical measures, persistent pain may exist. This may partly be explained by central sensitization that persists long after removal of the peripheral component. It is therefore conceivable that preoperative pain can be a strong predictor for possible postoperative pain.[93]

Taken together, dental pain mechanisms depend largely on the extent of pulpal injury and therefore on the severity of inflammation. Target receptors present on nociceptors, which show changes in their physical and molecular expression as well as central mechanisms leading to maintenance of a hyperalgesic state, contribute significantly to the overall symptoms of pain.

PAIN OF PULPAL ORIGIN

Activation of sensory neurons that innervate the pulp and periapical tissues commonly evokes a sensation of pain. The perception of "toothache" may result from activation of neurons innervating the pulp and periapical tissues as well as from central mechanisms of hyperalgesia. Experienced clinicians also find that, in some cases, the noxious input may not originate from the tooth the patient perceives to be associated with the pain. Instead, the pain may be referred from a distant site.

DENTIN HYPERSENSITIVITY

Dentin sensitivity is a common form of odontogenic pain and is described as a short, sharp pain from exposed dentin in response to thermal, tactile, osmotic, evaporative, or chemical stimuli, and that cannot be ascribed to any other pathosis or any form of dental defect.[94] The reported prevalence of dentinal hypersensitivity ranges from 3% to 98%.[95–99] The discrepancy in reported prevalence rates may be due to selection bias in the recruitment of study subjects, the use of different diagnostic criteria, and cultural factors.

Risk factors for dentin hypersensitivity include age, gingival recession, bleaching, periodontal treatment, abrasion, attrition, and anatomic defects.[100,101] Some studies report that the incidence of dentin hypersensitivity peaks in the third and fourth decades.[102] Gingival recession, erosion, and abfraction expose dentin tubules are likely to result in dentinal hypersensitivity. A recent study reported that the prevalence rate of dentin hypersensitivity was higher in subjects with gingival recession (PR 5.5; 95% CI 3.0–10.1).[103] The decrease in dentin hypersensitivity after the fourth decade may be due to the formation of tertiary dentin or loss of pulp vitality.

Several mechanisms have been proposed to explain dentinal hypersensitivity (see Chapter II, Figure 2-5). The neural theory proposes that neurons innervating the dentinal tubules are directly activated by stimuli. The hydrodynamic theory, the most widely accepted theory, proposes that movement of fluid in the dentinal microtubules generated by mechanical, thermal, or chemical forces activates nerve fibers, resulting in pain.[104] Movement of dentinal fluid induces shear stress that may, in turn, activate mechanosensitive ion channels, such as TREK1, TREK2, and ASICS3.[73] The direction of movement of dentinal fluid can be both inward (toward the pulp) and outward (away from the pulp). Cold stimuli elicit an outward flow and more rapid transient pain sensations, while hot stimuli elicit an inward flow.

The odontoblast transduction theory proposes that odontoblasts function as sensory receptors that transduce external stimuli and then transmit them to nerves terminals in the dentin or pulp. Odontoblasts are known to express cell surface receptors and ion channels, including TRPV1, TRPV2, TRPV4, TRPA1, TPRM3, and Ca and K channels,

thus suggesting that these cells can potentially function as sensory receptors.[105–111]

MANAGEMENT OF DENTIN HYPERSENSITIVITY

Treatment of dentin hypersensitivity usually takes one of the two approaches; reduction in dentinal fluid flow or reduction in activity of neurons innervating the dentinal neurons. Agents such as sodium fluoride, stannous fluoride, strontium chloride, potassium oxalate, calcium phosphate, calcium carbonate, and bioactive glasses reduce fluid flow by plugging dentinal tubules. Dentin adhesive sealers, including fluoride varnishes, oxalic acid and resin, glass ionomer cements, composites, and dentin bonding agents, occlude the tubules, resulting in attenuation of dentin hypersensitivity.

Neural modulating agents such as potassium nitrate are thought to act by depolarizing the nerves and preventing repolarization. A meta-analysis compared the effect of potassium containing tooth pastes to that of nonpotassium-containing tooth pastes on dentin hypersensitivity ($N = 390$).[111] The results showed that the use of potassium-containing tooth pastes results in a significant decrease in mean sensitivity to tactile, air blast, and thermal stimulation but not in subjective assessment.

PULPITIS

Inflammation of the pulp (and periapical tissues) is commonly associated with pain constituting ~90% of emergency visits with tooth pain as the chief complaint. This pain is attributed to activation of pulpal or periapical nociceptors.[112] Microbial infections of the pulp trigger an immune response designed to limit infection, signal injury, and promote repair. A plethora of immune mediators are released that trigger pain, inflammation, and, in advanced stages, pulpal necrosis. As the pulp becomes inflamed and transitions from a stage of normal to reversible pulpitis to irreversible pulpitis, patients often experience spontaneous or evoked pain.

Some patients with irreversible pulpitis present spontaneous pain. Descriptors used by patients to describe the quality of their pain are important clues that help differentiate odontogenic pain from pain referred from other sites. For more details, see Chapter XVII.

A study comparing the quality of pain in patient with odontalgia ($n = 359$) to those with craniofacial pain referred from cardiac origin ($n = 115$) showed that the quality descriptors, such as aching, burning, pressure, and throbbing, have high validity ($P = 0.53$) for differentiating between odontogenic and cardiac pain.[113]

While the descriptor "throbbing pain" is commonly associated with odontogenic pain, the underlying mechanisms are yet to be understood. It was previously thought that the sensation of throbbing was caused by the rhythmic activation of pain sensory neurons in close approximation to blood vessels. An elegant study on the relationship between throbbing pain and arterial pulsations in odontogenic pain patients showed that the throbbing rate (44 bpm ± 3 standard error of mean [SEM]) was much slower than the arterial pulsation rate (73 bpm ± 2 SEM) and, that there was no synchronicity between the two rhythms ($P < 0.001$) (Figure 7-7).[114] Therefore, it appears that the physiological mechanisms underlying these two rhythmic events are distinct.

Thermal hyperalgesia is considered a hallmark of symptomatic irreversible pulpitis pain. Several studies have examined the expression of thermoreceptors and ion channels in the dental pulp in order to better understand the mechanisms underlying thermal hyperalgesia in pulpitis. For example, studies on TRP receptors show that odontoblasts express TRPV1–3, TRPA1, and TRPM8[108,115]; dental pulp fibroblasts express TRPA1, TRPM2, and TRPM8[46]; and axons innervating the dental pulp express TRPA1, TRPM2, TRPM8, and TRPV1. Preclinical studies and *ex vivo* studies clearly show that these receptors are functional and that they are sensitized in the presence of infection (such as LPS) and inflammatory mediators.[43,69,116] However, data generated from clinical studies comparing pulps collected from patients with symptomatic pulpitis and normal pulps have yielded conflicting results. TRPM2

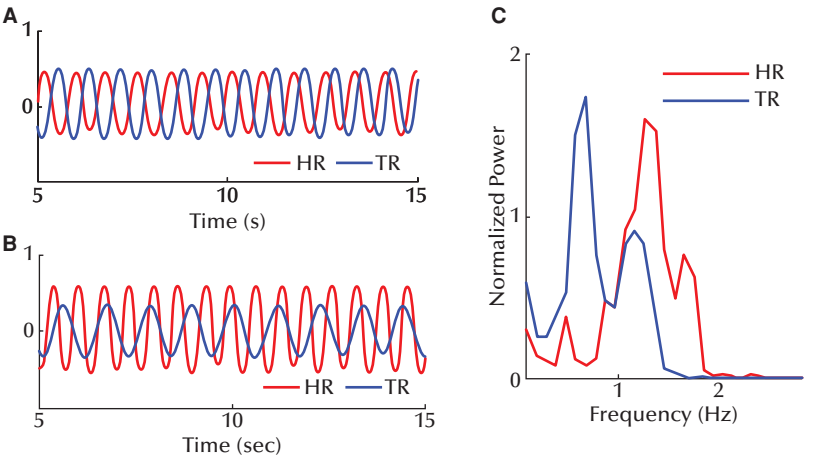

FIGURE 7-7 Spectral analysis of the rate of throbbing rhythm (TR) and the arterial pulse rate (HR) in odontogenic pain patients. ***A*** and ***B.*** show the superimposed and smoothed waveforms of TR and HR from two representative subjects. ***C.*** Shows the average normalized power spectral from all subjects. (Reproduced with permission from Mirza et al.[126])

expression is increased in fibroblasts but not in axons innervating symptomatic human dental pulps.[117] Axonal expression of TRPM8 is decreased (not as expected) in inflamed cold-sensitive pulpitis samples as compared to normal pulps.[118]

Mechanical Allodynia is a common finding in patients presenting with signs and symptoms consistent with irreversible pulpitis.[119] Several hypotheses have been proposed to explain the concurrent presentation of mechanical allodynia with irreversible pulpitis (Figure 7-8). One hypothesis is that mechanical allodynia is mediated by mechanoreceptive neurons innervating the pulp. This is supported by the findings that odontoblasts express the mechanoreceptors

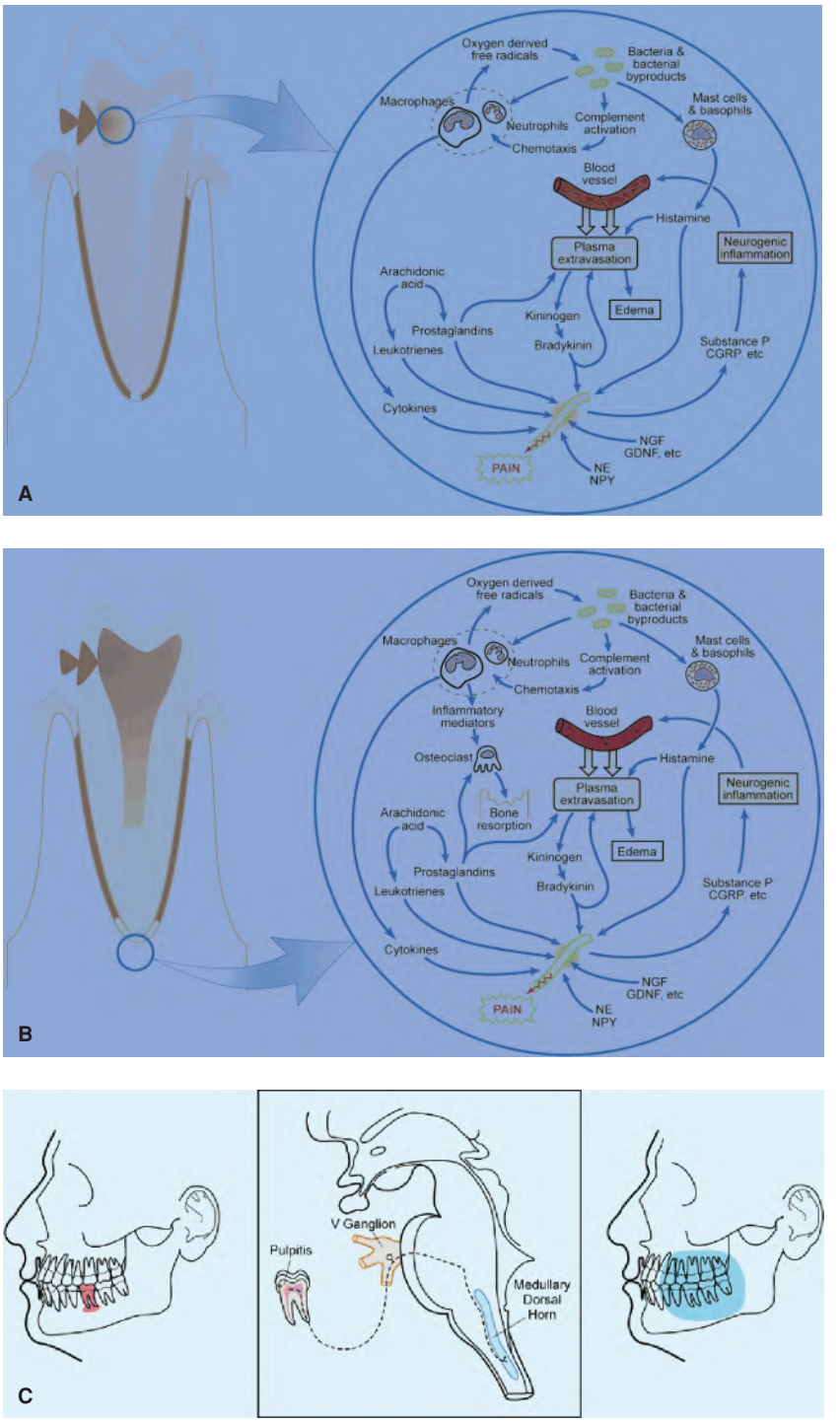

FIGURE 7-8 Diagrammatic representation of three hypotheses proposed to explain the incidence of mechanical allodynia is teeth with inflamed pulps. ***A.*** Microbial byproducts and inflammatory mediators in the pulp activate mechanoreceptors. ***B.*** Microbial byproducts and inflammatory mediators from the pulp diffuse into the periapical tissues and activate periradicular mechanoreceptors. ***C.*** Afferent barrage from pulpal nociceptors induces central sensitization resulting in expansion of receptor fields and mechanical allodynia. (Reproduced with permission from Owatz et al.[119])

TRPV4 and TRPM3 and that pulpal afferent A-fibers respond to mechanical stimulation.[70] Given that not all patients with symptomatic irreversible pulpitis present with mechanical allodynia, it is unlikely that the mechanical allodynia in odontogenic pain patients is activated via sensitization of pulpal mechanoreceptors.

A second theory is that inflammatory mediators and microbial byproducts from inflamed pulps diffuse into the periradicular space and activate or sensitize mechanoreceptors in the periodontal ligament. A recent study, comparing cone beam computerized tomography (CBCT) and periapical radiographs, detected periapical radiolucencies ≥1 mm in diameter in the CBCT images of almost 20% of teeth with vital pulps and no detectable radiolucencies in the periapical radiographs.[120] Another study found that 13.7% of teeth diagnosed with irreversible pulpitis had periapical lesions visible on CBCT as compared to only 3.3% on periapical radiographs.[121] This, coupled with histologic findings that periapical changes occur in teeth with vital pulps (prior to complete pulpal necrosis), support the hypothesis that the mechanical allodynia is teeth with irreversible pulpitis is due to activation of mechanoreceptors in the periodontal ligament.

A third hypothesis is that the mechanical allodynia results from central sensitization following activation of pulpal nociceptors. This is supported by preclinical studies showing that acute sensitization of TRPA1, expressing pulpal nociceptors, induces mechanical allodynia at distant sites.[122] A clinical study found that patients diagnosed with irreversible pulpitis and mechanical allodynia reported higher spontaneous pain as compared to those with irreversible pulpitis and normal periapical tissues.[119] Interestingly, the mechanical pain thresholds of the normal contralateral teeth in patients with irreversible pulpitis were lower than those in asymptomatic patients. Thus, there is both clinical and preclinical evidence supporting central sensitization in patients with irreversible pulpitis and mechanical allodynia.

REFERRED PAIN

Referred pain is a sensation felt in a site innervated by a nerve different from the one innervating the area from which the pain originates. Referred pain in the orofacial tissues is not uncommon. A systematic review and meta-analysis of nonodontogenic pain after endodontic therapy found that nonodontogenic factors accounted for 56% of dentoalveolar pain, present for ≥6 months after endodontic therapy.[123] A nested case series of patients (n = 19) with persistent tooth pain for >6 months after endodontic therapy found that nonodontogenic pain accounted for 42% of all cases.[123] The most common nonodontogenic reason was myofascial temporomandibular disorders with involvement of the masseter, temporalis, and lateral pterygoid muscles.

Pain referral commonly occurs in a laminated manner that reflects the order in which the nociceptors enter the spinal trigeminal tract. For more details, see Chapter XVII. Referred pain may present a diagnostic dilemma. The source of pain is usually identified by taking two approaches. The first is to elicit the pain (i.e., reproduce the chief complaint). Stimulation at the source elicits the pain while stimulation at the site of perception does not. The other approach is to block the pain using local anesthetics.[124] The prudent clinician appreciates the importance of listening to the patients' pain history, using appropriate diagnostic tests and replicating the chief complaint in order to arrive at the right diagnosis.

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Chapter 8

Examination and Diagnosis of Pulp, Root Canal, and Periapical/Periradicular Conditions

Paul V. Abbott

The first, and the most important, step in managing any condition is to determine what the condition is, that is, it must be diagnosed. In addition, the causes of the problem must be identified. Once this information has been gathered, the appropriate options for managing the problem will become obvious. This applies as much to diseases and conditions involving the dental pulp, the root canal system, and the periradicular tissues as it does to any other disease or condition of the human body. Hence, the importance of diagnosis must not be forgotten or downplayed at any time. However, unfortunately, many clinicians do not take enough time to establish a meaningful or correct diagnosis and they often proceed immediately to providing some form of treatment. This approach may work in some cases, but it is akin to a guess and inevitably some guesses will be incorrect, which in turn can lead to inappropriate management or treatment with potentially adverse outcomes for the patient. This chapter outlines the various pulp, root canal, and periradicular conditions and provides a detailed, yet simplified, approach to the diagnosis of these conditions. In most situations, the term *"condition"* is used rather than *"disease"* since not every state of these tissues is a disease process or an indication of pathosis of the tooth and surrounding tissues. The term *"disease"* will be used where there is clearly a disease process occurring.

THE IMPORTANCE OF DIAGNOSIS

When assessing any disease or condition, it is essential for the clinician to know

- What the condition is,
- Whether it should be treated,
- When it should be treated,
- Why it should be treated, and
- How it should be treated.

Diagnosing the condition, in conjunction with having a thorough understanding of the disease processes, will provide the abovementioned information. In particular, the diagnosis will indicate the management options available for the condition as it can be considered that "*Diagnosis Drives Dentistry*." The diagnosis will also determine the details of the treatment required.

There are several management options for each of the various pulp, root canal, and periradicular conditions that affect teeth. The option chosen needs to be carefully considered and for each particular case and situation. The possible endodontic management options for teeth with pulp, root canal, and periapical/periradicular conditions usually include one or more of the following, depending on the diagnosis of the specific presenting problem:

- No treatment—but review and reassess.
- Restoration of the tooth (or replacement of the existing restoration).
- Conservative pulp treatment—such as indirect or direct pulp capping, partial pulpotomy, pulpotomy, or partial pulpectomy.
- Pulpectomy and root canal treatment.
- Root canal treatment of pulpless teeth.
- Root canal retreatment.
- Periapical surgery—or other surgical procedure(s).
- Systemic medications—such as nonsteroidal anti-inflammatory drugs (NSAID's), analgesics, antibiotics, and so forth.

The procedure most often performed in the practice of Endodontics is commonly called "root canal treatment." However, such treatment should not be the same for all cases, and it should vary according to the diagnosis. The mechanical processes (root canal instrumentation, root canal filling, etc.) are similar for all cases BUT the treatment needs and the biological objectives will vary depending on the diagnosis. Therefore, the management must vary. The typical variations include

- The cause of the disease and how it is removed.
- Some cases have a pulp to remove while others do not have a pulp.
- In some cases, the primary aim of the irrigants is to dissolve pulp tissue while in others it is to disinfect the root canal system.
- The medicament regime varies according to whether the aim is to reduce inflammation, destroy bacteria and/or encourage hard tissue repair.
- Some cases require drainage of pus while others have no pus to be drained.

BOX 8-1 Examples of how the diagnosis affects the details of how teeth with different pulp, root canal, and periapical/periradicular conditions are managed

Example 1

Diagnosis—Chronic reversible pulpitis as a result of toothbrush abrasion (often referred to as "sensitive dentin")

- Typical management—desensitisation of the dentin.

Example 2

Diagnosis—Acute reversible pulpitis as a result of caries

- Options for management—with the actual procedure chosen depending on clinical findings once the caries has been removed:
  - ◊ Indirect pulp cap
  - ◊ Direct pulp cap
  - ◊ Partial pulpotomy
  - ◊ Pulpotomy.

Example 3

Diagnosis—acute irreversible pulpitis as a result of caries

- Options for management:
  - ◊ Extraction
  - ◊ Pulpectomy and root canal treatment—with the following considerations/variations:
    - » Pulp tissue is present and needs to be removed
    - » Sodium hypochlorite is used to dissolve pulp tissue remnants
    - » Anti-inflammatory medicament should be used to reduce periapical nerve sprouting and neuropathic pain, e.g., a corticosteroid/antibiotic compound
    - » If the tooth also has acute apical periodontitis—consider postoperative systemic NSAID's and analgesics.

Example 4

Diagnosis—a pulpless and infected root canal system

- Options for management:
  - ◊ Extraction
  - ◊ Root canal treatment—with the following considerations/variations:
    - » There is no pulp tissue present
    - » Sodium hypochlorite is used as an antibacterial agent (i.e., tissue dissolution less important)
    - » Antibacterial medicament is required, e.g., calcium hydroxide
    - » If the tooth also has acute apical periodontitis—consider using a corticosteroid/antibiotic compound in conjunction with the calcium hydroxide to reduce periapical inflammation and pain.
    - » If the tooth also has an acute apical abscess—consider drainage; consider systemic antibiotics.

Some typical examples of the variations in management as a result of different diagnoses are outlined in Box 8-1.

In order to be able to diagnose any condition or disease, it is essential that the clinician has a good understanding of the condition itself. The discipline of Endodontics is concerned with the diagnosis and management of conditions and diseases of the dental pulp, the root canal system and the periapical/periradicular tissues. Hence, clinicians must have a thorough understanding of these tissues, their various conditions and the disease processes that occur within them.

The clinician must also recognize and understand that conditions and especially diseases of the dental pulp, the root canal system and the periapical/periradicular tissues are not static and that they progress through various stages of the overall disease processes. For example, when the dental pulp is irritated by microorganisms, it will progress though the various stages of inflammation (reversible pulpitis, irreversible pulpitis) and then necrosis if the microorganisms are not removed. Within a few months, the necrotic pulp tissue will be removed by the microorganisms so the root canal system then becomes pulpless and infected.[1] None of these conditions occur instantaneously; they will always progress from one to the next over a period of time (Figure 8-1). Similarly, the periapical tissues progress through various stages of inflammation (apical periodontitis), infections (abscess, cellulitis) and possibly cyst formation if the source of irritation is not removed.[2] The periapical disease process is a dynamic one and the tissues may alternate between various conditions on a regular basis (Figure 8-2), depending on the overall local environment and the patient's general health.[3] Hence, the symptoms that any particular patient will describe will depend on the current stage of the condition or disease processes when the patient presents to a dentist for assessment.

The pulp, root canal, and periapical/periradicular tissues are all inter-related and the conditions within the pulp or root canal have a direct effect on the periapical and periradicular tissues.

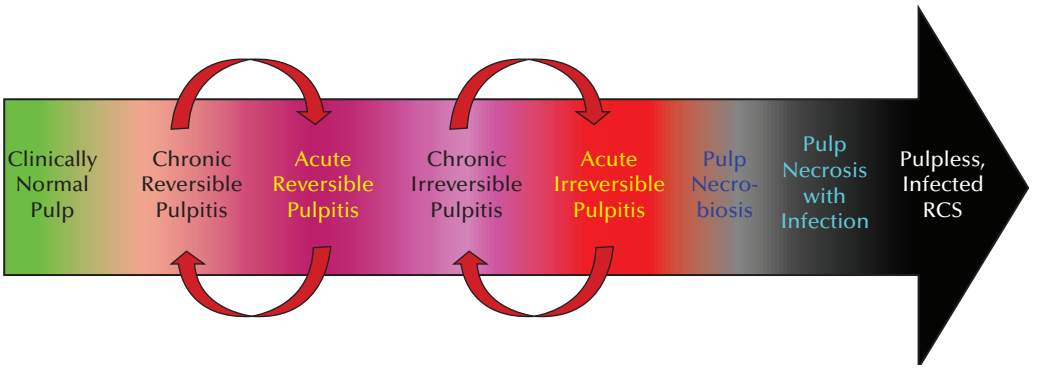

FIGURE 8-1 Schematic representation of the progression of pulp and root canal conditions through the various stages of the disease process.

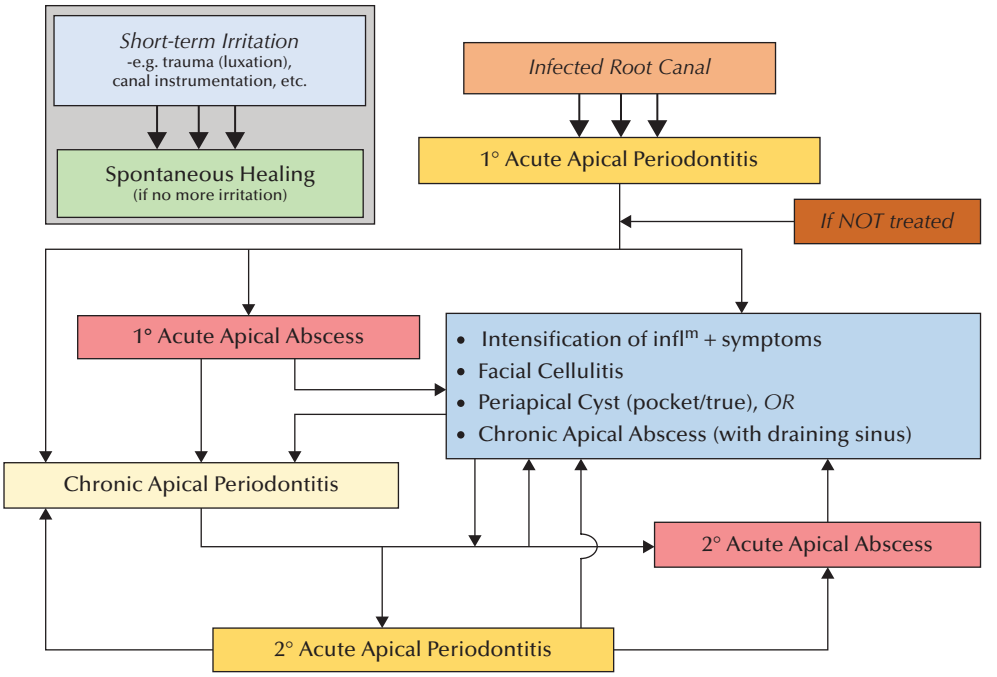

FIGURE 8-2 Schematic representation of the progression of periapical/periradicular conditions through the various stages of the disease process (adapted from Abbott[3]).

Hence, a thorough understanding of the interactions is also required. It is also essential to assess and diagnose the state of all tissues involved. When diagnosing endodontic conditions, there are four essential elements that must be determined.[4,5] These are the tooth involved, the state of the pulp (if present, otherwise the state of the root canal system), the state of the periapical/periradicular tissues, and the cause of the disease(s). If the condition of any tissue is missing, then the diagnosis is incomplete. Diagnosis is as much about assessing disease and abnormal conditions as it is about assessing the health of tissues. Hence, if one of the tissues is normal or healthy, then this should be stated as part of the diagnoses (see examples subsequently).

As mentioned earlier, not all teeth will have a pulp and therefore, these teeth cannot have the pulp assessed. Teeth that have become necrotic as a result of microbial invasion in the tooth will rapidly become pulpless and infected.[1] The tendency among many clinicians is to describe these teeth as having a "necrotic pulp" but this is inaccurate since there is no necrotic pulp tissue left in the root canal system. In any case, a necrotic pulp in itself does not cause apical periodontitis and therefore does not require root canal treatment. It is only when the necrotic pulp becomes infected that apical periodontitis occurs, and then root canal treatment is indicated.[6–10]

CLASSIFICATION OF CONDITIONS/DISEASES

The act of making a diagnosis is to determine what condition is present—in other words, to determine whether the tissues being assessed are normal or affected by a disease process. In essence, the final step of allocating a name (i.e., "the diagnosis") to any condition is one of summarising a

set of symptoms, clinical signs, test results, and radiographic observations into a few words. Hence, the words or terminology used must actually be possible to use in a clinical setting, meaningful with respect to what is happening in the tissues, and useful by leading to the management options. They should also be clear to all clinicians, and ideally used universally.[11] Unfortunately, this is not the case in the endodontic or dental literature when referring to pulp, root canal, and periapical/periradicular conditions. Many different classifications have been suggested over the years with virtually every textbook and many journal papers having their own classification. Likewise, many professional organizations and teaching institutions have their own classifications. Abbott and Yu[11] summarised 13 different classifications of pulp and root canal conditions used by various organizations and in textbooks. It is clear that there is no consistency between these classifications or the terminology used. Most of the classifications are either too simple (and miss many conditions) or too complex (and cannot be easily used in a clinical setting), and most of the classifications do not include all of the conditions that can occur. The end result is that there is no standardisation in terminology and this ultimately leads to confusion among clinicians. When the requirements for a clinical classification of diseases are considered, it is clear that most classifications of pulp, root canal, and periapical/periradicular conditions in the dental literature are inadequate, as they do not meet these criteria.

Another confounding factor in the diagnosis of pulp conditions has been the long-held belief by clinicians and researchers that there is no correlation between histological findings and the clinical presentation of pulp conditions. This belief was based on studies reported in the 1960s and 1970s[11–16] and it has led to confusion among clinicians and the subsequent use of many poor and inappropriate terms for the diagnosis of pulp conditions. However, Ricucci et al.[17] have reported that there is good correlation between the histological and clinical diagnoses as 96.6% of cases clinically diagnosed with either a normal pulp or reversible pulpitis had the same histologic diagnosis, and 84.4% of teeth clinically diagnosed with irreversible pulpitis had the same histologic diagnosis. In light of these findings, the old beliefs need to be reconsidered.

It is standard practice in medicine and dentistry to use the terms "acute" and "chronic" in a clinical sense without implying the histological response that may be occurring in the tissues.[11] For example, medical practitioners may diagnose "acute appendicitis" or "chronic appendicitis," depending on the severity of the presenting condition and the time that it has been present. Likewise, the dental profession uses terms such as chronic periodontal disease, acute ulcerative gingivitis, acute pericoronitis, and so forth. In endodontics, these terms have been used for many years but in more recent times, there has been a tendency toward not using them and instead to use "asymptomatic" and "symptomatic" even though they are inappropriate. As stated by Gutmann et al.,[18] there is "little, if any, support in the historic or contemporary peer-reviewed literature for the use of these terms," and they "have slowly crept into usage with little scientific basis for their applications or meanings."

Medical and dental dictionaries define "acute" and "chronic" for clinical use as follows:

- **Acute**—of or relating to a disease or a condition with a rapid onset and a short, severe course.[19] The pain is usually moderate to severe and the patient is typically seeking relief of the symptoms.
- **Chronic**—of long duration, used for a disease of slow progress and long continuance.[20] The pain associated with chronic conditions is usually mild and only occasionally present (i.e., "comes and goes"). The patient may only mention the problem in passing at a routine examination.

Hence, it is possible to use "acute" and "chronic" as clinical terms as long as the appropriate definitions are applied. These terms are appropriate for conditions of the pulp, root canal, and periapical/periradicular tissues. Hence, they will be used throughout this chapter and clinicians are encouraged to use them in their clinical practice in order to be consistent with other fields of dentistry and medicine.

Each pulp, root canal, and periapical/periradicular condition will have its own typical symptoms, clinical findings, and radiographic appearance that lead to the diagnosis. In addition, each pulp and root canal condition will have one or more typical associated periapical/periradicular conditions, and vice versa. Some conditions may appear to be similar to other conditions but there are key findings from the examination that allow clinicians to differentiate between them.

Abbott and Yu[11] and Abbott[5] have developed clinical classifications (Tables 8-1 and 8-2) for pulp, root canal, and periapical/periradicular conditions that meet the criteria for classifications, that is, they are possible to use in a clinical setting, meaningful with respect to what is happening in the tissues, and useful by leading to the management options. These classifications and each condition are outlined subsequently.

PULP AND ROOT CANAL CONDITIONS

This section describes each pulp and root canal condition listed in Table 8-1 to guide clinicians in the diagnostic process with a brief description of the typical symptoms reported by the patient, the typical clinical findings found on examination and testing of the tooth, the typical radiographic observations seen on periapical radiographs, the typical periapical/periradicular conditions associated with each pulp/root canal condition, the key findings that lead to the particular diagnosis, and the findings that distinguish each condition from other similar conditions.

The various forms of internal resorption will not be discussed in this chapter as they are discussed in detail elsewhere in this book.

TABLE 8-1 Classification of the Conditions of the Pulp and Root Canal System—See Text for a Description of the Typical Symptoms, Signs, and So Forth of Each Condition

No signs of disease:
- **Clinically normal pulp** *(based on history, clinical examination, tests, radiographs, etc.)*

Pulpitis:
- **Reversible pulpitis - acute***
  - chronic*
- **Irreversible pulpitis - acute***
  - chronic*

** Notes:*
- **Acute**—rapid onset and a short, severe course; moderate to severe pain; patient is seeking urgent relief of the symptoms.
- **Chronic**—long-standing; no pain or only mild and occasional pain; patient not seeking urgent relief.

Pulp necrosis:
- **Pulp necrobiosis** *(part of pulp is necrotic and infected; the rest is irreversibly inflamed)*
- **Pulp necrosis with NO signs of infection**
- **Necrotic and infected pulp**

Pulpless teeth:
- **Pulpless and infected root canal system**
- **Teeth with previous root canal treatment**
  - ◊ **Root-filled with NO signs of infection**
  - ◊ **Root-filled and infected root canal system**
  - ◊ **Incomplete endodontic treatment**** *(e.g., treatment started elsewhere)*
    *** Note: Must also specify whether the root canal system is infected or not*
  - ◊ **Technical standard** *(based on the radiographic appearance)*
    - » **Adequate**
    - » **Inadequate**
  - ◊ **Other problems**—*specify the problem: e.g., perforation, untreated canal(s), fractured instrument, silver point, etc.)*

Degenerative changes:
- **Pulp canal calcification**** *(based on radiographic appearance)*
  *** Note: Must also specify the diagnosis/condition of the pulp or root canal system*
- **Pulp hyperplasia** *(note: a form of irreversible pulpitis; specify whether acute or chronic)*

Note: the various forms of internal resorption are not included as these are discussed elsewhere in this book. (Adapted from Abbott[3] and Abbott and Yu.[11])

TABLE 8-2 Classification of the Conditions of the Periapical/Periradicular Tissues—See Text for a Description of the Typical Symptoms, Signs, and So Forth of Each Condition

No signs of disease:
- **Clinically normal periapical/periradicular tissues** *(based on the history, clinical examination, tests, radiographs, etc.)*

Inflammatory conditions:
- **Apical periodontitis - Acute: Primary** *(i.e., NO p-ap. radiolucency)*
  Secondary *(i.e., has a p-ap. radiolucency)*
  - Chronic *(i.e., has a radiolucency)*
  - Condensing osteitis *(i.e., radiopacity)*
  (Note: a form of chronic apical periodontitis)
- **Foreign body reaction**

Infections:
- **Apical abscess - Acute: Primary** *(i.e., NO p-ap. radiolucency)*
  Secondary *(i.e., has a p-ap. radiolucency)*
  - Chronic *(i.e., has a draining sinus)*
- **Facial cellulitis**
- **Extra-radicular infection**

Cysts:
- **Periapical pocket cyst**
- **Periapical true cyst**

Scar:
- **Periapical scar**

Notes: Definitions for acute and chronic are included in Table 1. The various forms of external resorption are not included as these are discussed elsewhere in this book. (Adapted from Abbott.[3,5])

Clinically Normal Pulp

The term "clinically normal pulp" is used for a pulp that has no signs or symptoms to suggest that any form of disease is occurring. The term "clinically" is used since such a pulp may not be histologically normal and/or may have some mild inflammation, fibrosis (scarring) as a result of previous injury or stimuli such as caries, restorations, trauma, and so forth. However, for all intents and purposes, the tooth does not have any pulp pathosis that requires treatment.

Typical symptoms—no symptoms.

Clinical findings—normal reaction to cold and electric pulp sensibility tests as long as there has been no calcification of the coronal pulp space; will not respond to heat; percussion and palpation are normal.

Radiographic findings—no radiographic signs of pathosis; normal periodontal ligament (PDL) space and lamina dura.

Associated periradicular conditions—clinically normal periapical tissues.

Key findings for diagnosis—no symptoms and no abnormal findings.

Pulpitis

Pulpitis can take several forms—it can be reversible or irreversible, and both of these stages of the disease process can

be chronic or acute in nature. Chronic reversible and chronic irreversible pulpitis are conditions that have been present for a reasonable length of time (typically weeks, months, or years) while acute reversible and acute irreversible pulpitis are recent conditions (typically only present for a few hours or days). The acute conditions have more intense pain than the corresponding chronic conditions. Patients with pulpitis typically complain of pain associated with temperature changes brought on by having hot or cold foods and drinks. It is most likely that pulps have chronic pulpitis for some time before the condition exacerbates and becomes acute.

Reversible pulpitis implies that the pulp is capable of healing following conservative management. Clinically, this cannot be accurately assessed but the diagnosis is based on the symptoms being relatively mild and requiring greater temperature change to stimulate the pain compared to irreversible pulpitis. Reversible pulpitis is effectively a provisional diagnosis that can only be confirmed following treatment and resolution of the symptoms with the pulp returning to a clinically normal state, as shown by a review examination and pulp sensibility tests.

Irreversible pulpitis implies that the pulp is not capable of healing following conservative management and therefore the pulp or the tooth needs to be removed. Clinically, it is not possible to determine how advanced the inflammation is, nor is it possible to determine whether the entire pulp is inflamed. In some cases, the inflammation may be limited to just part of the pulp but the pain is quite intense and suggests irreversible pulpitis. Despite the uncertainty at times, Ricucci et al.[17] have shown good correlation between the clinical and histological diagnoses of pulp conditions, as discussed previously.

Chronic Reversible Pulpitis

Typical symptoms—the pain has been present for a long time (e.g., months); occasional sensitivity to heat, cold and/or biting; pain only occurs with extreme temperature changes (e.g., ice cream and cold drink from the fridge); pain is sharp but mild; pain is of short duration (e.g., a few seconds); tooth may have had a restoration prior to the onset of symptoms.

Typical clinical findings—there may be caries, a restoration breaking down or a crack; thermal pulp sensibility tests can reproduce the pain associated with heat and/or cold stimuli; the pain with thermal testing will be sharp and of short duration; there may be pain on biting or tenderness to percussion on a specific cusp if a crack undermines the cusp; caries may be seen if extensive enough or the restoration may have signs of marginal breakdown.

Radiographic findings—normal PDL space and lamina dura; occasionally may be condensing osteitis; rarely, a slightly widened PDL space.

Associated periapical/periradicular conditions—usually clinically normal periapical tissues; occasionally may have primary acute apical periodontitis or occasionally condensing osteitis indicating chronic reversible pulpitis and chronic apical periodontitis for some time.

Key findings for diagnosis—the nature of the pain (thermal sensitivity, sharp pain, short duration).

Distinguish from acute reversible pulpitis by the length of time the problem has been present and the occasional nature of the symptoms.

Distinguish from acute and chronic irreversible pulpitis by the short duration of the symptoms and the extreme temperature changes required to cause the pain.

Acute Reversible Pulpitis

Typical symptoms—the pain has only been present for a short time (e.g., a few hours or days); the pain is present every time the stimulus is applied to the tooth; there is sensitivity to heat, cold and/or biting; pain only occurs with extreme temperature changes (e.g., ice cream and cold drink from the fridge); pain is sharp but mild and of short duration (e.g., a few seconds); may have had a restoration prior to onset of symptoms.

Clinical findings—may be caries, a restoration breaking down or a crack; thermal pulp sensibility tests can reproduce the pain associated with heat and/or cold stimuli; pain with thermal testing will be sharp, and of short duration; may be pain on biting or tenderness to percussion on a specific cusp if a crack undermines the cusp; caries may be seen if extensive enough or restoration may have signs of marginal breakdown.

Radiographic findings—normal PDL space and lamina dura; occasionally may have condensing osteitis; rarely, a slightly widened PDL space may be evident.

Associated periapical/periradicular conditions—usually clinically normal periapical tissues; occasionally may have primary acute apical periodontitis; occasionally condensing osteitis indicating chronic reversible pulpitis and apical periodontitis for some time before acute presentation.

Key findings for diagnosis—the nature of the pain (thermal sensitivity, sharp pain, short duration).

Distinguish from chronic reversible pulpitis by the length of time the problem has been present and the regular nature of the symptoms.

Distinguish from acute irreversible pulpitis by the short duration of the symptoms and the extreme temperature changes required to cause pain.

Chronic Irreversible Pulpitis

Typical symptoms—the pain has been present for a long time (e.g., months); occasionally pain to heat, cold and/or biting; pain occurs with minor temperature changes (e.g., tap water); pain is sharp and severe, then becomes a dull ache; pain lingers (e.g., more than five minutes); may have had a restoration prior to onset of symptoms.

Clinical findings—may be caries, a restoration breaking down or a crack; thermal pulp sensibility tests can reproduce the pain associated with heat and/or cold stimuli; pain with thermal testing will be sharp, painful and lingers; may be pain on biting or tenderness to percussion on a specific cusp if a crack undermines the cusp.

Radiographic findings—caries may be seen if extensive enough; normal PDL space and lamina dura; occasionally condensing osteitis; a slightly widened PDL space may be evident.

Associated periapical/periradicular conditions—usually clinically normal periapical tissues; occasionally may have primary acute apical periodontitis; occasionally condensing osteitis indicating chronic reversible or irreversible pulpitis and apical periodontitis for some time.

Key findings for diagnosis—the nature of the pain (thermal sensitivity, sharp pain, then a dull ache that lingers).

Distinguish from acute irreversible pulpitis by the long time that the pain has been present and the occasional nature of the symptoms.

Distinguish from acute reversible pulpitis and *chronic reversible pulpitis* by the lingering pain, and only minor temperature changes required to cause the pain.

Acute Irreversible Pulpitis

Typical symptoms—the pain has only been present for a short time (e.g., a few days or less); pain to heat, cold and/or biting; pain occurs with minor temperature changes (e.g., tap water); pain is sharp and severe, then becomes a dull ache; pain lingers (e.g., more than five minutes); pain may be spontaneous; pain may occur when lying down; pain may wake the patient at night; may have had a recent restoration.

Clinical findings—may be caries, a restoration breaking down or a crack; thermal pulp sensibility tests can reproduce the pain associated with heat and/or cold stimuli; pain with thermal testing will be sharp, very painful and lingers; may be pain on biting or tenderness to percussion on a specific cusp if a crack undermines the cusp.

Radiographic findings—caries may be seen if extensive enough; normal PDL space and lamina dura; occasionally condensing osteitis; a slightly widened PDL space may be evident.

Associated periapical/periradicular conditions—often have primary acute apical periodontitis; occasionally condensing osteitis indicating chronic apical periodontitis for some time prior to the acute presentation; sometimes clinically normal periapical tissues (e.g., if no tenderness to percussion).

Key findings for diagnosis—the nature of the pain (thermal sensitivity, sharp and severe pain, then a dull ache that lingers). Spontaneous pain, waking at night, and worse lying down are positive signs of acute irreversible pulpitis.

Distinguish from chronic irreversible pulpitis by the short time that the pain has been present and the regular nature of the symptoms.

Distinguish from acute and chronic reversible pulpitis by the severity and lingering pain, and only minor temperature changes required to cause pain.

Pulp Necrobiosis

A tooth with necrobiosis has both inflamed (acute irreversible pulpitis) and necrotic (usually infected) pulp tissue.[21] Many dentists use "partial necrosis" for this stage of the disease process. However, Grossman[21] suggested the term "necrobiosis" because it more accurately indicates the condition with *"necro-"* indicating necrosis and *"-bio-"* indicating live tissue. The necrotic tissue may be in the pulp chamber with the inflamed tissue in the root canal(s), or the different conditions may exist in different canals of a multicanal tooth. Teeth with this condition are difficult to diagnose since they often present with a mixture of the symptoms and signs of both pulpitis and necrosis with infection.[11] Typically, this stage of the disease process is only present for a short time—perhaps a few hours only or up to a few days.

Typical symptoms—the pain has only been present for a short time (e.g., a few days or less); typically history of very recent pain heat, cold and/or biting; if temperature changes cause pain then usually only minor temperature changes required (e.g., tap water); pain may be sharp and severe, then may become a dull ache; pain lingers for a long time; pain may be spontaneous; patient typically reports a mixture of symptoms suggesting pulpitis or an infected root canal system; pain can be quite distressing for the patient; may have had a recent restoration.

Clinical findings—may be caries, a restoration breaking down or a crack; pulp sensibility test results are mixed and frequently inconclusive or inconsistent with the patient's description of symptoms; often tenderness to percussion; may be pain on biting. Pain may be severe and may be relieved by "swishing" iced water around the tooth; relief lasts for seconds to a minute and may require constant application of cold water; a "bead" of pus may emerge when the pulp chamber is opened and this relieves the pain.

Radiographic findings—caries may be seen if extensive enough; usually have normal PDL space and lamina dura; sometimes a slightly widened PDL space may be evident.

Associated periapical/periradicular conditions—usually clinically normal periapical tissues (e.g., if no tenderness to percussion); sometimes have primary acute apical periodontitis (e.g., if tender to percussion).

Key findings for diagnosis—the patient's symptoms are often mixed (i.e., symptoms of pulpitis and an infected root canal system at the same time). The pulp sensibility tests usually do not correlate with the patient's symptoms, and the pain is often severe and distressing.

Distinguish from acute irreversible pulpitis by the mixed symptoms and inconsistency between symptoms and pulp sensibility tests.
Distinguish from acute and *chronic reversible pulpitis* by the nature of the pain and its severity, plus the pulp sensibility tests results.
Distinguish from necrotic and *infected pulp* by the nature of the pain and inconsistent symptoms.
Distinguish from a pulpless and *infected root canal system* as there is no periapical radiolucency.

Pulp Necrosis with No Signs of Infection

The dental pulp may undergo necrosis for several reasons. The most common reason is due to the bacterial invasion through caries, cracks or restoration breakdown that will ultimately lead to the pulp necrosing and becoming infected. However, some pulps may necrose as a result of trauma when the tooth is luxated or displaced sufficiently to sever the apical blood vessels. If the pulp cannot revascularize, then it necroses but does not necessarily become infected unless there is a coronal pathway of entry for bacteria. In cases of necrosis without infection, there will be no periapical tissue response[6–10]; that is, there will be no apical periodontitis (Figure 8-3).

The term used for this condition includes *"no sign of necrosis"* because it is not possible to categorically state that the tooth is *"not infected,"* based on the information obtained from a clinical and radiographic examination. It is known that radiographic signs of apical periodontitis (i.e., a radiolucency) take at least several months to be visible once a pulp becomes infected,[1] hence, the lack of a radiolucency does not necessarily indicate the lack of an infection. It merely means there was no sign of apical periodontitis (and hence no sign of root canal infection) at the time the radiograph was exposed.

Typical symptoms—no pulp-related symptoms (i.e., no thermal sensitivity); may have a history of trauma to the tooth.
Clinical findings—the tooth does not respond to pulp sensibility tests; may be some tooth discolouration; tooth may have a crack or fracture (e.g., if history of trauma), caries or a restoration.
Radiographic findings—no abnormal observations of the tooth unless trauma has caused a fracture; normal PDL space and lamina dura.
Associated periapical/periradicular conditions—clinically normal periapical tissues.
Key findings for diagnosis—no symptoms, no response to pulp sensibility tests but no other abnormal findings.
Distinguish from pulp necrobiosis as there are no symptoms and no signs of apical periodontitis.
Distinguish from a necrotic and infected pulp as there are no signs of apical periodontitis.

Necrotic and Infected Pulp

When a pulp necroses as a result of bacterial invasion of the tooth, it will become infected over time. The bacteria will remove the necrotic tissue quite rapidly (within one to two months, depending on whether or not the pulp is exposed to the mouth)[1] so the root canal system becomes "pulpless and infected." During the first few months following pulp necrosis, a periapical radiolucency will not be evident (Figure 8-4) as this takes several months more to manifest. Within these first few months, the pulp is still considered necrotic but there are signs that it is infected, as outlined subsequently. This stage of the disease process (i.e., necrotic and infected pulp) is only present for a very short period of time.

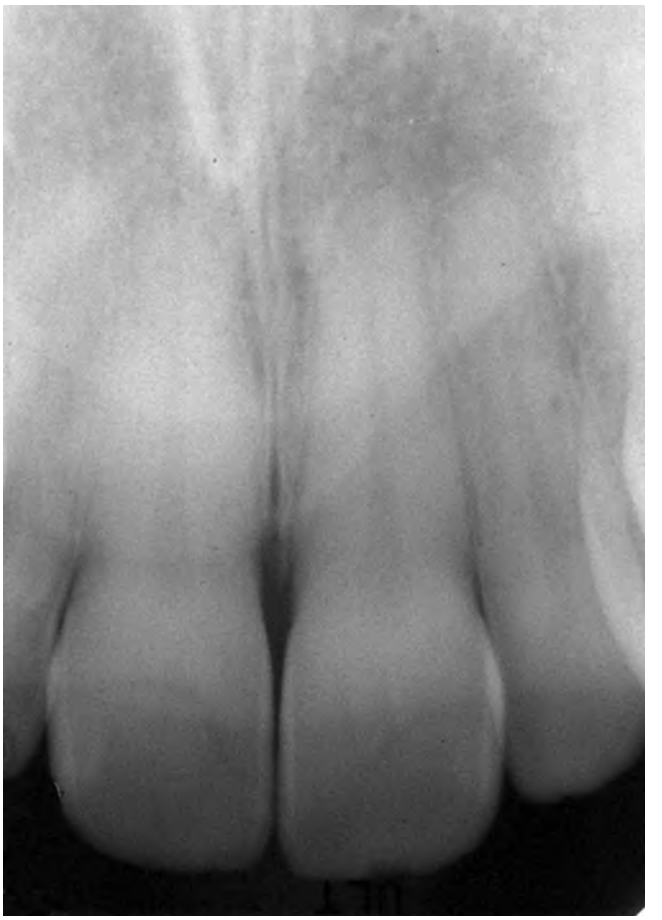

FIGURE 8-3 The maxillary left central incisor is likely to have pulp necrosis but there are no clear signs of the canal being infected since there are no periapical changes evident radiographically. This tooth had a history of trauma (extrusion) four years previously and the tooth was repositioned orthodontically after one week of being in the extruded position. The tooth does not respond to cold and electric pulp sensibility testing. It did not show any signs of blood flow when tested with a laser Dopplerflowmeter.

Typical symptoms—no pulp-related symptoms (i.e., no thermal sensitivity); may be no symptoms at all; any symptoms will be from the periapical tissues—hence, symptoms depend on the periapical status; may have pain associated with biting or pressure (depends on the periapical status); occasionally pain with heat that is temporarily relieved by applying very cold water/ice to the tooth.
Clinical findings—may be caries, a restoration breaking down or a crack; tooth does not respond to pulp sensibility tests; may be tender to percussion

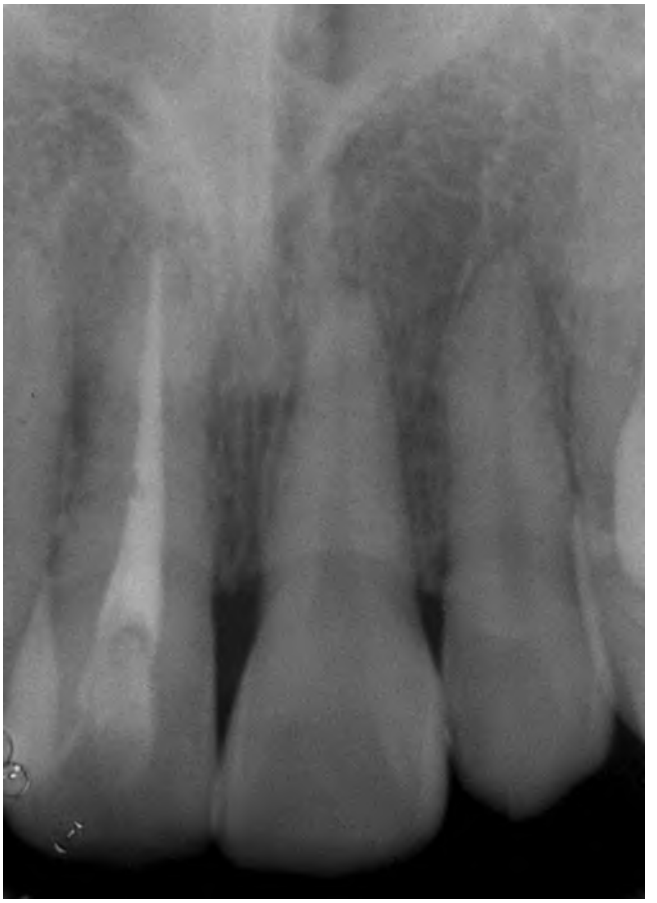

FIGURE 8-4 The maxillary left central incisor was laterally luxated and now has a necrotic and infected pulp with primary acute apical periodontitis.

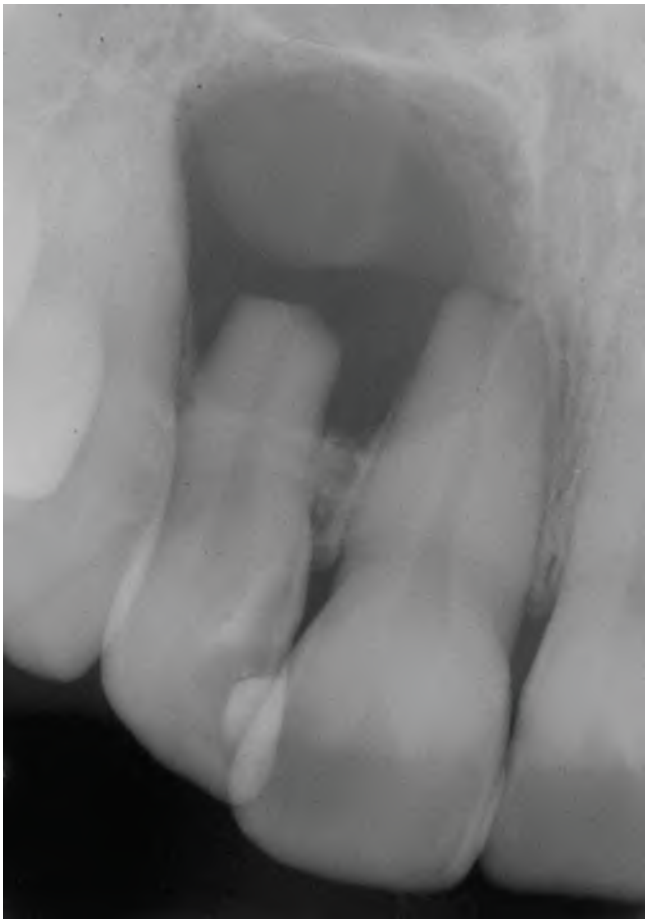

FIGURE 8-5 The maxillary right lateral incisor has a pulpless and infected root canal system with chronic apical periodontitis as a result of lateral luxation seven years previously. Note, there is also apical inflammatory resorption of the lateral incisor.

(depends on the periapical status); may be some tooth discolouration.

Radiographic findings—caries may be seen if extensive enough; usually have normal PDL space and lamina dura; sometimes a slightly widened PDL space and loss of lamina dura may be evident periapically.

Associated periapical/periradicular conditions—may have clinically normal periapical tissues (in the early stages); may have primary acute apical periodontitis (if pain and tender to percussion); may have a primary acute apical abscess (if pain, tender to percussion, swelling, and localized collection of pus).

Key findings for diagnosis—no response to pulp sensibility tests and no periapical changes seen radiographically.

Distinguish from pulp necrosis with no signs of infection by the symptoms and the apical periodontitis.

Distinguish from a pulpless and infected root canal system as there is no periapical radiolucency.

Pulpless and Infected Root Canal System

A root canal system that has a necrotic and infected pulp will rapidly (within one to two months) become pulpless and infected.[1] A periapical radiolucency will then develop over the next few months (Figure 8-5). Hence, once a periapical radiolucency is present, it is safe to assume that the root canal system is pulpless and infected—at this stage, it should no longer should be called a necrotic and infected pulp.

This condition should not be confused with a tooth that has had previous root canal treatment as specific terminology is used for those teeth (see subsequently). The term "pulpless and infected" is used to demonstrate that the tooth has no pulp because of the bacteria in the canal.

Typical symptoms—no pulp-related symptoms (i.e., no thermal sensitivity); may be no symptoms at all; any symptoms will be from the periapical tissues—hence, symptoms depend on the periapical status; may be an occasional dull ache or "awareness of tooth" if chronic apical periodontitis or a chronic apical abscess is present; will have pain associated with biting or pressure if secondary acute apical periodontitis or a secondary acute apical abscess is present.

Clinical findings—may be caries, a restoration breaking down or a crack; the tooth does not respond to pulp sensibility tests; may feel "different" on percussion if chronic periapical condition present; will be tender to

percussion if acute periapical condition present; may have increased mobility if acute periapical condition present; may be some tooth discolouration.

Radiographic findings—caries may be seen if extensive enough; a periapical radiolucency will be present.

Associated periapical/periradicular conditions—may have any of the following—depending on the symptoms, clinical and radiographic findings: chronic apical periodontitis (no symptoms or only occasional ache); a chronic apical abscess (if draining sinus present); secondary acute apical periodontitis (if pain and tenderness to percussion); a secondary acute apical abscess (if pain tender to percussion, swelling, and localized collection of pus), or facial cellulitis (if facial swelling due to spreading infection).

Key findings for diagnosis—no response to pulp sensibility tests and there will be a periapical radiolucency.

Distinguish from a necrotic and infected pulp by the presence of a periapical radiolucency.

Distinguish from pulp necrosis with no signs of infection as there is a periapical radiolucency.

Distinguish from a tooth with *previous endodontic treatment* as there is no history or radiographic evidence of previous treatment.

Previous Endodontic Treatment with No Signs of Infection

There are various procedures that can be grouped as "endodontic treatment"—essentially any procedure that involves treatment of the pulp or root canal system. These include direct pulp capping, partial pulpotomy, pulpotomy, partial pulpectomy, root canal filling, root canal retreatment, and the commencement of root canal treatment (i.e., commenced but not completed). Separate diagnostic categories for each type of treatment are not required but when assessing teeth with previous endodontic treatment, the nature of that treatment should be included in the diagnosis.

The term "no signs of infection" is used because it is not possible to categorically state that the tooth is "*not infected*," based on the information obtained from a clinical and radiographic examination. It is known that radiographic signs of apical periodontitis (i.e., a radiolucency) take at least several months to be visible once a pulp or root canal system becomes infected[1]—hence, the lack of a radiolucency does not necessarily indicate the lack of an intracanal infection. It merely means that there were no signs of apical periodontitis (and hence no sign of root canal infection) at the time the radiograph was exposed (Figure 8-6).

Typical symptoms—no symptoms; will have a history of some form of previous pulp/root canal treatment, such as: pulp capping, partial pulpotomy, pulpotomy, partial pulpectomy, root canal filling (on one or more occasions), or incomplete root canal treatment (e.g., if started as an emergency but not yet completed).

Clinical findings—the coronal restoration is intact and "clinically satisfactory"; no caries, no cracks or fractures of tooth; no abnormal findings except the tooth may or may not respond to pulp sensibility tests (depends on type of previous pulp treatment, e.g., may respond if previous pulp cap, partial pulpotomy, or pulpotomy) and there may be some tooth discolouration.

Radiographic findings—no caries evident; restoration appears satisfactory; no abnormal observations of tooth; normal PDL space and lamina dura; evidence of previous pulp treatment or root canal filling; if root canal filling present, it may or may not be technically satisfactory; other deficiencies or procedural errors may be evident in some cases but without associated radiolucency—for example: perforation, untreated canal, blocked canal, fractured file, incomplete root canal treatment, and so forth.

Associated periapical/periradicular conditions—clinically normal periapical tissues.

Key findings for diagnosis—history and/or radiographic evidence of previous pulp/root canal treatment, plus no symptoms, and no other abnormal findings.

Distinguish from previous endodontic treatment with an infected root canal system as there is no periapical radiolucency and no signs indicating apical periodontitis.

Distinguish from a pulpless, infected root canal system as there is a history or radiographic evidence of previous endodontic treatment.

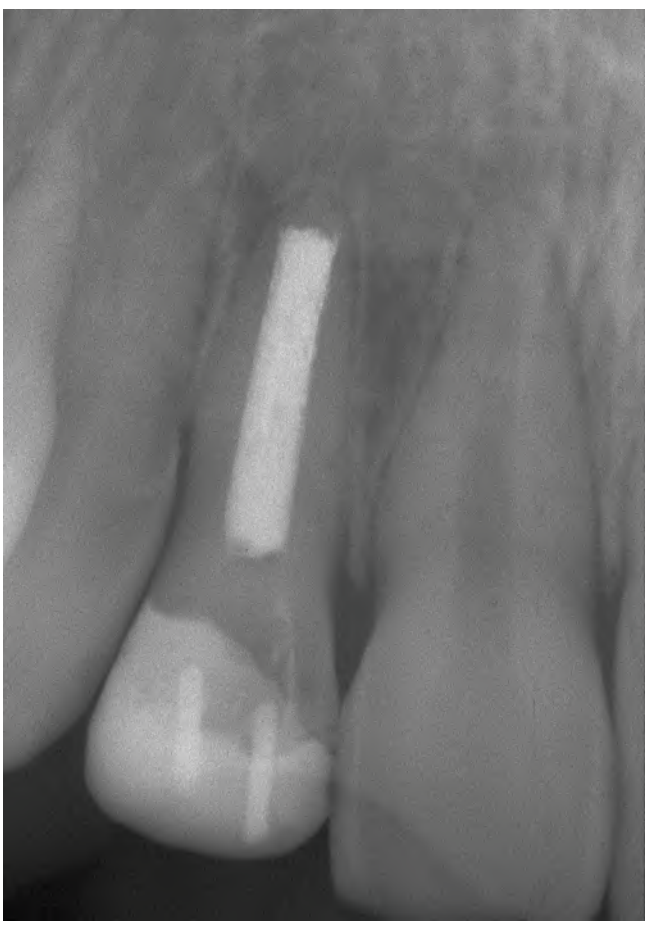

FIGURE 8-6 The maxillary right lateral incisor is a root-filled tooth following apexification with no signs of infection.

Previous Endodontic Treatment with an Infected Root Canal System

Once a tooth with previous endodontic treatment develops a periapical radiolucency, then the root canal system should be considered to be infected (Figure 8-7). The endodontic treatment could be any procedure that involves treatment of the pulp or root canal—these include direct pulp capping, partial pulpotomy, pulpotomy, partial pulpectomy, root canal filling, root canal retreatment, and the commencement of root canal treatment (i.e., commenced but not completed). Separate diagnostic categories for each type of treatment are not required but when assessing teeth with previous endodontic treatment, the nature of that treatment should be included in the diagnosis.

A tooth that has had root canal treatment commenced, but not completed, may still have a periapical radiolucency. This may represent a continuation of chronic apical periodontitis or the periapical tissues may be healing (but not completely healed). It is not possible to differentiate between these scenarios and hence it is safer to assume the root canal system may still be infected and to continue treating the tooth accordingly.

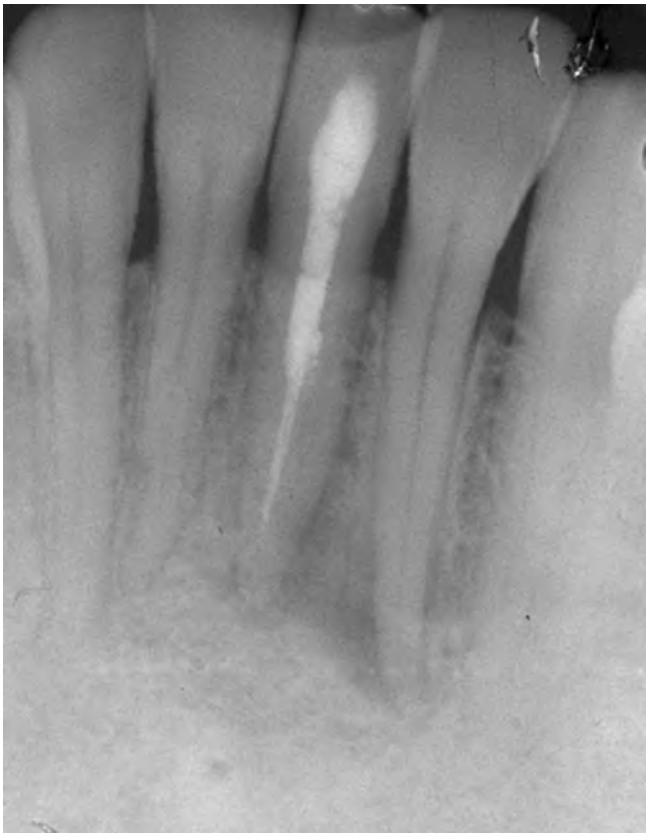

FIGURE 8-7 The mandibular right central incisor has a root-filled and infected root canal system with secondary acute apical periodontitis as a result of breakdown of the composite restoration and an unsatisfactory root canal filling. The patient had a continuous dull ache and the tooth was very sore to bite on. The radiolucency extends over the apex of the adjacent tooth root that has a normal PDL space and normal responses to pulp testing.

Typical symptoms—any symptoms will be from the periapical tissues—hence, symptoms depend on the periapical status; may be an occasional dull ache or "awareness of tooth" if chronic apical periodontitis or a chronic apical abscess is present; will have pain associated with biting or pressure if secondary acute apical periodontitis or a secondary acute apical abscess is present; will have a history of some form of previous pulp/root canal treatment, such as pulp capping, partial pulpotomy, pulpotomy, partial pulpectomy, root canal filling (on one or more occasions), or incomplete root canal treatment (e.g., if started as an emergency but not yet completed).

Clinical findings—may be caries, a restoration breaking down or a crack; the tooth does not respond to pulp sensibility tests; may feel "different" on percussion if chronic periapical condition present; will be tender to percussion if acute periapical condition present; may have increased mobility if acute periapical condition present; may be some tooth discolouration.

Radiographic findings—caries may be seen if extensive enough; a periapical radiolucency will be present; evidence of previous pulp treatment or root canal filling; if root canal filling present, it may or may not be technically satisfactory; other deficiencies or procedural errors may be evident in some cases and may have an associated radiolucency (e.g., perforation, untreated canal, blocked canal, fractured file, incomplete root canal treatment, etc.).

Associated periapical/periradicular conditions—may have any of the following depending on the symptoms, clinical and radiographic findings: chronic apical periodontitis (no symptoms or only occasional ache), a chronic apical abscess (if draining sinus present), secondary acute apical periodontitis (if pain and tenderness to percussion), a secondary acute apical abscess (if pain, tender to percussion, swelling, and localized collection of pus), or facial cellulitis (if facial swelling due to spreading infection).

Key findings for diagnosis—history and/or radiographic evidence of previous pulp/root canal treatment, plus the presence of a periapical radiolucency.

Distinguish from previous endodontic treatment without signs of infection as there is a periapical radiolucency.

Distinguish from a pulpless and infected root canal system as there is a history and/or radiographic evidence of previous endodontic treatment.

Pulp Atrophy

Atrophy of the pulp is a normal physiologic process that occurs with age. Clinically, it is difficult to distinguish whether this is present but it is likely in older patients. This condition does not require any specific treatment but it is important for clinicians to recognize that it does occur as it can affect the ability to diagnose the pulp status of some teeth.

Typical symptoms—no symptoms.
Clinical findings—usually no abnormal findings but the tooth may have a restoration; the tooth may or may not respond to pulp sensibility tests; the patient is usually elderly.
Radiographic findings—usually no abnormal observations of the tooth but it may have a restoration; some pulp canal calcification (PCC) may be evident; normal PDL space and lamina dura.
Associated periapical/periradicular conditions—clinically normal periapical tissues.
Key findings for diagnosis—no symptoms and no abnormal findings.

Pulp Canal Calcification

PCC could be considered as a radiographic observation of the appearance (especially the width) of the root canal(s) rather than as a distinct condition of the pulp or root canal system. It is not necessarily indicative of a disease process or pathological state of the tooth and therefore it is included as a "condition" rather than as a "disease." It is included in this classification of root canal conditions as its presence affects the management of the tooth—since it may affect the diagnostic process (e.g., pulp tests are less reliable) as well as the treatment (e.g., difficulty locating and negotiating the root canal). Clinicians should recognize that the pulp contained within a calcified canal can be normal or diseased, or the canal may be pulpless and infected, and so forth[11]—that is, all pulp and root canal conditions could be associated with PCC (Figure 8-8). Hence, the complete diagnosis of a tooth with PCC must include the diagnosis of the pulp or root canal condition as well as the PCC.[11]

The term "PCC" is the preferred term.[11] Some clinicians and authors use the term "pulp canal obliteration" but this is not accurate and hence not appropriate. The word "obliterate" is defined as "complete removal or destroying all traces of."[22] When a pulp undergoes calcification (defined as deposition of calcific material), it is not possible for the root canal to be completely removed or destroyed. There will always be a very small, narrow canal present even when the canal cannot be seen radiographically. The remaining canal may only be a few microns in width that is enough for several cells and it may or may not be possible to negotiate it with a root canal file; however, the canal still exists.[11] Hence, it is more appropriate to refer to the process as "PCC" as this accurately describes what is truly happening inside the tooth.

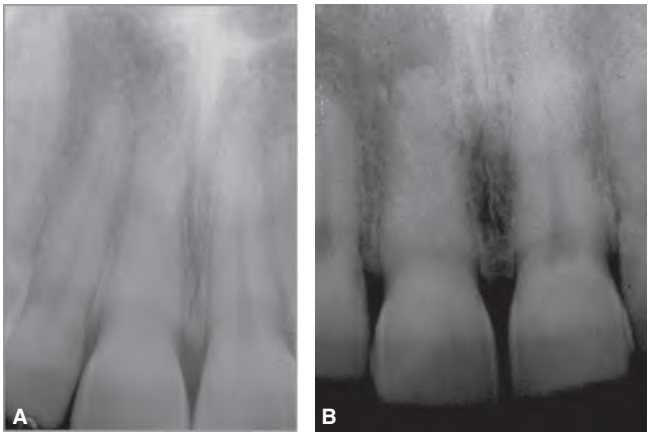

FIGURE 8-8 ***A.*** The maxillary right central incisor has pulp canal calcification. There was no response to CO_2 and electric pulp testing but there are no radiographic signs of any periapical changes; hence, there are no signs of the root canal system being infected and no treatment is required. ***B.*** The maxillary right central incisor has pulp canal calcification with a pulpless and infected root canal system and chronic apical periodontitis as a result of trauma 22 years previously.

Typical symptoms—symptoms will depend on the pulp or root canal condition—any of the following may be present with the typical symptoms as outlined earlier: clinically normal pulp, chronic reversible pulpitis, acute reversible pulpitis, chronic irreversible pulpitis, acute irreversible pulpitis, pulp necrobiosis, pulp necrosis with no signs of infection, necrotic and infected pulp, pulpless and infected root canal system, previous endodontic treatment with no signs of infection, previous endodontic treatment with an infected root canal system, pulp atrophy.
Clinical findings—clinical findings will depend on the pulp or root canal condition—with the typical clinical findings for each condition as outlined earlier; if a clinically normal pulp, then response to pulp sensibility testing may be variable, as follows: usually no response to a cold test, may or may not respond to an electric pulp test; if any form of pulpitis present, then the tooth should respond to cold pulp sensibility testing but some cases do not respond; the tooth should respond to an electric pulp test; if pulp necrosis, pulpless and infected or previous endodontic treatment, then there will be no response to pulp sensibility tests.
Radiographic findings—PCC is evident—this may be partial (vertically and/or horizontally) or it may involve the entire pulp space; other radiographic observations will depend on the pulp or root canal condition—with the typical radiographic observations for each condition as outlined earlier.
Associated periapical/periradicular conditions—this will depend on the pulp or root canal condition with the various possible periapical/periradicular condition(s) as outlined earlier for each particular pulp/root canal condition.
Key findings for diagnosis—radiographic evidence of PCC.
Distinguish from ***other conditions*** that do not respond to pulp sensibility tests by the symptoms, other clinical and radiographic findings, plus the associated periapical/periradicular condition(s).

Pulp Hyperplasia

Pulp hyperplasia almost exclusively occurs in young teeth with an abundant blood supply and a large carious lesion.[11]

It is essentially an overgrowth of granulation tissue and it appears as a polyp arising from the pulp. In some cases, it may appear to be connected to the gingival tissues.

Typical symptoms—often no symptoms; some cases may have pain when eating if the hyperplastic tissue is contacted by food, drinks, and so forth; some cases may bleed when the hyperplastic tissue is touched, brushed, and so forth; some cases will have the typical symptoms of pulpitis, as outlined earlier—the pulpitis can be chronic reversible pulpitis, acute reversible pulpitis, chronic irreversible pulpitis, or acute irreversible pulpitis.

Clinical findings—a "pulp polyp" is evident, within a large carious cavity where the pulp has been exposed by the caries; the pulp polyp may bleed on probing; the pulp polyp may or may not be sensitive to probing; the tooth will respond to pulp sensibility tests with the nature of the response depending on the type of pulpitis present, as outlined earlier; the pulp polyp will sometimes appear to be continuous with the gingival tissues if the cavity extends to the gingivae.

Radiographic findings—large carious lesion evident; other radiographic observations will depend on the pulp or root canal condition—with the typical radiographic observations for each condition as outlined earlier; as pulp hyperplasia is usually a long-standing problem, there is likely to be some minor widening of the PDL space or condensing osteitis, with both appearances indicating chronic apical periodontitis.

Associated periapical/periradicular conditions—the associated periapical/periradicular condition(s) will depend on the type of pulpitis present, as outlined earlier; typically there will be chronic apical periodontitis.

Key findings for diagnosis—clinical appearance of a "pulp polyp."

Distinguish from the ***various forms of pulpitis*** without hyperplasia by the presence of the pulp polyp.

Distinguish from ***all types of infected root canal systems*** by the symptoms, the clinical appearance of a pulp polyp and the response to pulp sensibility tests.

PERIAPICAL/PERIRADICULAR CONDITIONS

This section will describe each periapical/periradicular condition listed in Table 8-2 to guide clinicians in the diagnostic process with a brief description of the typical symptoms reported by the patient, the typical clinical findings found on examination and testing of the tooth, the typical radiographic observations seen on periapical radiographs, the typical pulp/root canal conditions associated with each periapical/periradicular condition, the key findings that lead to the particular diagnosis, and the findings that distinguish each condition from other similar conditions.

The term "periapical" is commonly used by most practitioners although "periradicular" is more accurate. Most periradicular conditions manifest in the periapical tissues because these tissues are adjacent to the apical foramen of the root canal(s). However, the same responses can manifest in the PDL and adjacent bone anywhere along the length of the root surface. Lateral canals are the same as the main canal in that they are part of the root canal system and they can contain inflamed pulp tissue that may become necrotic and eventually the lateral canals become pulpless and infected. In addition, pulp and root canal conditions can cause other periradicular conditions such as various forms of external root resorption. Hence, the term "periradicular" is more comprehensive, more accurate and more appropriate when discussing conditions caused by pulp and root canal conditions.

The various forms of external resorption will not be discussed in this chapter as they are discussed in detail in Chapter 15.

Clinically Normal Periapical/Periradicular Tissues

The term "clinically normal" is used when there are no signs or symptoms to suggest that any form of disease is occurring. The term "clinically" is used since the periapical/periradicular tissues may not be histologically normal and/or may have some degree of inflammation or fibrosis (scarring) as a result of previous injury or stimuli (such as some form of apical periodontitis, etc.). However, for all intents and purposes, the tooth does not have any periapical/periradicular pathosis that requires treatment.

Typical symptoms—no symptoms from the periapical or periradicular tissues; if there are any symptoms, these will be from the pulp—hence, symptoms depend on the status of the pulp.

Clinical findings—no abnormal findings; no tenderness to percussion; no tenderness to palpation; no increased mobility; various pulp and/or root canal conditions may be present—any clinical findings will depend on what condition is present; the tooth may or may not respond to pulp sensibility tests—this depends on the state of the pulp/root canal system.

Radiographic findings—no abnormal observations of the tooth; normal PDL space and lamina dura; some cases may have evidence of previous endodontic treatment.

Associated pulp/root canal conditions—may have any of the following—depends on the symptoms, clinical and radiographic findings: clinically normal pulp, reversible pulpitis—chronic or acute, irreversible pulpitis—chronic or acute, pulp necrobiosis, necrosis with no signs of infection, necrotic and infected pulp, previous endodontic treatment with no signs of infection, pulp atrophy, PCC, pulp hyperplasia, internal surface resorption, internal inflammatory resorption, or internal replacement resorption.

Key findings for diagnosis—no symptoms from the periapical/periradicular tissues and no abnormal findings.

Primary Acute Apical Periodontitis

The term *"apical periodontitis"* implies inflammation of the periapical tissues. When this first commences, there will not be a radiolucency as it takes some time for the bone (and possibly some of the tooth) to be resorbed to the extent where it is visible radiographically. The first stage of apical periodontitis is called "primary acute apical periodontitis"[2,5] and its key feature is the lack of a radiolucency (Figure 8-4), yet there is considerable pain. The pain occurs, and is usually intense, because the inflammation is confined within the hard bony tissues and the PDL where there is no space for the swelling, exudate, and so forth to expand.

- *Typical symptoms*—the pain has only been present for a short time (e.g., a few days or less); the pain is usually quite severe; pain to biting and pressure on the tooth; pain to touch the tooth—even with light pressure; may be pain due to acute irreversible pulpitis if present; occasionally may have pain due to acute reversible pulpitis.
- *Clinical findings*—may be caries, a restoration breaking down or a crack; severe pain to percussion; pain when pushing on the tooth; usually no response to pulp sensibility tests—unless acute reversible pulpitis or acute irreversible pulpitis in which case the tooth will respond to cold pulp testing which reproduces the thermal sensitivity pain that the patient will also complain of; sometimes the tooth is in traumatic occlusion due to being extruded from its normal position as a result of the periapical inflammation being constrained by the hard bone structure that has not yet resorbed to create space.
- *Radiographic findings*—caries may be seen if extensive enough; normal PDL space and lamina dura; a slightly widened PDL space may be evident if the tooth is extruded from its normal position; some cases may have evidence of previous endodontic treatment; occasionally may see a radiolucency within the tooth root indicating internal inflammatory resorption.
- *Associated pulp/root canal conditions*—usually due to an infected root canal system—may have any of the following depending on symptoms, clinical and radiographic findings: pulp necrobiosis, a necrotic and infected pulp, previous endodontic treatment with an infected root canal system, or internal inflammatory resorption; may be associated with acute irreversible pulpitis; rarely associated with acute reversible pulpitis.
- *Key findings for diagnosis*—there are no radiographic periapical changes, the pain is recent and severe; there is pain when touching the tooth, with pressure and on percussion.
- *Distinguish from* ***secondary acute apical periodontitis*** by the lack of radiographic signs of any periapical changes.
- *Distinguish from* ***chronic apical periodontitis*** by the lack of radiographic signs of any periapical changes, plus the presence of pain and the tenderness to percussion.
- *Distinguish from a* ***primary acute apical abscess*** by the lack of swelling.
- *Distinguish from a* ***secondary acute apical abscess*** by the lack of swelling.
- *Distinguish from a* ***chronic apical abscess*** by the lack of a draining sinus.

Secondary Acute Apical Periodontitis

Secondary acute apical periodontitis occurs when there has been chronic apical periodontitis or a chronic apical abscess present for some time so there is also a periapical radiolucency (Figure 8-7). Secondary acute apical periodontitis is usually a result of an imbalance occurring between the intra-canal infection and the host defence system whereby some bacteria and/or their endotoxins escape through the apical foramen and cause an acute exacerbation of the chronic inflammatory response that has been present for some time. The patient then becomes aware of symptoms. This process may occur on numerous occasions (e.g., patient reports that the pain "comes and goes") so in effect it is not necessarily just the second time that an acute inflammatory reaction occurs but every time that it changes from chronic to acute inflammation. The process is dynamic and the secondary acute apical periodontitis can return to being chronic apical periodontitis if the environmental conditions are suitable and the host defence response is adequate.

- *Typical symptoms*—the pain has only been present for a short time (e.g., a few days or less); the pain is usually quite severe; pain to biting or pressure on the tooth; no thermal sensitivity.
- *Clinical findings*—may be caries, a restoration breaking down or a crack; very tender to percussion; no response to pulp sensibility tests; may be tender to palpation.
- *Radiographic findings*—caries may be seen if extensive enough; a periapical radiolucency is present—this indicates chronic apical periodontitis has been present for some time; some cases may have evidence of previous endodontic treatment; occasionally may see a radiolucency within the tooth root indicating internal inflammatory resorption.
- *Associated pulp/root canal conditions*—will be due to an infected root canal system, hence, may have any of the following depending on the symptoms, clinical and radiographic findings: pulpless and infected root canal system, previous endodontic treatment with an infected root canal system, or internal inflammatory resorption.
- *Key findings for diagnosis*—there is a periapical radiolucency, the pain is recent and severe; pain with pressure and on percussion.
- *Distinguish from* ***primary acute apical periodontitis*** by the presence of a periapical radiolucency.

Distinguish from ***chronic apical periodontitis*** by the pain and the tenderness to percussion.
Distinguish from a ***primary acute apical abscess*** by the lack of swelling.
Distinguish from a ***secondary acute apical abscess*** by the lack of swelling.
Distinguish from a ***chronic apical abscess*** by the lack of a draining sinus.

Chronic Apical Periodontitis

After an initial period of primary acute apical periodontitis, and if the tooth has not been treated, the periapical inflammation can become chronic. After several months, this usually manifests as a periapical radiolucency (Figure 8-5). Bone is resorbed to create space for the inflammatory reaction to occur so there are usually no, or only occasional mild symptoms or an occasional "awareness" of the tooth feeling different to the other teeth.

Typical symptoms—usually no symptoms; the patient may report a history of occasional "awareness" of the tooth feeling different; the patient may report a history of previous pulp-related symptoms some time ago; the patient may report a history of previous endodontic treatment.
Clinical findings—may be caries, a restoration breaking down or a crack; the tooth is not tender to percussion but will sometimes feel "different" to percussion; no response to pulp sensibility tests.
Radiographic findings—caries may be seen if extensive enough; a periapical radiolucency will be present; some cases may have evidence of previous endodontic treatment; occasionally may see a radiolucency within the tooth root indicating internal inflammatory resorption.
Associated pulp/root canal conditions—will be due to an infected root canal system—may have any of the following depending on the symptoms, clinical and radiographic findings: a pulpless and infected root canal system, previous endodontic treatment with an infected root canal system, or internal inflammatory resorption.
Key findings for diagnosis—there is a periapical radiolucency and no pain or only occasional "awareness" of the tooth.
Distinguish from ***primary acute apical periodontitis*** by the presence of a periapical radiolucency.
Distinguish from ***secondary acute apical periodontitis*** by the lack of symptoms.
Distinguish from a ***primary acute apical abscess*** by the lack of swelling and lack of symptoms.
Distinguish from a ***secondary acute apical abscess*** by the lack of swelling and lack of symptoms.
Distinguish from a ***chronic apical abscess*** by the lack of a draining sinus.

Condensing Osteitis

Condensing osteitis is a form of chronic apical periodontitis but it manifests as a radiopacity instead of a radiolucency (Figure 8-9). It is most commonly associated with teeth that have long-standing pulpitis—usually, or at least initially, chronic reversible pulpitis.[5] Hence, it is essentially an extension of the inflammatory response that began in the pulp or it is an indication that the periapical tissues have been irritated for some time with the result that more bone is laid down rather than being resorbed. The chronic reversible pulpitis may have episodes of acute reversible pulpitis that may be the time when the patient presents for treatment. Alternatively, the chronic reversible pulpitis may develop into chronic irreversible pulpitis, acute irreversible pulpitis or pulp necrosis with infection. Also, the root canal system may become pulpless and infected. The state of the pulp/root canal will depend on when the patient presents for treatment whereas the increased bone density that is evident radiographically may take many years to resolve and may not resolve at all. When the root canal system becomes infected, a radiolucency will typically develop immediately adjacent to the root apex and this may be surrounded by the sclerotic bone of the condensing osteitis, thus appearing as both radiopacity and radiolucency.

Typical symptoms—no symptoms from the periapical or periradicular tissues; no tenderness to percussion; any symptoms will be from the pulp—hence, symptoms depend on the status of the pulp; usually associated with long-standing chronic reversible pulpitis—hence symptoms will be typical of this condition (see earlier); the chronic reversible pulpitis may progress to acute reversible pulpitis, chronic irreversible pulpitis or acute irreversible pulpitis by the time the patient presents for treatment—hence the symptoms vary according to the stage of pulp disease.
Clinical findings—may be caries, a restoration breaking down or a crack; the tooth is not tender to percussion but may occasionally feel "different" to percussion; the tooth will usually respond to pulp sensibility tests (the nature of the

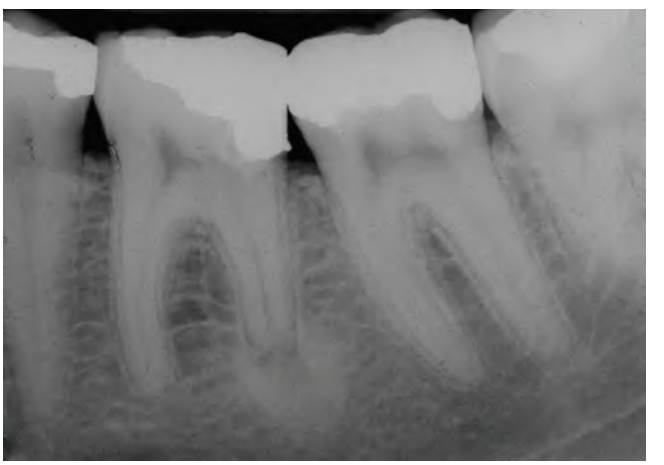

FIGURE 8-9 Condensing osteitis associated with the distal root of a mandibular first molar that had a long-standing history of chronic reversible pulpitis that subsequently became acute irreversible pulpitis two days prior to the patient presenting for treatment. The pulpitis developed as a result of breakdown of the 30-year old amalgam restoration. Note there is also evidence of slight apical inflammatory resorption of the distal root.

response depends on the state of the pulp) unless the pulp state has progressed to pulp necrosis, and so forth.

Radiographic findings—caries may be seen if extensive enough; the periapical region appears more radiopaque than the surrounding bone due to increased bone deposition (i.e., condensing osteitis); some cases may present later after the pulp has necrosed and the root canal system has become infected with various forms of apical periodontitis or apical abscesses—in these cases, some condensing osteitis may still be evident radiographically and there may be a widened PDL space or a radiolucency surrounded by the denser bone of condensing osteitis.

Associated pulp/root canal conditions—will be long-standing chronic reversible pulpitis initially; the chronic reversible pulpitis may progress to any of the following depending on the symptoms, clinical and radiographic findings: acute reversible pulpitis, chronic irreversible pulpitis or acute irreversible pulpitis; some cases may present later after the pulp has necrosed and the root canal system has become pulpless and infected with various forms of apical periodontitis or apical abscesses—in these cases, some condensing osteitis may still be evident radiographically but the process has changed to bone resorption instead of increased bone deposition.

Key findings for diagnosis—there is a radiopacity indicating denser bone (i.e., condensing osteitis) present in the periapical region.

Distinguish from ***primary acute apical periodontitis*** by the presence of the radiopacity and lack of symptoms.

Distinguish from ***secondary acute apical periodontitis*** by the presence of the radiopacity and lack of symptoms.

Distinguish from ***chronic apical periodontitis*** by the presence of the radiopacity (although it should be noted that condensing osteitis is essentially a form of chronic apical periodontitis).

Distinguish from a ***primary acute apical abscess*** by the lack of swelling and lack of symptoms, and the presence of the radiopacity.

Distinguish from a ***secondary acute apical abscess*** by the lack of swelling and lack of symptoms, and the presence of the radiopacity.

Distinguish from a ***chronic apical abscess*** by the lack of a draining sinus and the presence of the radiopacity.

Primary Acute Apical Abscess

An abscess is defined as a *"localized collection of pus."* When this occurs in the early stages of the periapical disease process, there will not be a radiolucency as it takes some time for the bone (and possibly some of the tooth) to be resorbed to the extent where it is visible radiographically (Figure 8-10). Hence, an abscess in its early stages is called "primary acute apical abscess"[2,5] and its key feature is the lack of a radiolucency, yet there is considerable pain. The pain occurs, and is usually intense, because the inflammation and the pus are confined within the hard bony tissues and the PDL where there is no space for the pus and swelling to expand.

Typical symptoms—the pain has only been present for a short time (e.g., a few days or less); the swelling has had rapid onset (e.g., hours, or less than one day); pus will be present; the pain is severe; pain to biting and pressure on the tooth; pain to touch the tooth—even with light pressure; occasionally the patient may complain of feeling unwell and having fever.

Clinical findings—may be caries, a restoration breaking down or a crack; swelling associated with the tooth;

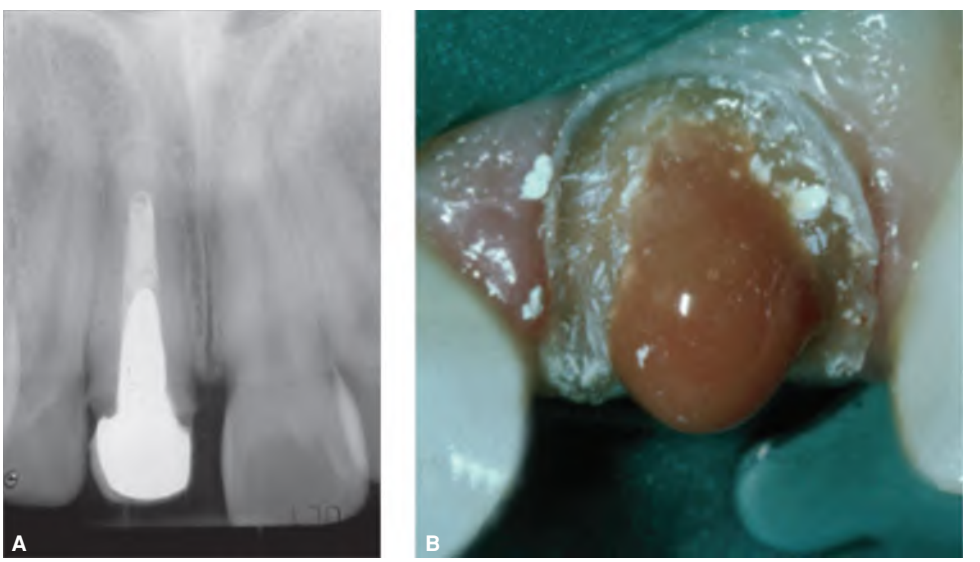

FIGURE 8-10 The maxillary right central incisor has a root-filled and infected root canal system and a primary acute apical abscess as a result of breakdown of the crown restoration. ***A.*** Periapical radiograph showing no periapical changes. ***B.*** Pus draining as soon as the existing root canal filling was removed.

severe pain to percussion; pain when pushing on the tooth; pain on palpation; no response to pulp sensibility tests; usually the tooth is in traumatic occlusion due to being extruded from its normal position; occasionally the patient may have fever, malaise and lymphadenopathy.

Radiographic findings—caries may be seen if extensive enough; normal PDL space and lamina dura; a slightly widened PDL space may be evident if the tooth is extruded from its normal position; some cases may have evidence of previous endodontic treatment; occasionally may see a radiolucency within the tooth root indicating internal inflammatory resorption.

Associated pulp/root canal conditions—will be due to an infected root canal system—hence, may have any of the following depending on the symptoms, clinical and radiographic findings: a necrotic and infected pulp, previous endodontic treatment with an infected root canal system, or internal inflammatory resorption.

Key findings for diagnosis—there are no radiographic periapical changes, there is swelling present, the pain is recent and severe, and there is pain when touching the tooth, with pressure and on percussion.

Distinguish from ***primary acute apical periodontitis*** by the presence of the swelling and pus.

Distinguish from ***secondary acute apical periodontitis*** by the lack of periapical radiolucency, and the presence of the swelling and pus.

Distinguish from ***chronic apical periodontitis*** by the lack of periapical radiolucency, and the presence of pain, swelling tenderness to percussion, and pus.

Distinguish from a ***secondary acute apical abscess*** by the lack of periapical radiolucency.

Distinguish from a ***chronic apical abscess*** by the lack of a draining sinus and no periapical radiolucency.

Secondary Acute Apical Abscess

A secondary acute apical abscess occurs when there has been chronic apical periodontitis or a chronic apical abscess present for some time so there is also a radiolucency (Figure 8-11). A secondary acute apical abscess is usually a result of an imbalance occurring between the intra-canal infection and the host defense system whereby some bacteria and/or their endotoxins (or both) escape through the apical foramen and cause an acute exacerbation of the chronic inflammatory response that has been present for some time. The acute exacerbation will be in the form of a localized collection of pus (i.e., an abscess). It may also occur when the draining sinus of a chronic apical abscess closes or becomes blocked so the pus cannot drain.

Typical symptoms—the pain has only been present for a short time (e.g., a few days or less); the swelling has had rapid onset (e.g., hours, or less than one day); pus will be present; the pain is usually quite severe; pain to biting or pressure on the tooth; no thermal sensitivity; occasionally the patient may complain of feeling unwell and having fever.

Clinical findings—may be caries, a restoration breaking down or a crack; the tooth is very tender to percussion; no response to pulp sensibility tests; tender to palpation; occasionally the patient may have fever, malaise and lymphadenopathy.

Radiographic findings—caries may be seen if extensive enough; there will be a periapical radiolucency present—indicates chronic apical periodontitis has been present for some time before the abscess developed; some cases may have evidence of previous endodontic treatment; occasionally may see a radiolucency within the tooth root indicating internal inflammatory resorption.

Associated pulp/root canal conditions—will be due to an infected root canal system—hence, may have any of

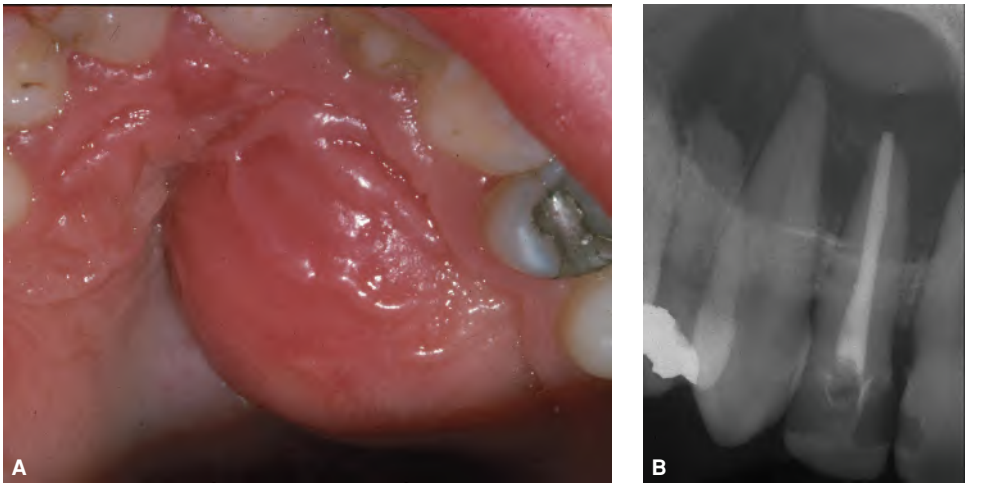

FIGURE 8-11 The maxillary right lateral incisor has a root-filled and infected root canal system with a secondary acute apical abscess. ***A.*** A large swelling on the palate that was full of pus and required drainage via an incision. ***B.*** The preoperative periapical radiograph shows a large periapical radiolucency.

the following depending on the symptoms, clinical and radiographic findings: a pulpless and infected root canal system, previous endodontic treatment with an infected root canal system, or internal inflammatory resorption.

Key findings for diagnosis—there is a periapical radiolucency, swelling is present, the pain is recent and severe, and there is pain when touching the tooth, with pressure and on percussion.

Distinguish from ***primary acute apical periodontitis*** by the presence of a periapical radiolucency and the swelling.

Distinguish from ***secondary acute apical periodontitis*** by the presence of the swelling and if the patient has fever, malaise, and so forth.

Distinguish from ***chronic apical periodontitis*** by the presence of swelling, pain, fever, and so forth.

Distinguish from a ***primary acute apical abscess*** by the presence of a periapical radiolucency.

Distinguish from a ***chronic apical abscess*** by the lack of a draining sinus and the presence of swelling and symptoms.

Chronic Apical Abscess

A chronic apical abscess is characterized by the presence of a draining sinus[5]—either intra-orally or extra-orally on the face (Figure 8-12). The draining sinus may "come and go" and this will be dependent on whether there is pus to drain.

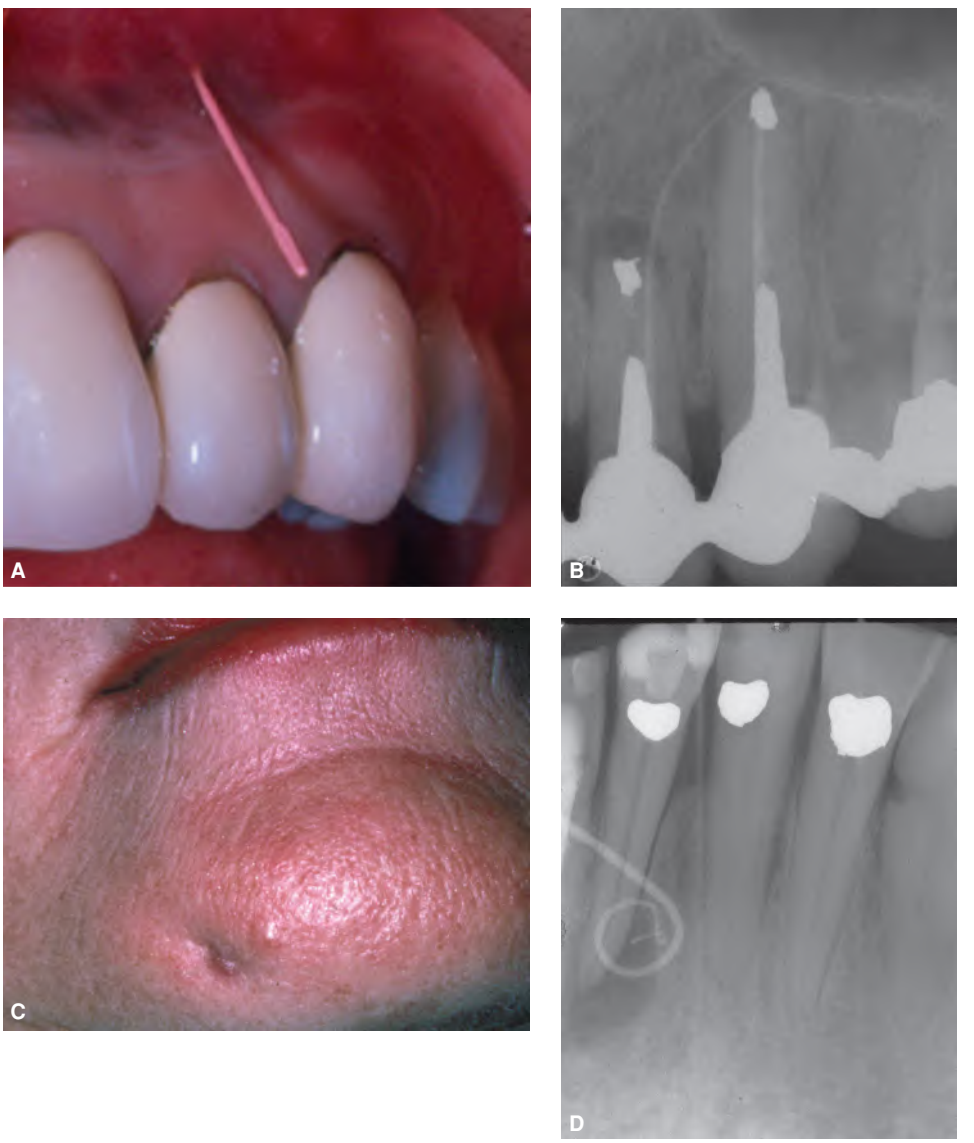

FIGURE 8-12 Examples of two chronic apical abscesses. ***A***. An intra-oral draining sinus that was tracked by inserting a gutta-percha point into the sinus and taking a radiograph. ***B.*** The periapical radiograph shows the gutta-percha point tracing the draining sinus to the periapical radiolucency associated with the maxillary left canine. The tooth has a root-filled and infected root canal system, previous periapical surgery with a root-end amalgam filling and a chronic apical abscess as a result of breakdown of the crown restoration. ***C.*** An extra-oral draining sinus (on the face) tracks to the mandibular right lateral incisor. ***D.*** The periapical radiograph shows the gutta-percha point tracing the draining sinus track. The tooth has incomplete root canal treatment, an infected root canal system and a chronic apical abscess as a result of breakdown of the composite resin restorations.

Hence, the condition may oscillate between being chronic apical periodontitis and a chronic apical abscess.

Typical symptoms—usually there are no symptoms; some patients may be aware of the presence of a draining sinus (they may refer to it as a "gum boil," "lump," "ulcer," etc.); the draining sinus is usually intra-oral (on the oral mucosa) but may be extra-oral (on the face); the patient may report a history of occasional "awareness" of the tooth feeling different; the patient may report a history of previous pulp-related symptoms some time ago; may have history of previous endodontic treatment.

Clinical findings—may be caries, a restoration breaking down or a crack; the tooth is not tender to percussion; may feel "different" to percussion; no response to pulp sensibility tests; draining sinus evident—intra-oral or extra-oral; palpation or pressure on the tissues overlying the periapical region may cause pus to exude from the draining sinus; can often insert a gutta-percha point into the draining sinus to trace it radiographically.

Radiographic findings—caries may be seen if extensive enough; there is a periapical radiolucency present; a gutta-percha point inserted into the draining sinus can trace its origin to the infected tooth; some cases may have evidence of previous endodontic treatment; occasionally may see a radiolucency within the tooth root indicating internal inflammatory resorption.

Associated pulp/root canal conditions—will be due to an infected root canal system—hence, may have any of the following depending on the symptoms, clinical and radiographic findings: a pulpless and infected root canal system, previous endodontic treatment with an infected root canal system, or internal inflammatory resorption.

Key findings for diagnosis—there is a draining sinus, a periapical radiolucency and no pain or only occasional "awareness."

Distinguish from ***secondary acute apical periodontitis*** by the presence of the draining sinus and lack of symptoms.

Distinguish from ***chronic apical periodontitis*** by the presence of the draining sinus.

Distinguish from a ***secondary acute apical abscess*** by the presence of the draining sinus, and the lack of swelling and symptoms.

Distinguish from a ***foreign body reaction*** by the presence of the draining sinus and the lack of radiographic evidence of a foreign body (but some foreign bodies may not be radiopaque and therefore may not be evident radiographically).

Facial Cellulitis

Facial cellulitis occurs when an infected root canal system develops a periapical abscess (usually a secondary acute apical abscess, although it could be a primary acute apical abscess) and the infection spreads between the fascial planes of the muscles of the face, head, and/or neck (Figure 8-13). It is a spreading infection and it can be superficial or deep. In either case, it can be life-threatening if the resultant swelling restricts the airway. Patients with facial cellulitis require immediate and aggressive treatment—that is, not just oral antibiotic therapy but active dental treatment to remove the source of the bacteria. They often require hospitalisation and even management in an intensive care unit to protect the airway and provide life support in severe cases. For more details, see Chapter 30.

Typical symptoms—facial swelling that is increasing in size—often rapidly; the swelling has had rapid onset (e.g., hours); the patient has considerable pain and discomfort; the pain has only been present for a short time (usually less than 24 hours); there is pain to biting or pressure on the tooth; no thermal sensitivity; the patient has fever and feels unwell.

Clinical findings—facial swelling is evident; may be caries, a restoration breaking down or a crack; very tender to percussion; no response to pulp sensibility tests; tender to palpation; the patient has fever, malaise and lymphadenopathy; severe cases may have airway involvement; typically also have localized signs of a secondary acute apical abscess as the abscess usually occurs before the infection spreads to become facial cellulitis.

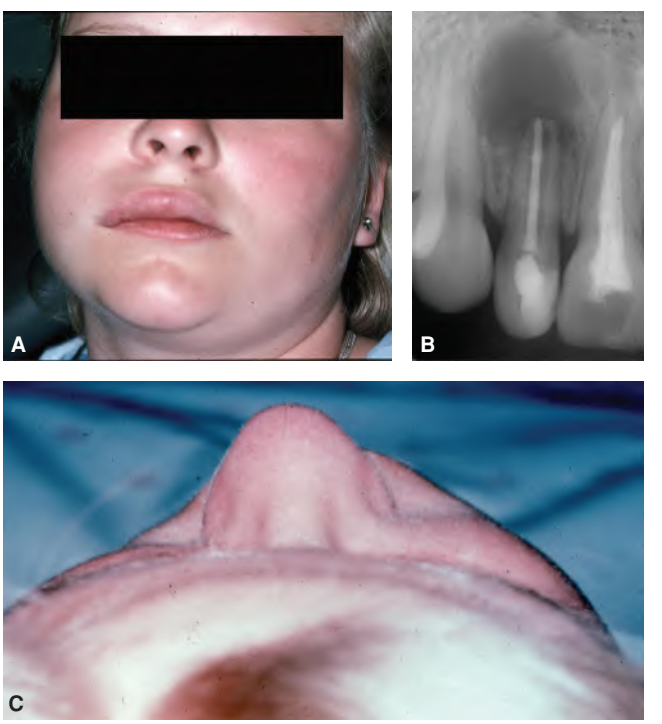

FIGURE 8-13 Facial cellulitis originating from a poorly root-filled and infected root canal system in the maxillary right lateral incisor as a result of breakdown of the composite restoration. ***A.*** Facial view showing considerable swelling and evidence of a spreading infection. ***B.*** Viewing the patient from above and behind demonstrates the extent of the swelling. ***C.*** Periapical radiograph showing a large periapical radiolucency and an inadequate root canal filling.

Radiographic findings—caries may be seen if extensive enough; there is a periapical radiolucency present—indicates chronic apical periodontitis has been present for some time before the abscess and facial cellulitis developed; some cases may have evidence of previous endodontic treatment; occasionally may see a radiolucency within the tooth root indicating internal inflammatory resorption.

Associated pulp/root canal conditions—will be due to an infected root canal system, hence, may have any of the following depending on the symptoms, clinical and radiographic findings: a pulpless and infected root canal system, previous endodontic treatment with an infected root canal system, or internal inflammatory resorption.

Key findings for diagnosis—there is facial swelling of rapid onset, the swelling is spreading, the patient has fever, feels unwell, has a periapical radiolucency, and pain with pressure and percussion.

Distinguish from ***primary acute apical periodontitis*** by the presence of a periapical radiolucency, facial swelling, fever, and malaise.

Distinguish from ***secondary acute apical periodontitis*** by the presence of facial swelling, fever, and malaise.

Distinguish from ***chronic apical periodontitis*** by the pain, facial swelling and the tenderness to percussion.

Distinguish from a ***primary acute apical abscess*** by the presence of a periapical radiolucency, facial swelling, fever, and malaise.

Distinguish from a ***secondary acute apical abscess*** by the swelling spreading throughout the face, and so forth.

Distinguish from a ***chronic apical abscess*** by the lack of a draining sinus and the presence of pain and facial swelling.

Extra-Radicular Infection

An extra-radicular infection occurs when bacteria establish colonies on the external root surface within the periapical region. It is usually a sequel to an infected root canal system and the extra-radicular bacteria are similar to those found in infected root canals.[2,23–25] Actinomyces species are often found in these cases (Figure 8-14).

Extra-radicular infections cannot be diagnosed clinically and they can only be diagnosed histologically. However, clinicians should recognize that this condition does occur and it should be part of the differential diagnosis of a persistent radiolucency following endodontic treatment.

Typical symptoms—may have no symptoms; may be associated with persistent swelling and/or draining sinus; patient may report a history of occasional "awareness" of the tooth feeling different; history of recent or current root canal treatment that is not resolving the problem.

Clinical findings—may be caries, a restoration breaking down or a crack; tooth has signs of recent root canal treatment (e.g., access cavity restoration); not tender to percussion or may feel "different" to percussion; no response to pulp sensibility tests; some cases will have a persistent draining sinus despite recent or ongoing root canal treatment—the draining sinus may be intra-oral or extra-oral; palpation or pressure on the tissues overlying the periapical region may cause pus to exude from the draining sinus, if present; can insert a gutta percha point into the draining sinus, if present, to trace it radiographically.

Radiographic findings—caries may be seen if extensive enough; a periapical radiolucency is present; the radiolucency is persisting or may be increasing in size despite recent root canal treatment; root canal filling or intracanal dressing evident; gutta percha point inserted into the draining sinus, if present, can trace its origin to the affected tooth.

Associated pulp/root canal conditions—usually considered as part of the differential diagnosis of a persistent radiolucency despite recent or ongoing endodontic

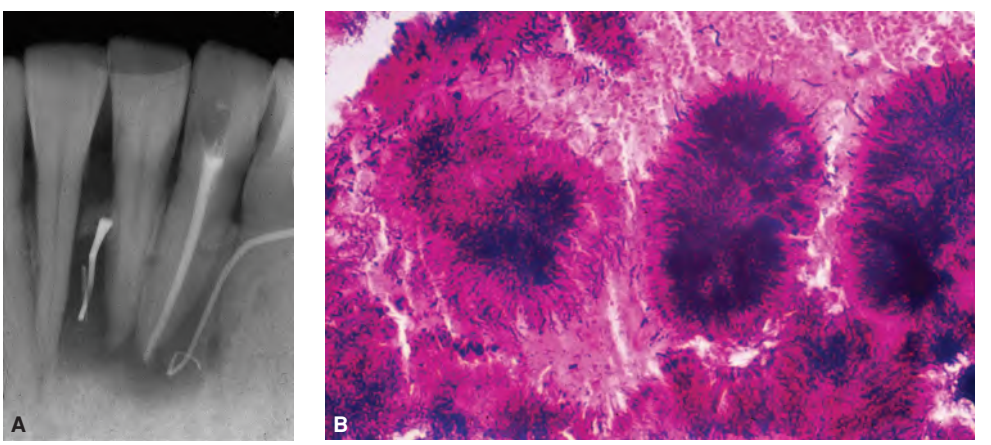

FIGURE 8-14 An extra-radicular infection associated with the mandibular left central and lateral incisors. ***A.*** The central incisor had a pulpless, infected root canal system while the lateral incisor had a root-filled and infected root canal system. Despite root canal treatment of the central incisor and root canal retreatment of the lateral incisor, the two draining sinuses persisted. Hence, a periapical curettage was done immediately after the root canals were filled. ***B.*** The histological diagnosis was "periapical actinomycosis."

treatment—hence, it will initially be associated with an infected root canal system; may be associated with previous endodontic treatment with an infected root canal system—treatment may be incomplete with an intracanal medicament in place; or treatment may be complete with a root canal filling evident.

Key findings for diagnosis—a periapical radiolucency and no pain, typically has had recent root canal treatment without resolution of the periapical radiolucency.

Distinguish from a foreign body reaction, a periapical pocket cyst, a periapical true cyst, and a periapical scar by histological examination of a biopsy specimen.

Foreign Body Reaction

A foreign body reaction is an inflammatory response to a foreign material within the periapical tissues.[2,26,27] The most common foreign material is excess root filling material, such as gutta percha or root canal cement that has been extruded through the apical foramen.[28,29] Radiographically, foreign body reactions usually appear as a periapical radiolucency surrounding some radiopaque material (Figure 8-15) but there have been reports of other materials that are not radiopaque—such as talcum powder and food.[29]

Foreign body reactions cannot be diagnosed clinically and they can only be diagnosed histologically. However, clinicians should recognize that this condition does occur and it should be part of the differential diagnosis of a persistent radiolucency following endodontic treatment.

Typical symptoms—usually no symptoms; the patient may report a history of occasional "awareness" of the tooth feeling different; history of recent or current endodontic treatment without resolution of the periapical radiolucency and/or symptoms.

Clinical findings—the tooth has signs of recent root canal treatment (e.g., access cavity restoration); the tooth is not tender to percussion or may feel "different" to percussion; no response to pulp sensibility tests.

Radiographic findings—a periapical radiolucency is present; the radiolucency is persisting or may be increasing in size despite recent root canal treatment; root canal filling or intracanal dressing evident; radiopaque material may be seen in the periapical tissues; some cases may be due to materials that are not radiopaque and therefore not evident radiographically.

Associated pulp/root canal conditions—usually considered as part of the differential diagnosis of a persistent radiolucency despite recent or ongoing endodontic treatment—hence, it will initially be associated with an infected root canal system; may be associated with previous endodontic treatment with an infected root canal system—treatment may be incomplete with an intracanal medicament in place; or the treatment may be complete with a root canal filling evident.

Key findings for diagnosis—there is a periapical radiolucency; usually no pain; typically has had root canal treatment without resolution of the periapical radiolucency; many cases have radiopaque material present within the periapical radiolucency.

Distinguish from an extra-radicular infection, a periapical pocket cyst, a periapical true cyst, and a periapical scar by histological examination of a biopsy specimen.

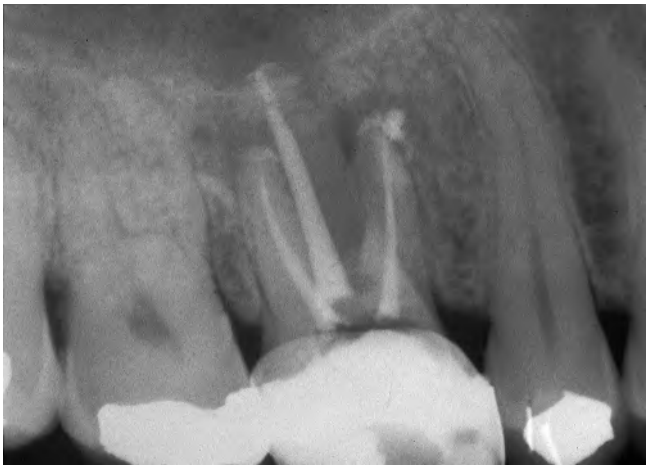

FIGURE 8-15 A foreign body reaction may occur in the periapical tissues of all three roots of this maxillary right first molar as a result of extrusion of root filling materials into the periapical tissues. However, further observation is necessary and a surgical biopsy may be required if the symptoms and signs continue.

Periapical Pocket Cyst

A cyst is a sac-like structure lined by epithelium and containing fluid or semisolid material. Nair[2,30,31] has reported and defined two types of periapical cysts—pocket cyst and true cyst. Both are essentially forms of chronic apical periodontitis that develop as a sequel to an infected root canal system but they have different histological appearances. A pocket cyst has an opening that communicates with the root canal system (Figure 8-16), hence it is not a true cyst according to the definition. It is believed that pocket cysts are likely to heal after the root canal system has been treated, but if no treatment is provided a pocket cyst may "break away" from the root canal and the tooth apex, and the opening closes to become a "true cyst."

Periapical pocket cysts and periapical true cysts cannot be differentiated clinically or radiographically from other periapical conditions that manifest as radiolucencies. In the past, many practitioners have erroneously thought that well-defined borders of the radiolucency indicated a cyst but this is only indicative of a slowly developing lesion. A diffuse border is more likely to indicate a rapidly developing lesion. Likewise, the size of the radiolucency is not indicative of a cyst if it is large—since apical periodontitis, abscesses and cysts can be small or large. A large radiolucency is more likely to indicate a long-standing problem. For more details, see Chapter 6.

Periapical pocket cysts cannot be diagnosed clinically and can only be diagnosed histologically. However, clinicians should recognize that this condition does occur and it should be part of the differential diagnosis of a persistent radiolucency following endodontic treatment.

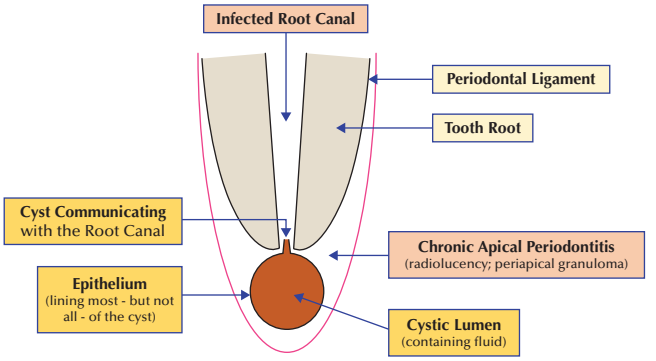

FIGURE 8-16 Schematic representation of a periapical pocket cyst showing communication of the lumen of the cyst with the root canal system (adapted from Abbott[3]).

Typical symptoms—usually no symptoms; may have symptoms if the cyst has become infected (e.g., becomes an acute apical abscess, chronic apical abscess or extra-radicular infection); the patient may report a history of occasional "awareness" of the tooth feeling different; usually a history of recent or current endodontic treatment without resolution of the periapical radiolucency and/or symptoms.

Clinical findings—may be caries, a restoration breaking down or a crack; the tooth has signs of recent root canal treatment (e.g., access cavity restoration); the tooth is not tender to percussion or palpation (unless it has become infected) or it may feel slightly "different" to percussion; no response to pulp sensibility tests.

Radiographic findings—caries may be seen if extensive enough; a periapical radiolucency is present; the radiolucency is persisting or may be increasing in size despite recent endodontic treatment; root canal filling or intracanal dressing evident.

Associated pulp/root canal conditions—usually considered as part of the differential diagnosis of a persistent radiolucency despite recent or ongoing endodontic treatment—hence, it will initially be associated with an infected root canal system; may be associated with previous endodontic treatment with an infected root canal system—treatment may be incomplete with an intracanal medicament in place; or the treatment may be complete with a root canal filling evident.

Key findings for diagnosis—no symptoms and no abnormal findings; there is a periapical radiolucency and no pain, typically has had root canal treatment without resolution of the periapical radiolucency.

Distinguish from an extra-radicular infection, a foreign body reaction, a periapical true cyst and a periapical scar by histological examination of a biopsy specimen.

Periapical True Cyst

As discussed earlier, Nair[2,30,31] has reported and defined two types of periapical cysts—pocket cyst and true cyst. A true cyst has a complete epithelial lining (Figure 8-17) and contains fluid and often cholesterol crystals. It is a self-propagating lesion that is no longer dependent on the root canal system being infected.

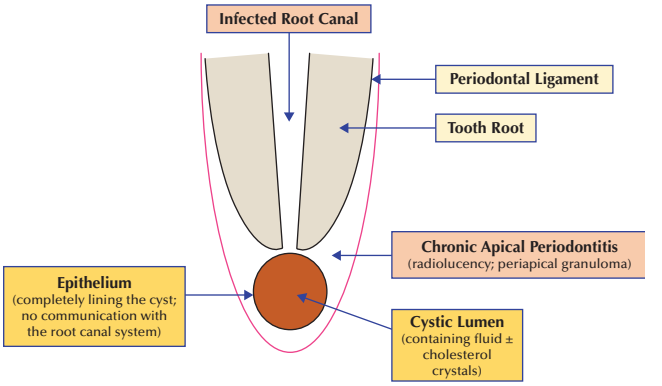

FIGURE 8-17 Schematic representation of a periapical true cyst. There is no communication between the cyst lumen and the root canal system (adapted from Abbott[3]).

Hence, root canal treatment alone will not resolve a periapical true cysts and surgical removal will be necessary.

As also discussed earlier, periapical pocket cysts and periapical true cysts cannot be differentiated clinically or radiographically from other periapical conditions that manifest as radiolucencies. The size of the radiolucency and the appearance of the borders are not specific diagnostic signs that can be used to diagnose a true cyst.

Periapical true cysts cannot be diagnosed clinically and can only be diagnosed histologically. However, clinicians should recognize that this condition does occur and it should be part of the differential diagnosis of a persistent radiolucency following endodontic treatment.

Typical symptoms—usually no symptoms; may have symptoms if the cyst has become infected (e.g., becomes an acute apical abscess, chronic apical abscess or extra-radicular infection); the patient may report a history of occasional "awareness" of the tooth feeling different; usually a history of recent or current endodontic treatment without resolution of the periapical radiolucency and/or symptoms.

Clinical findings—may be caries, a restoration breaking down or a crack; the tooth has signs of recent root canal treatment (e.g., access cavity restoration); not tender to percussion or palpation (unless it has become infected) or it may feel slightly "different" to percussion; no response to pulp sensibility tests.

Radiographic findings—caries may be seen if extensive enough; a periapical radiolucency is present; the radiolucency is persisting or may be increasing in size despite recent endodontic treatment; root canal filling or intracanal dressing evident.

Associated pulp/root canal conditions—usually considered as part of the differential diagnosis of a persistent radiolucency despite recent or ongoing endodontic treatment—hence, it will initially be associated with an infected root canal system; may be associated with previous endodontic treatment with an infected root canal system—treatment may be incomplete with an intracanal medicament in place; or the treatment may be complete with a root canal filling evident.

Key findings for diagnosis—no symptoms and no abnormal findings; there is a periapical radiolucency and no pain; typically has had root canal treatment without resolution of the periapical radiolucency.

Distinguish from an ***extra-radicular infection***, *a* ***foreign body reaction***, *a* ***periapical pocket cyst*** and a ***periapical scar*** by histological examination of a biopsy specimen.

Periapical Scar

A periapical scar is not a disease or a pathological condition. It is a healing response where fibrous, connective tissue forms instead of bone and/or PDL. Typically the radiolucency reduces in size over time but it does not completely disappear (Figure 8-18). Scar tissue may form following either treatment of an inflammatory condition with bone resorption (i.e., any form of apical periodontitis) or following surgical endodontic treatment.[31]

Periapical scars cannot be definitively diagnosed clinically; they can only be diagnosed histologically but surgery in order to biopsy a suspected periapical scar is not justified or indicated. Clinicians should recognize that this condition does occur and it should be part of the differential diagnosis of a persistent radiolucency following endodontic treatment.

Typical symptoms—no symptoms; history of root canal treatment or a surgical endodontic procedure.

Clinical findings—the tooth will have a restoration; the tooth has signs of root canal treatment (e.g., access cavity restoration); there are normal responses to percussion and palpation; no response to pulp sensibility tests.

Radiographic findings—the tooth has a restoration; the periapical radiolucency is smaller than it was preoperatively and immediately postoperatively but it is persisting following root canal treatment or endodontic surgery; a root canal filling is evident; a root-end root canal filling may be evident if there is a history of previous endodontic surgery.

Associated pulp/root canal conditions—usually considered as part of the differential diagnosis of a reduced but persistent radiolucency following root canal treatment or endodontic surgery; history of previous endodontic treatment with no signs of infection.

Key findings for diagnosis—no symptoms and no abnormal findings; there is a periapical radiolucency and no pain; typically the tooth has had root canal treatment with some, but not complete, resolution of the periapical radiolucency.

Distinguish from an ***extra-radicular infection***, *a* ***foreign body reaction***, *and a* ***periapical pocket cyst*** by histological examination of a biopsy specimen (however, a biopsy is not indicated if the clinical assessment is indicative of a periapical scar—simply observe and reassess over time).

THE DIAGNOSTIC PROCESS

Formulating a diagnosis is part of a sequence of steps or procedures which can be referred to as the "diagnostic process." The 11 stages of this process are outlined in Box 8-2. A summary of the consultation and examination procedures used in the diagnostic process is provided in Table 8-3.[11]

Each step is an essential part of the diagnostic process. If one step is omitted, or if insufficient information is obtained, then the clinician may not have the required details to make an accurate diagnosis. This process can be likened to a jigsaw puzzle where a missing piece may mean the puzzle cannot be solved although it may be possible to guess the answer—which may or may not be correct. Likewise, when diagnosing a patient's presenting condition, it may be possible in some cases to "guess" the diagnosis correctly but there is always a risk of an incorrect diagnosis that may then lead to incorrect treatment.

The diagnosis will be much stronger and more reliable if there are several conforming or corroborative findings rather than just one or two. As an example, if a patient has no response to pulp sensibility tests, a different sensation on percussion of the tooth and a periapical radiolucency, then a diagnosis of a pulpless, infected root canal system with chronic apical periodontitis is likely to be a very strong or reliable diagnosis compared to a patient who only has no response to pulp tests. The latter may be a result of PCC or a false-negative test result.

Recommended Consultation Procedure

Many patients presenting to a dentist will be apprehensive about the situation. The level of apprehension is generally

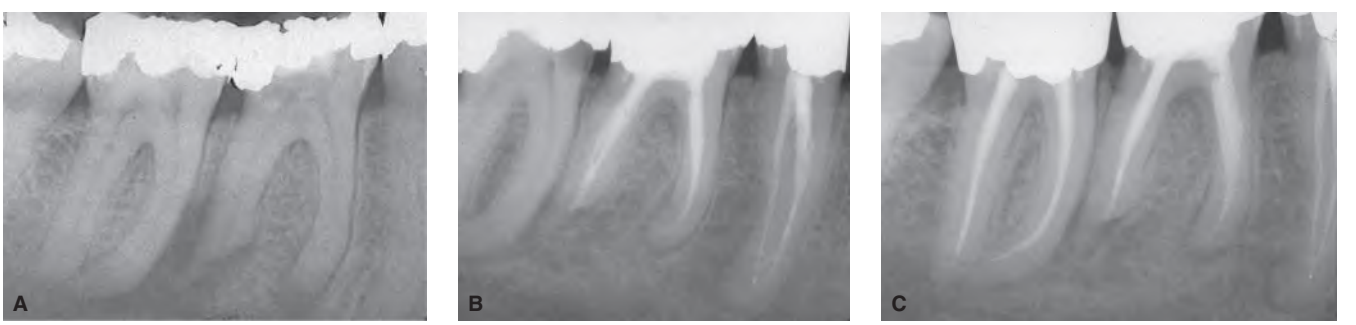

FIGURE 8-18 The mandibular right first molar is very likely to have a periapical scar, based on the radiographic follow-up. ***A.*** Preoperative radiograph shows a large radiolucency. ***B.*** One year after root canal treatment, the radiolucency is much smaller. ***C.*** The radiolucency has remained of a similar size when reviewed after three more years.

higher when the patient sees a particular dentist for the first time because the patient will not be familiar with the dentist and his/her approach, abilities, communication style, and overall office procedures. The apprehension can be even further heightened when the patient is experiencing pain, with greater levels of pain usually meaning greater apprehension. Clinicians must be sensitive to this and approach the patient with a calming and confident manner.

BOX 8-2 The 11 stages of the "diagnostic process"

1. History taking—medical, dental, and the presenting complaint
2. Clinical examination
3. Clinical tests
4. Taking and interpretation of radiographs and/or other images
5. Formulating the diagnosis/diagnoses
6. Identifying the cause(s) of the disease(s)
7. Assessing the management options
8. Discussion with the patient
9. Finalizing the management plan with the patient's consent
10. Tooth investigation—suitability for restoration/case selection
11. Records—note all findings and discussions in the patient's clinical record

Most dentists' offices consist of a dental chair and various other equipment surrounding it—such as the dental light, the dental unit with various handpieces, X-ray machines, sometimes an operating microscope, and possibly various other devices. This creates an overwhelming sight for a patient entering the room for the first time or when already apprehensive. Some patients may even feel intimidated and their level of anxiety may increase on seeing all these things surrounding them when they sit in the dental chair. Then, many dentists sit behind or to the side of the patient that does not create an ideal situation for communication with the patient to discuss their problems. This can be further exacerbated by the dentist and the assistant staff wearing face masks, safety glasses, and so forth.

Patients are usually quite familiar with consulting medical practitioners where they are more likely to sit at a desk facing the practitioner who will proceed to ask them various questions about their presenting condition before they undergo a physical examination. Dentists should ideally adopt this approach, as it is more likely that the patient will be more relaxed and will then communicate more effectively. The act of taking detailed medical and dental histories and obtaining details about the patient's presenting complaint requires effective communication. This is more readily achieved in a nonthreatening environment with the practitioner demonstrating empathy and concern for the patient.

Once sufficient information has been obtained from the patient through this communication process, the clinician

TABLE 8-3 Summary of the Examination Procedures That Should Be Performed as Part of Any Routine Dental Examination and Diagnosis but Particularly Whenever Pulp, Root Canal, and Periapical/Periradicular Pathosis Is Suspected

General medical history	As required for all dental procedures
Presenting complaint	Long- and short-term history Past and current symptoms Past and recent treatment Medications being used (prescribed, self-prescribed, alternative) Description of PAIN: location, onset, nature, duration, stimuli, relief, referred
Clinical examination	Visually examine tissues for puckering, indentation, draining sinus, facial asymmetry, swelling, etc. Probe pits, fissures, grooves and tooth surfaces for caries Assess restorations and probe all margins Transillumination of the tooth with a fibre-optic light (re cracks) Periodontal probing Mobility Biting on individual cusps (e.g., with a 'Tooth Slooth', 'FracFinder')
Clinical tests	
Pulp sensibility tests	Cold (e.g., CO_2—dry ice) Electric Heat (if required, e.g., when only complaint is sensitivity to heat)
Periradicular tests	Percussion Palpation
Radiographic examination	Periapical radiographs (all cases) Other images may be required for some cases (e.g., tube shift periapical radiographs, bitewing radiographs, occlusal radiograph, panoramic radiograph, CT scans, etc.)
Tooth investigation	Remove all restorations, caries, cracks Transillumination of the cavity, cusps, marginal ridges, etc. Assess whether, and how, the tooth can be restored again Assess the need for any other treatment (e.g., periodontal) Assess the long term prognosis of the tooth (consider endodontic, periodontal and restorative aspects)

(Adapted from Abbott[3] and Abbott and Yu.[11])

should be able to form the provisional diagnoses that can then direct the clinical examination and tests that need to be performed. The nature of the examination can be explained to the patient along with informing the patient that radiographs need to be taken. It is at this stage that the patient can be invited to move to the dental chair for the examination.

When a patient presents with pain suggesting pulp, root canal or periapical/periradicular disease, a systematic approach to diagnosis should be followed. Specific symptoms and signs will aid the diagnosis as shown in Figure 8-19. The first decision is whether the pain is a result of pulpitis or an infected root canal system (implying periapical symptoms)—this can initially be established from the history and symptoms. If the pain is originating from the pulp, then the operator must decide whether the pulpitis is reversible, irreversible and whether acute or chronic, plus whether apical periodontitis (acute or chronic) is also present. If the pain is a result of an infected root canal system, then the patient can present in various ways that can be distinguished via the history, pulp tests and radiographs. The periapical tissues must then be assessed to determine whether the problem is a result of apical periodontitis, an abscess or cellulitis, and whether the condition is acute (primary or secondary) or chronic in nature.

The clinical examination should be performed in a systematic manner as outlined subsequently. The clinical examination allows the clinician to:

- Establish the definitive diagnoses—note that when dealing with pulp, root canal, and periradicular conditions, there should always be at least two diagnoses—that is, the condition of the pulp (or the root canal system) and the condition of the periapical/periradicular tissues (as earlier),
- Identify the cause(s) of the condition(s)/disease(s),
- Assess the overall oral condition, and
- Determine the appropriate management for the presenting problem.

Once the clinical examination has been completed, the patient should be invited back to the consultation area/desk in order to discuss the findings, the diagnoses and the options for managing the problem(s). Other issues such as costs and further restoration of the tooth should also be discussed at this stage so that fully informed consent for treatment can be given by the patient.

Once the patient has consented to treatment, the patient can return to the dental chair for the commencement of the treatment, or further appointments can be arranged, as appropriate.

History

Medical History

The medical condition of the patient is an important aspect that should be assessed as part of all dental examinations. Typically, this begins with the patient filling in a written questionnaire and there are many examples of such questionnaires. Most dental institutions (e.g., schools, hospitals) will have their own versions and many professional dental organizations have developed medical history forms for use by their dentist members. The purpose of a medical history is to identify any risks that the patient may be exposed to during dental treatment and to alert the clinician to any precautions that need to be taken. The history may also alert the dentist to any risks posed to the clinician and staff. Since root canal treatment and other endodontic procedures do not carry any significantly different risks than other dental treatment, a routine medical history form can be used. The history should include questions about any medications being taken by the patient—this should include prescribed, over-the-counter, and alternate medications. The topic of medical history taking and medical conditions affecting dental treatment is well discussed in various texts and will not be elaborated here.

Particular medications of concern when managing pulp, root canal, and periradicular conditions are antibiotics, analgesics and anti-inflammatory agents. Some patients may have been prescribed, or may self-prescribe, these drugs prior to seeking dental treatment. Antibiotics are not commonly indicated and their over-use is problematic in many ways. In particular, patients may develop resistant strains of bacteria that may compromise the root canal treatment that is necessary. Inappropriate use of antibiotics is common even with conditions such as pulpitis—patients should be advised to cease taking antibiotics if they are not truly indicated. If they are required, then the clinician may need to change the antibiotic medication if the current one is not being effective.

If a patient has been using analgesics and/or anti-inflammatory agents prior to the consultation then this may provide some insight into the severity of the pain, as well as the patient's perceptions of pain and their pain-coping mechanisms. Some drugs may also affect the results of pulp testing procedures that may then affect the clinician's ability to form a diagnosis.

For more details, see Chapter 31.

Dental History

The patient should be questioned about their general dental history so the clinician can gauge the patient's general attitude to, and use of, dental services. Details such as when the patient last saw a dentist, what treatment was done, why the treatment was done, the preoperative symptoms, how often the patient seeks a dental check-up, whether they only attend when they have problems, their oral hygiene habits (regularity, bushing method, use of floss), and so forth all provide valuable information to the clinician which can be used when formulating a management plan for the presenting problem.

The Presenting Complaint

The patient should be questioned about why they have attended for dental treatment. If the patient is presenting for a general or regular check-up, then there may not be a particular problem that needs special assessment. However, if the presenting problem is pain or a "toothache," then further details are required.

General (or open-ended) questions should be used initially to encourage the patient to describe the pain or presenting problem in his/her own words. While listening to this description, the clinician should be listening for key symptoms or events that will help to diagnose the problem.

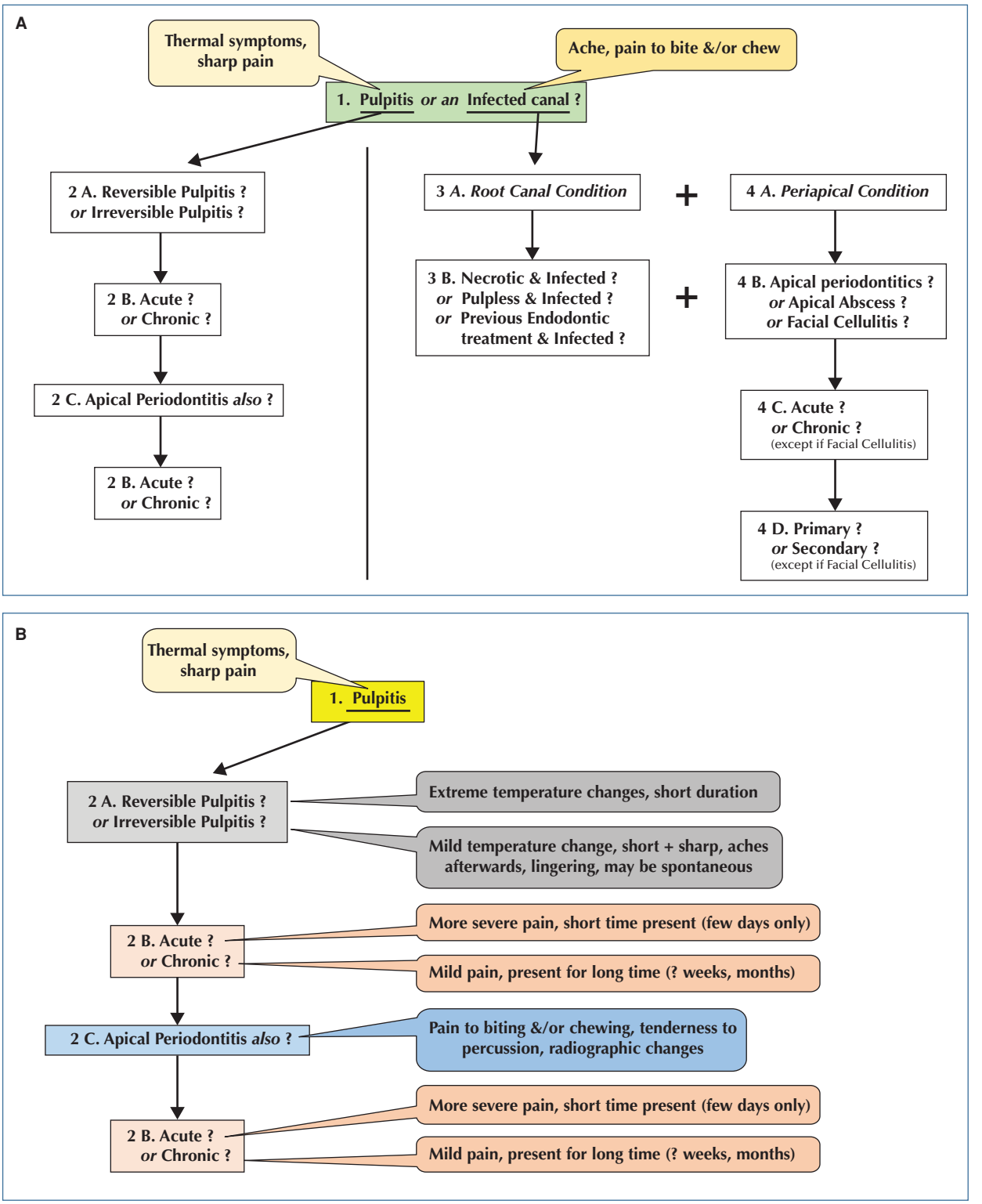

FIGURE 8-19 When a patient presents with pain suggesting pulp, root canal or periapical/periradicular disease, a systematic approach to diagnosis should be followed. Specific symptoms and signs will aid the diagnosis as shown. ***A***. The first decision is whether the pain is a result of pulpitis or an infected root canal system (that implies periapical symptoms). ***B***. If the pain is originating from the pulp, then the clinician must decide whether the pulpitis is reversible, irreversible and whether acute or chronic, plus whether apical periodontitis (acute or chronic) is also present. (*Continued on facing page*)

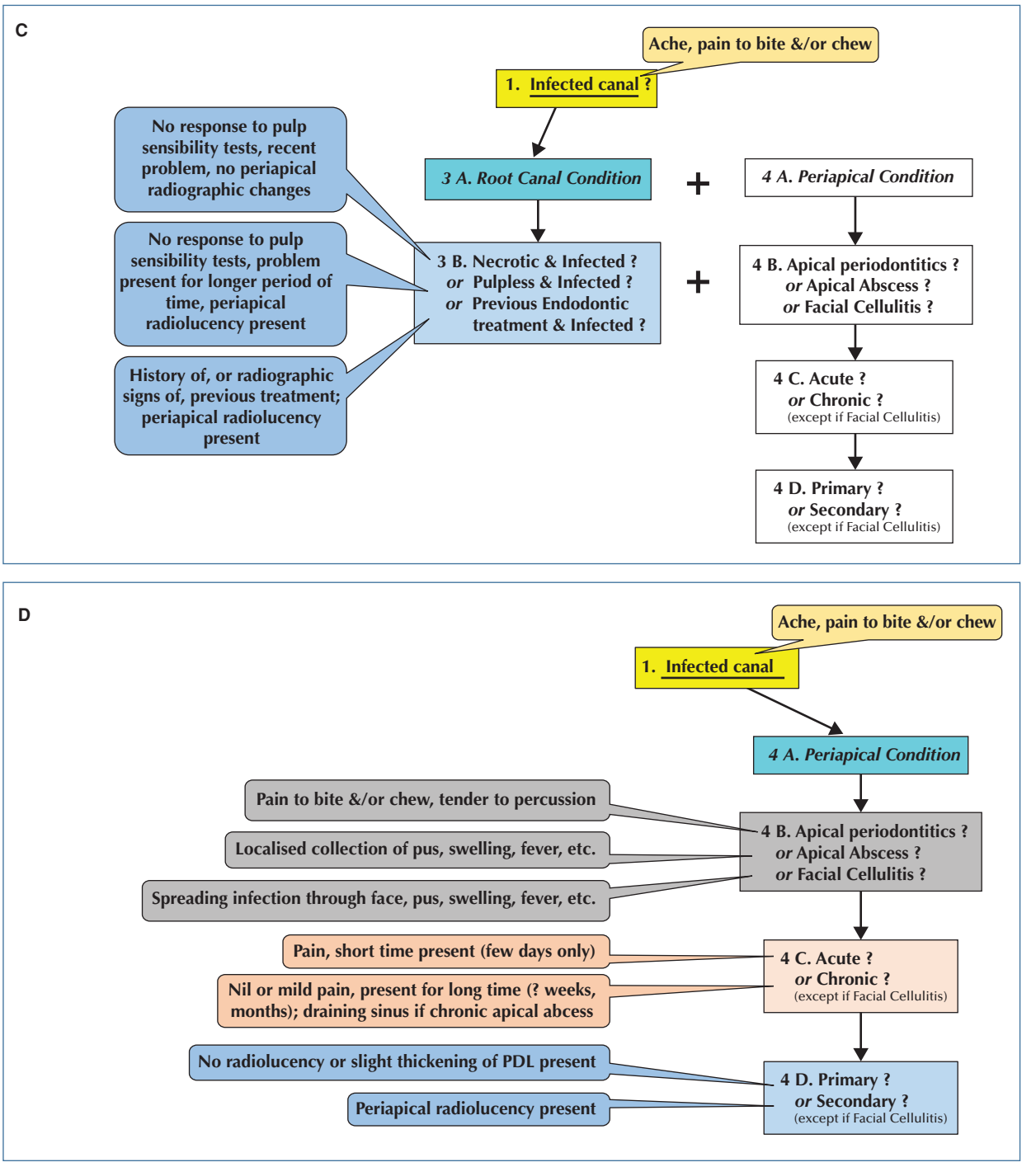

FIGURE 8-19 (*Continued*) ***C***. An infected root canal system can present in various ways that can be distinguished by the history, pulp tests and radiographs. ***D***. The periapical tissues must then be assessed to determine whether the problem is apical periodontitis or an abscess/cellulitis and whether acute (primary or secondary) or chronic.

Leading questions should be avoided wherever possible since many patients will respond in the way that they think the dentist wants them to respond, whereas allowing the patient to outline their problem without direct prompting is generally more reliable. As an example, ask the patient to describe the pain rather than asking whether it is a sharp pain. Another example is to ask the patient what stimulates the pain rather than asking if a cold drink causes the pain.

It is essential to ask the patient about the pain they are experiencing, if they present with a pain problem. This includes the stimulus required to produce the pain, the nature of the pain (sharp, dull, etc.) and how long the pain lasts. There are many dental and oral conditions that can cause pain. Each has some typical characteristics although some may be common. Hence, the more information obtained, the more reliable the diagnostic process will be.

Patients will often use the word *"sensitivity"* but this could have several meanings and could indicate different conditions. For example, sensitivity to cold or heat usually indicates pulpitis (reversible or irreversible), sweet sensitivity usually indicates a restoration is breaking down (causing reversible pulpitis), sensitivity to biting may indicate a crack undermining a cusp (causing pulpitis) or sensitivity to biting may indicate acute apical periodontitis. Hence, the patient should be asked *"what do you mean by 'sensitive'?"* in order to gather more detailed information. This also avoids the dentist being misled by what he/she perceives as *"sensitivity."*

The history should include questions about any recent dental treatment, especially in the region of the presenting problem. For example, if a restoration was done recently by another dentist, then this tooth may have pulpitis. Knowing the reasons why the restoration was done and any symptoms prior to the restoration are important when assessing what has happened since the restoration. In many cases, these patients will provide an almost textbook-like answer that describes the progression of pulp disease, thus facilitating the diagnosis.

Once the clinician has obtained a detailed history of the presenting problem, the provisional diagnoses should be made. There should be at least two provisional diagnoses at this stage—one for the pulp (if a pulp is present, otherwise for the root canal system) and the other for the periapical/periradicular tissues. The provisional diagnoses should be based on what the patient tells the clinician—hence, the value of the history should not be under-estimated. Once the clinician has the provisional diagnoses in mind, the clinical examination can be performed and the provisional diagnoses will help to target this examination with the appropriate tests and procedures.

The Clinical Examination

The clinical examination should ideally include an extra-oral examination and a detailed examination of all the soft tissues in the mouth, the lips, and the teeth. However, in an emergency or pain situation, a limited examination is acceptable in order to diagnose and manage the presenting problem. In this scenario, the patient should be advised to return for a comprehensive examination of the entire mouth at a later date.

The limited dental examination should include a screening of the soft tissues in order to eliminate any problems such as oral cancer or ulcerations that may be present and causing the pain. A screen of all teeth should also be performed in order to identify any major and/or urgent problems that require attention. This screening of the mouth and teeth also provides the clinician with a general overview of the patient's oral conditions that will help with planning how to manage the presenting problem(s).

The limited examination can then focus on the area of interest with the provisional diagnoses in mind. The provisional diagnoses will help to determine which clinical tests will be the most appropriate to lead to definitive diagnoses. For example, if the provisional diagnosis is acute irreversible pulpitis, then a cold pulp sensibility test is the most appropriate and reliable test, whereas a pulpless, infected root canal system with chronic apical periodontitis will be more reliably assessed through radiographic examination. This does not mean that all tests are not required; rather they should all be performed but the relative weight of the information obtained from each test will vary with the different conditions.

The overall aims of diagnostic testing are two-fold. The first aim is to gain objective data from the patient's symptoms and clinical signs as well as from the results of diagnostic tests. The second aim is to reproduce the patient's chief complaint if the patient has been experiencing pain. If the patient has been experiencing pain and this pain cannot be reproduced during the examination, then the practitioner should be wary of providing any treatment that may be well-intentioned but might result in a poor outcome, since that treatment may not be related to the chief complaint and may not actually have been necessary.

In order to diagnose teeth with pulp, root canal or periradicular conditions, the following procedures and tests should be carried out:

Extra-oral Examination

The face should first be observed while taking the medical and dental histories. Swellings are usually obvious but occasionally they may be only slight and not so obvious. When the patient sits in the dental chair, the clinician should observe the patient from behind and above the head to see if there is any asymmetry between the two sides of the face. Any swellings that are identified should be palpated to determine whether they are hard or soft, fluctuant, painful, hot to touch, red or have a different colour. The lymph nodes in the head and neck should also be palpated for any patient presenting with an infection or inflammatory condition. The jaw opening should also be assessed to determine if there is trismus that may impede treatment, particularly when there is swelling associated with an apical abscess or facial cellulitis.

The face should also be examined for the presence of an extra-oral draining sinus, or signs of a scar that may indicate a previous draining sinus that has temporarily healed. An extra-oral draining sinus may be indicative of a chronic apical abscess associated with an infected root canal system. The extra-oral draining sinus should be traced by placing a gutta-percha point into the draining sinus and exposing a periapical radiograph to trace its origin.

Intra-oral Examination

As mentioned earlier, the mouth should be screened for any soft tissue lesions, that is, visually inspect the gingivae, lips, tongue, cheeks and look for any abnormalities such as ulcerations, swellings or other signs. In particular, if the history suggests an infected root canal system, then check carefully for the presence of a draining sinus, or evidence of a draining sinus that may no longer be active.

The teeth should then be checked for the presence of caries, restorations, cracks, fractures, discolorations, developmental defects or any other abnormalities. The first stage of assessing teeth is to visually examine them using a good light source. The teeth should be probed to assess pits, fissures, grooves, restoration margins and any other defects. Cracks and fractures are best identified by transilluminating the tooth using an intense light source, as described subsequently.

Many text books and teachers refer to one or more of the following procedures as *"special tests."* However, except where mentioned subsequently, they should all be considered as ***routine and mandatory tests*** that are required for all cases where pulp, root canal, and periradicular diseases are suspected. As discussed earlier, as much information as possible should be obtained before the diagnoses are finalized. If these tests are only considered to be "special tests," then the tendency will be to only use them in special circumstances rather than routinely. All dentists should be encouraged to undertake these tests for every "new patient examination" and prior to doing any restorative dental treatment to a tooth. If the tests are not done, then chronic conditions without any symptoms or obvious clinical manifestations will remain undetected and will progress to more serious problems over time. As an example, not performing pulp sensibility tests prior to restoring a tooth is akin to not doing periodontal probing. It is well known that periodontal disease is often undetected because clinicians do not routinely do periodontal probing. Medical examples of undetected conditions as a result of not undertaking appropriate, regular and simple tests are hypertension (if blood pressure is not checked) and diabetes (if not blood tests). Likewise, pulp disease often goes undetected because clinicians do not do pulp tests routinely and they only use them if the patient reports a problem. Many teeth have pulps that progress through the various stages of the pulp/root canal disease process to become pulpless and infected root canal systems with chronic apical periodontitis for a long time before secondary acute apical periodontitis develops and the patient then seeks dental care. A study undertaken at the University of Michigan[32] reported that about 40% of teeth with infected root canal systems and periapical radiolucencies had not had any symptoms of pulpitis prior to their presentation to the University clinic. The presence of periapical radiolucencies associated with these teeth indicates that the disease process had been present for a considerable time.

Pulp Sensibility Tests

It is essential to understand the concepts of pulp sensibility testing. These tests are performed to help with the diagnosis of the condition of the pulp. Traditionally, these tests have been called "vitality" tests but this is an incorrect term to use. The use of this incorrect term over many years has resulted in many clinicians mis-diagnosing the pulp condition and not understanding the test process. The nature of the tests and the information they provide should be understood by clinicians so they can be applied correctly.

"Sensibility" is defined as "the ability to respond to a stimulus"[33] whereas "vitality" is defined as "the capacity to live, grow, or develop."[34] In dental settings, "vitality" is taken to imply that there is a viable blood supply to the pulp.[11] The typical tests used to assess pulps are either thermal (cold, heat) or electric pulp tests—these all test whether the pulp responds to the applied stimulus and hence they are "sensibility tests."[11] They do not assess the pulp's blood supply or whether it has the "capacity to live, grow or develop."[11] Specific tests are available for testing the pulp's blood supply (e.g., Laser Doppler flowmetry, pulse oximetry) but these tests have not been developed to the point where they are clinically useful on a day-to-day basis. They have been used for research but the costs and the time taken, plus the difficulties and limitations in using them, render them unsuitable for routine clinical use. In addition, they do not reproduce the symptoms reported by the patient for pulpitis cases so their application is limited.

If the tests are considered to be testing the "vitality" of the pulp, then the result can only be "vital" or "nonvital." However, these terms do not truly indicate the state of the pulp or the root canal system. In addition, these terms do not meet the criteria outlined earlier for a possible, useful and meaningful classification of diseases/conditions. In particular, they do not distinguish between many conditions with 12 of the above pulp/root canal conditions that could be called "vital" and 10 that could be called "nonvital"—the treatment for these conditions varies and therefore these terms do not help to decide the management or treatment required. Hence, these terms are inappropriate and should no longer be used in modern dentistry.

Some clinicians erroneously use the term "sensitivity tests" instead of sensibility tests. "Sensitivity" is the state of being "easily irritated or annoyed." A pulp that responds to a stimulus is displaying "sensibility" but the response may be exaggerated or painful, as when there is inflammation present (i.e., pulpitis), in which case it is then responding in a "sensitive" manner. It is the response that differs but the test is still the same (i.e., a pulp sensibility test).

Pulp sensibility tests can be used in different ways and for different reasons—their use and interpretation will be determined by the history provided by the patient and the situation being assessed. In general, pulp sensibility tests are used to:

- Assess the health status of the pulp:
  - ◊ Prior to restorative, endodontic, orthodontic, periodontal or surgical procedures,
- Locate and diagnose a tooth with pulpitis (reversible or irreversible) when the history suggests such a condition exists:
  - ◊ In such a situation, the pulp sensibility tests can reproduce the pulpitis pain (especially when associated with cold stimuli) and

◊ These tests can also be used to differentiate between reversible and irreversible pulpitis by observing the patient's response to the test stimulus,

- Locate and diagnose a tooth that has a necrotic pulp or one that has become pulpless and infected where the history suggests such a condition exists,
- As a follow-up, and to monitor, the pulp after trauma to the teeth, or
- As part of a differential diagnostic process (such as to exclude periapical pathosis of pulp origin).

There are two aspects to consider—the first is whether the pulp responds to the stimulus, while the second is the nature of the response. If a tooth does not respond to the stimulus, then this suggests that the pulp is necrotic or the root canal system has become pulpless and infected. In contrast, if a tooth does respond to the stimulus, then the pulp is either "clinically normal" or it has some form of pulpitis. The nature of the response will then determine the diagnosis—if the response is "normal" (compared to other teeth in that same patient's mouth), then the pulp is likely to be "clinically normal." However, if the response is exaggerated and the test reproduces the pain that the patient has been experiencing, then the general diagnosis will be pulpitis. The clinician must then determine what type of pulpitis—this is based on the history, the duration and the nature of the pain. The latter two aspects can be reproduced by the pulp sensibility test (especially a cold test) that then leads to the diagnosis.

There are three types of pulp sensibility tests commonly used in dental practice. They are cold, heat and electric tests. It is important to note that all pulp tests should be performed and interpreted in conjunction with all other clinical findings and periapical radiographs. If other information and radiographs are not available, then the pulp tests may not be meaningful—for example, a tooth may not respond to a cold pulp test because it has PCC that can only be determined radiographically. Another example is a tooth that does not respond to both cold and electric pulp tests because it has had previous root canal treatment—the presence of the root canal filling can only be noted radiographically or sometimes by clinically observing a typical endodontic access cavity restoration in the tooth.

Cold Pulp Sensibility Tests

There are several methods for performing cold pulp sensibility tests. These include the use of dry ice sticks (carbon dioxide, CO_2) (Figure 8-20), refrigerant sprays (Figure 8-21) and ice. The principle of using these tests is to apply the cold stimulus to the enamel of the tooth or on the restoration so the temperature of the tooth is reduced. The nerve fibres within the pulp will feel this change in temperature if they are functioning normally or if they are inflamed. In the inflamed situation

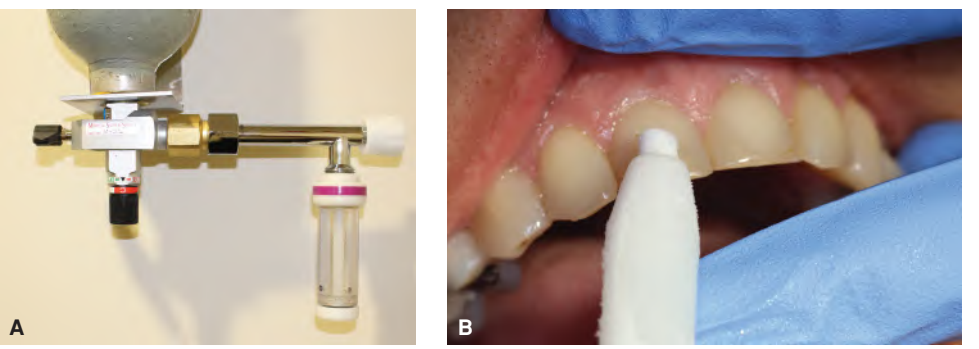

FIGURE 8-20 ***A.*** The Odontotest carbon dioxide pulp-testing device. ***B.*** The Odontotest being used to test the anterior teeth of a patient.

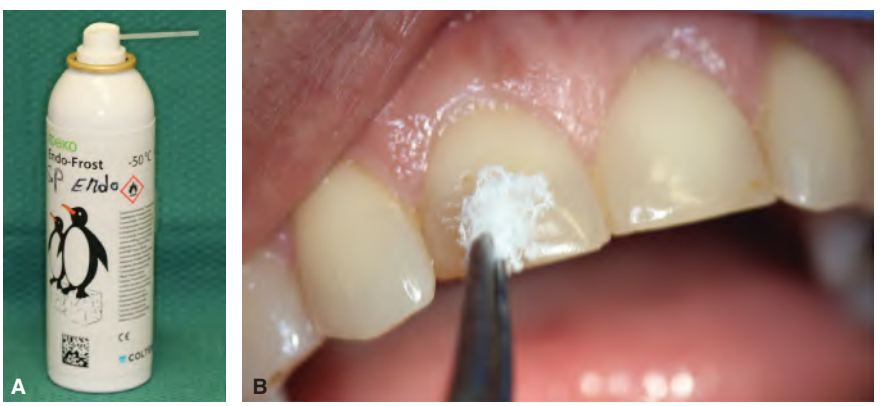

FIGURE 8-21 ***A.*** An example of a cold spray used for pulp testing. ***B.*** A cotton pellet held by tweezers and sprayed with the cold spray gas being used to pulp test a tooth.

(i.e., pulpitis), the sensation will be greater and typically reproduces the pain that the patient has been experiencing.

Chen and Abbott[35] have reported that dry ice sticks were the most reliable cold test to use in most diagnostic situations. It is also the cheapest and quickest method of testing the pulp's reaction to a cold stimulus. The dry ice is formed in a device known as the Odontotest (Fricar A.G., Zurich, Switzerland). This device uses pressurized liquid carbon dioxide that is forced through a small orifice. It becomes dry ice when it comes under atmospheric pressure. The device produces a "stick" of dry ice inside a thin plexiglass tube. A plunger is used to compress the dry ice and to extrude a small amount from the tube so it can be applied to the tooth surface. The temperature of the dry ice is approximately −78°C when it exits the cylinder with an effective temperature of about −56°C when applied to the teeth.[36] The dry ice stick is applied directly to the tooth surface or to a restoration if present. It will not stick to the tooth or restoration and will not damage them.

There are several manufacturers who produce refrigerant (cold) sprays. The original gas used was ethyl chloride which was largely replaced by dichlorodifluoromethane until environmental concerns arose. Now typically tetrafluoroethane or a mixture of gases (e.g., propane/butane/isobutane) is used. Tetrafluoroethane has a temperature of about −26°C when first sprayed from the pressurized can but this reduces to about −18.5°C in the mouth while the propane/butane/isobutane mixture temperature is approximately −50°C and reduces to about −28°C in the mouth.[36] These sprays are somewhat effective for pulp sensibility testing but not ideal or reliable since they also have the disadvantage of becoming liquid quite rapidly in the warm environment of the mouth. The liquid can then flow on to the gingivae or adjacent tooth where it may be felt and misinterpreted as being from the pulp of the tooth being tested. They are generally less effective than dry ice, especially when testing teeth with porcelain crowns. These cold gases must be sprayed on to a cotton pellet that is then applied to the tooth or restoration surface. As they are gases, they will evaporate as their temperature increases, especially in the mouth. Hence, the cotton pellet must be resprayed with the gas for every tooth, or at least every second tooth, in order to maintain a reasonably constant temperature. If the pellet is not cold enough, then false responses will be obtained and this will complicate the diagnosis.

Ice has been used by freezing water in local anaesthetic cartridges and then applying the extruded portion to the tooth. This is considered to be an ineffective and unreliable test, as well as being largely impractical to use in the mouth since the ice melts very quickly and the cold water may be felt by the gingivae or an adjacent tooth, and thus misinterpreted as being from the pulp of the tooth being tested.

Heat Pulp Sensibility Tests

Pulp sensibility testing with heat is not a particularly useful or common test.[37] This is largely because it is difficult to test teeth with hot devices and there is a high risk of burning the tooth surface or the patient's lip, cheek, or tongue. Hence, there is a degree of danger associated with them. Fortunately, most patients that have sensitivity associated with hot stimuli also have pain associated with cold stimuli and therefore a cold test can be more easily performed. Heat tests are only useful if sensitivity to hot stimuli is the only symptom reported by the patient. In such a case, a cold test is usually still useful and may provide the required information anyway. In addition, teeth with clinically normal pulps do not respond to heat—they only respond to heat when they are inflamed. Hence, the heat test cannot be used to differentiate a normal pulp from a necrotic pulp or a tooth with a pulpless and infected root canal system.

Various methods have been proposed for heat testing—these include the use of a heated ball burnisher, a rotating rubber polishing cup, a heated gutta-percha stick, an electronic device (e.g., System B; Sybron Endo, Orange, CA, USA), or the application of hot water.[37] However, none of these methods are ideal and some can be dangerous.

If a heated ball burnisher is used, it needs to be heated over a Bunsen burner flame until the metal is glowing "red hot." The instrument is then held about 1–2 mm away from the labial or buccal surface of the tooth so the heat can radiate from the instrument to the tooth (Figure 8-22). It is important to avoid touching the tooth surface as the hot instrument will burn the enamel and leave a black spot. The amount of heat that effectively reaches the pulp is unpredictable and therefore this test must be used and interpreted with caution. The lips and cheeks must be carefully protected to avoid burning them.

If a rubber polishing cup is rotated as fast as possible in a low speed handpiece while in contact with the tooth surface, heat will be generated by friction. However, the amount of heat is unpredictable and may vary from one tooth to another. The vibration produced by this method may be misinterpreted by the patient as pain, especially if there is any apical periodontitis present. Hence, this method is not recommended.

Gutta-percha sticks can be heated over a Bunsen burner flame until they are almost molten at which stage they can

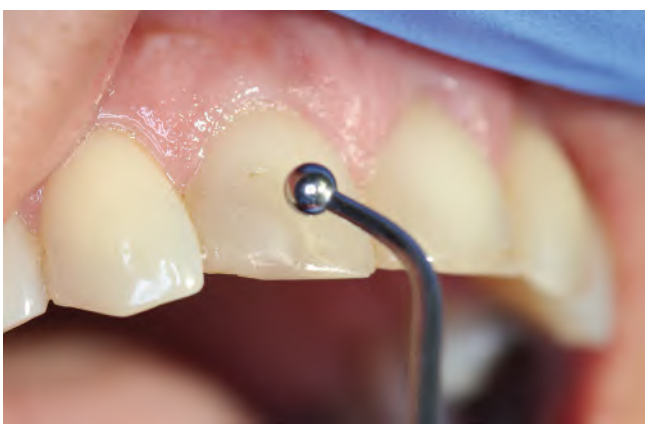

FIGURE 8-22 A ball heated burnisher being used to test a tooth with heat. Note that the instrument must not touch the tooth. It should be held 1–2 mm away from the tooth so the heat can radiate onto the tooth.

be applied to the tooth surface. However, this method is also unreliable as the temperature varies and the gutta percha cools rapidly. It is a dangerous method as the gutta-percha may drip on to the patient's lip and cause a burn. The tooth surface should be coated with a lubricant (e.g., Vaseline) to prevent the gutta-percha from sticking to it.

Electronic devices such as the System B (Sybron Endo, Orange, CA, USA) have been advocated by some clinicians. Special tips are required and the temperature can be set so it is consistent. A temperature of 65°C is recommended as this is similar to warm water that is tolerable in the mouth. This type of device is relatively safe as the surrounding tissues are unlikely to be affected.

Bathing the tooth with hot water can be a useful test at times, particularly when all other tests have not led to a conclusive diagnosis and the patient has been experiencing pain with hot stimuli. In order to do this test, the clinician needs to isolate one tooth at a time with rubber dam. Heavy body rubber dam is recommended as the thicker sheet of rubber insulates the adjacent teeth and soft tissues more effectively. The rubber dam needs to be applied carefully so the tooth is completely sealed from the mouth by the rubber dam. Water should be heated in a kettle so it is quite hot—but not boiling. It is then loaded into a syringe so it can be syringed onto the tooth to bathe it. If the tooth has pulpitis, it will usually react with pain quite quickly so the dental assistant should be ready to evacuate the hot water as soon as the patient feels the pain. Ideally one or two teeth that are expected to have normal pulps should be tested first with the suspect tooth being tested last so the patient does not become overly apprehensive. The first tooth to be tested should be the most distal tooth in the region. This procedure is a slow process since the rubber dam must be applied to one tooth at a time and then removed before being applied to the next tooth. Hence, this test is not often perfumed but it can be useful in some cases.

Electric Pulp Sensibility Tests

The first reported use of an electric pulp test dates back to 1921 when Raper[38] outlined 22 circumstances for using an electric pulp test. The majority of these reasons are still valid today. The concept of an electric pulp test is to apply a low level electric current to the tooth to assess whether the patient can feel a "tingling sensation" in the tooth. The assumption is that if the patient feels this sensation, then the pulp has viable nerve fibres although many dentists have erroneously interpreted this as meaning the pulp is "vital" (see earlier). Electric pulp tests are sensibility tests that demonstrate the pulp's ability to respond to an electric stimulus.

There are various electric pulp testers (EPTs) available. Some of these are "benchtop" devices (Figure 8-23) while some are hand held devices. They are usually battery-powered and require an electric circuit to be completed. The circuit usually includes the pulp testing device itself, the tooth, the patient and sometimes the operator all making contact to provide a complete circuit for current flow. Several methods have been proposed, depending on the device being used (Figure 8-24). Most devices have a metal

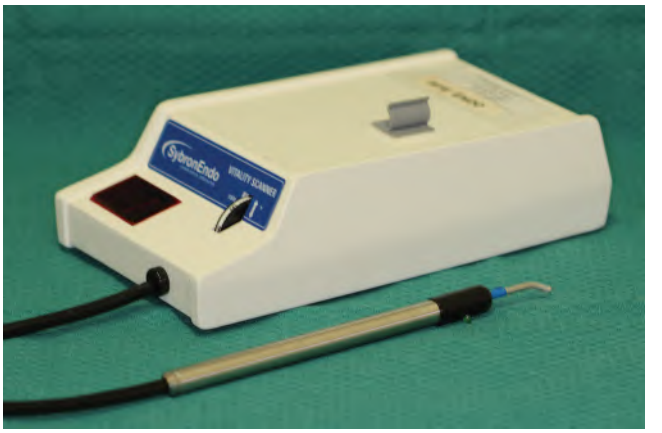

FIGURE 8-23 A "benchtop" style of electric pulp tester.

component to the probe which can be held or touched by the patient or the operator. If the patient touches the metal component, then this provides the electric circuit. If the operator holds the device, then the operator needs to remove the glove from one hand and touch the patient's skin, such as on the face, to complete the circuit. Alternatively, some devices have a metal "lip clip" which can be placed over the patient's lip and is attached to the probe. The method chosen by the operator depends on their personal preference but the operator using an un-gloved hand to touch the patient's face and hold the device is the simplest as it does not require the patient's involvement that can be cumbersome.

Some EPTs automatically increase the amount of electric current being applied to the tooth once the circuit is complete while other devices require the operator to manually increase the current—typically by turning a dial on the device. The latter is more difficult and somewhat cumbersome to use compared to the automatic devices. The automatic devices usually have a digital display to indicate the level of current being applied whereas the manual devices may have a numbers on the dial or there may be a digital display.

Most EPTs provide a scale of responses. The scale varies between the various devices that are commercially available. Some have a scale ranging from 0 to 80, while others are from 0 to 4 or 0 to 5. The scales used are not indicative of any particular amount of electric current being applied apart from indicating an increase from the lowest to the highest level.

The level at which a response is felt by the patient (or in other words, the amount of current applied to the tooth) is not critical and cannot be used to assess the health or state of the pulp tissue. That is, a tooth with pulpitis does not necessarily respond at a lower level of current than a tooth with a clinically normal pulp. Electric pulp tests do not induce pain and therefore they cannot be used to reproduce the symptoms reported by a patient. Hence, the results of electric pulp tests should only be considered as either "a response" or "no response"—in the same manner as thermal pulp tests.

Other Pulp Tests

Other pulp tests are available—such as the laser Doppler flowmeter[35] or pulse oximetry.[39,40] Both of these are truly "vitality" tests as they do test for the presence of blood within

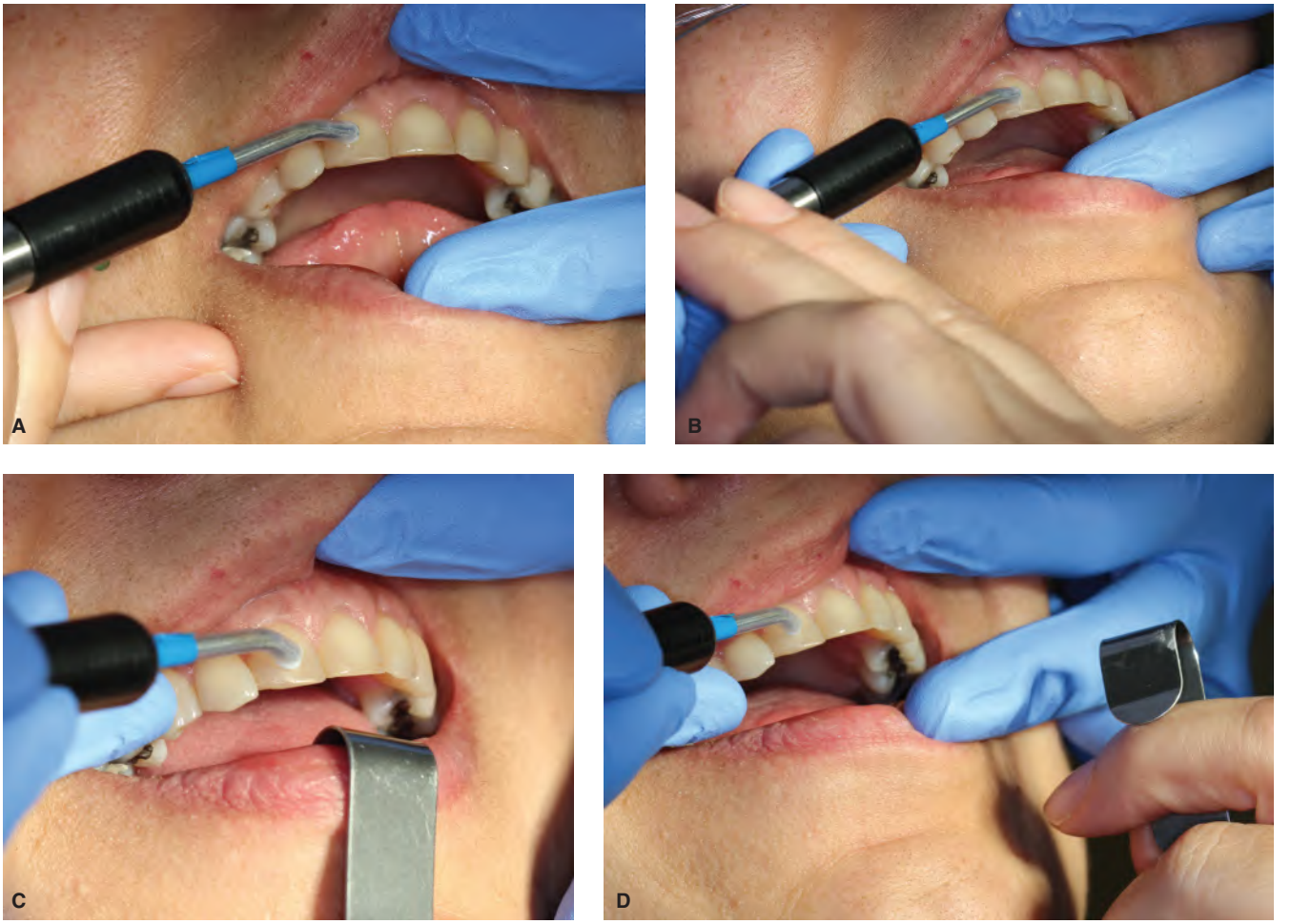

FIGURE 8-24 The electric pulp tester being used on a patient; four different methods for completing the circuit. ***A.*** The operator using one finger to touch the patient's face. ***B.*** The patient touching the metal handle of the pulp tester probe. ***C.*** The lip clip placed over the patient's lip. ***D.*** The patient holding the lip clip in her hand.

the pulp. Specifically, they test blood flow. However, studies have not been conclusive with respect to their usefulness in day-to-day clinical practice although they are useful tools for research purposes.

Laser Doppler flowmeters are expensive to purchase and very time consuming to use. They require the construction of a stent using a rubber-based impression material or acrylic in order to block out the effects of blood flow in the surrounding tissues (e.g., gingiva, adjacent tooth) as well as to provide a stable method for applying the probe to the tooth. Pulse oximetry tests the tissues for oxygen saturation.[39,40] When testing pulps with this method, the assumption is that higher oxygen saturation implies more blood flow. However, this method still has some clinical limitations such as lack of specifically designed proves and inaccurate reading in the presence of restorations.

Which Pulp Sensibility Test Is Best?

Many studies have been conducted to compare the various pulp sensibility and vitality tests under different circumstances. However, it is difficult to compare the results as different methods and statistical analyses have been used. The following is a brief summary of the most relevant and useful findings. They mainly concern pulp sensibility tests, as vitality tests are not clinically useful at present (as discussed earlier).

Chen and Abbott[35] compared CO_2, electric, cold spray (EndoFrost), ice tests and laser Doppler flowmetry. They reported that pulp sensibility testing of teeth with clinically normal pulps can be reliably performed with CO_2, electric and the cold spray tests. However, when testing teeth with diseased pulps, the CO_2 test was the most likely test to identify pulp disease and this was particularly the case when the teeth had restorations. Teeth with nonmetallic restorations were more difficult to pulp test accurately, especially when the pulp was diseased. Although the electric pulp test was accurate when there was no disease, it was not reliable when pulp disease was present. The cold spray was less reliable and less accurate than CO_2 and electric pulp tests. Ice was of little value and unreliable, as well as difficult to use. Laser Doppler flowmetry was suitable for unrestored teeth but less reliable than CO_2 and electric tests, as well as being difficult to use in restored teeth, expensive, and time consuming.

Clinically normal teeth were tested in a study by Fuss et al.[42] They reported that CO_2 and electric tests were the most reliable. Ice and ethyl chloride spray were unreliable.

When they tested immature teeth, electric pulp tests were less reliable than in fully developed teeth while the reliability of CO_2 was similar in both groups. The CO_2 test also had a greater rate of temperature decrease than ice and ethyl chloride that probably contributed to its reliability.

Peters et al.[43] used CO_2 and electric pulp tests on 1488 diseased and clinically normal pulps in 60 adults. They reported that no response to both tests (and with no history of trauma to the tooth) almost certainly indicated a diseased pulp. When there was no response to CO_2 but there was a response to the electric test, then pulp disease was possible but further clinical and radiographic investigations were required in order to make a diagnosis. If the tooth responded to CO_2 but not the electric test, then the electric test was more likely to be a false response.

Ethyl chloride, hot gutta-percha and an electric pulp test were compared in a study of 75 teeth with either clinically normal or diseased pulps.[44] Ethyl chloride was more sensitive and slightly more accurate than the electric test but their specificity and positive predictive value were the same. The heat test was the least accurate and the least specific plus it had the lowest positive and negative predictive values. Hence, heat is not a very useful test in most cases.

Jones et al.[45] compared CO_2 and a cold spray (Endo-Ice). There was no difference in producing a pulp response with these two tests but two teeth had a false-positive response. These two teeth had metal crowns and the false responses were with the cold spray—this may have been a result of gingival stimulation as the cold spray became liquid and flowed over the surface of the crown. They also reported that CO_2 was cheaper in the long term and this is consistent with the findings of Chen and Abbott.[35]

Many dentists have the perception that teeth with crowns cannot be pulp tested. However, this is not entirely true. Electric pulp tests cannot be performed when crowns are present unless there is some exposed tooth structure on which the probe can be placed. However, cold pulp tests can be used on all types of crowns, as shown by Miller et al.[46] They tested the conductivity of CO_2, a cold spray (Endo-Ice) and ice through extracted teeth with full gold crowns, porcelain-fused-to-metal (PFM) crowns, all-ceramic crowns and controls with no restorations by measuring temperature change at the pulp-dentin junction. All crown types had similar temperature reduction compared to that of intact teeth without crowns. Ice was the least effective and the temperature change associated with CO_2 lasted the longest. This study showed that PFM and ceramic crowns do not "insulate" the pulps. Hence, teeth with porcelain restorations can be effectively pulp tested with cold tests.

Weislader et al.[47] used CO_2, a cold spray (Endo-Ice) and an electric test on 150 teeth and compared their findings to the status of the pulp (bleeding or no bleeding) on gaining access to the pulp chamber. Of the teeth that responded to all three tests, 97% had bleeding on access. Of the teeth that did not respond to all three tests, 90% had no bleeding. Varying responses (i.e., responded to one or two but not all three tests) occurred in 25% of the teeth; of these, 54% had bleeding and 46 % had no bleeding. Overall, there was bleeding in 83% of the teeth that responded only to the electric pulp test, compared to 97% of teeth that responded to both a cold and electric test. These authors concluded that combinations of cold and electric pulp tests provided a more accurate assessment of the pulp condition.

Pulp testing in children is often not performed, as many practitioners believe the children cannot be relied upon to provide reliable responses, or the tests may affect their behaviour in the dental surgery. However, there is no conclusive evidence to support this belief and studies such as the one by Hori et al.[48] have demonstrated that electric, cold and heat tests can be reliably used in primary teeth. They tested teeth in children aged 6–8 years old and compared their findings to the state of the pulp on gaining access to the pulp chamber. They did not report any difficulties or behavioural issues with the children. Many practitioners regularly use pulp sensibility tests in children following trauma and there are no reports of problems with performing these tests. Hence, the age of the patient may not a barrier to the use of pulp sensibility tests.

Which Pulp Sensibility Test Should be Used and When?

There is not one single pulp test that can be recommended as a universal test. Different tests need to be applied to different clinical scenarios, based on the reason why the test is required (i.e., the provisional diagnosis obtained from the history) and the state of the tooth.

Pulp testing is performed for two main reasons—first to identify diseased pulps when a patient presents with pain or a potential pulp problem, and second, to assess the pulp status when performing a general dental examination which may be prior to restoration of the tooth, following trauma to the teeth, prior to orthodontic treatment, etc. The results of the studies discussed earlier have been combined to form recommendations (Table 8-4) for the typical clinical situations where pulp testing is required.

Practitioners should have BOTH a cold pulp test system and an EPT as the abovementioned studies have shown the value of having more than one test system. Of the various cold tests available, the CO_2 pulp tester has been shown to be more reliable, easier to use, faster, and cheaper than the cold sprays and ice.

Percussion

The percussion test is a simple means of assessing whether there is inflammation or other changes occurring in the periradicular tissues. It is an indirect test in that the periradicular tissues themselves are not directly contacted by the test. Instead, the tooth has a force applied to it via percussion and this force is transmitted through the tooth to the surrounding tissues. Percussion testing does not specifically indicate periapical changes in all cases as inflammation or changes anywhere along the entire length of the tooth root can produce different responses to the percussion test.

The simplest instrument to use for the percussion test is the handle of the dental mirror (Figure 8-25). The tooth does not need to be struck with great force—just a light tapping on the tooth is sufficient to determine whether there are

TABLE 8-4 Recommendations for the Use of Pulp Sensibility Tests in Different Clinical Situations. These Pulp Tests Should Be Performed and Interpreted in Conjunction with all Other Clinical Findings and Periapical Radiographs

Clinical Scenario	Recommended Pulp Test(s)	Reason(s) for Recommendation(s)	Comments
Routine clinical examination	Use both **CO_2** and **EPT**	• Two tests more reliable than a single test. • CO_2 and EPT are reliable for assessing clinically normal pulps.	• All teeth should be pulp tested at initial examination and regularly thereafter. • If no reason to suspect pulp disease (e.g., unrestored tooth with no caries and no history of trauma), then can use one test only—CO_2 is the easiest to perform.
Prerestoration assessment	Use both **CO_2** and **EPT**	• Two tests more reliable than a single test. • CO_2 and EPT are reliable for assessing clinically normal pulps but CO_2 is the best for diseased pulps.	• All teeth should be pulp tested prior to a new restoration being done in order to identify any pulp disease if present. • If only one test to be used—then use CO_2 as it is the most reliable (i.e., in case pulp disease is present).
Trauma follow-up	Use both **CO_2** and **EPT**	• Two tests more reliable than a single test	• Traumatized teeth will require regular follow-up. Some teeth may respond to one test but not the other.
Pulpitis (all forms of pulpitis—i.e., acute and chronic reversible pulpitis; acute and chronic irreversible pulpitis)	**CO_2**	• The most reliable test, plus • It reproduces the presenting symptom • Response can be assessed to confirm the diagnosis (e.g., severity, lingering, etc.) and to distinguish between reversible and irreversible pulpitis	• EPT has little value when testing for pulpitis as they do not differentiate between an inflamed pulp or a clinically normal pulp. • Heat tests are not reliable but can be used if the patient complains of heat sensitivity—especially if that is the only symptom.
Necrotic Pulp (with or without signs of infection)	Use both **CO_2** and **EPT**	• CO_2 is the most reliable test	• Use EPT also to confirm the CO_2 findings.
Pulpless, infected root canal system	Use both **CO_2** and **EPT**	• CO_2 is the most reliable test	• Use EPT also to confirm the CO_2 findings.
Pulp canal Calcification (PCC)	**EPT**	• EPT is the most reliable for PCC. • CO_2 and other cold tests are not reliable with PCC	• Calcification reduces conductivity of temperature changes throughout the tooth. • EPT results must be interpreted carefully as some teeth with PCC will not respond even though there is no pulp disease present. • Periapical radiographs are essential.
Crowns/Bridges	**CO_2**	• CO_2 is the most reliable test. • Temperature change lasts longer with CO_2 than cold sprays used on crowns	• Coldness is readily conducted through crowns. • Interpret carefully with bridges as difficult to know which tooth is responding (due to conduction of temperature through the bridge).
Concurrent endodontic and periodontal diseases	Use both **CO_2** and **EPT**	• Two tests more reliable than a single test.	
During orthodontic treatment	**CO_2**		• The arch wires will conduct electric current to other teeth—hence, EPT unreliable.
Children (for both primary and permanent teeth)	Use both **CO_2** and **EPT**	• Two tests more reliable than a single test.	

Note: CO_2 = carbon dioxide/dry ice pulp test; EPT = electric pulp test.

tissue changes. The greater the force used, the more likely it is that the patient will interpret the test as being painful, in which case the test results may not be meaningful.

The percussion test is performed by tapping on the crown of the tooth—usually on the incisal edge of anterior teeth and the occlusal surface of posterior teeth (Figure 8-25). Teeth can also be percussed from other directions (i.e., on other surfaces) that can be helpful in some cases of tooth fracture or periodontal disease. If the patient's history is suggestive of a crack undermining a cusp of a posterior tooth, then percussion of individual cusps from different directions may help to identify the problem cusp, although it should be noted that the lack of tenderness when doing this does not mean that there is no crack.

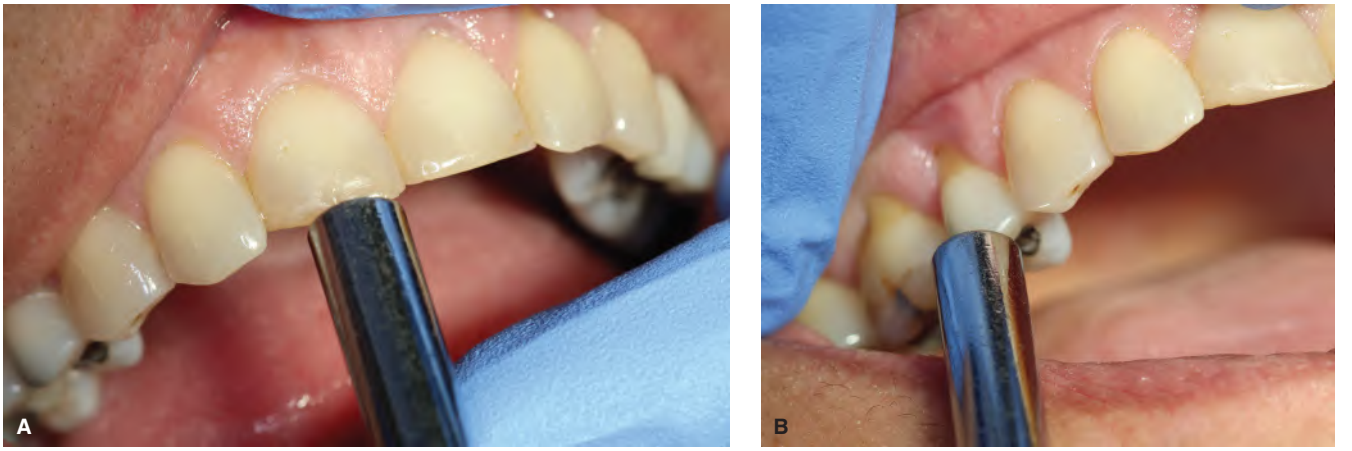

FIGURE 8-25 Performing a percussion test with the handle of a mirror. ***A.*** Percussing the incisal edge of an anterior tooth. ***B.*** Percussing the buccal cusp of a premolar tooth. All cusps of the teeth being examined should be tested in this manner.

As with all clinical tests for teeth, the tooth suspected as having apical periodontitis should not be the first tooth that is percussed. Other teeth in the same quadrant should be percussed first so the patient is aware of the nature of the test, and what is a normal response for their teeth, before the painful tooth is assessed. The clinician can also gauge how each individual patient reacts to the percussion test on normal teeth and compare this to the suspect tooth.

The results of percussion tests should be graded and noted as one of the following:

- **"Normal"**—the same feeling as that patient's other teeth, and with no tenderness or pain; this typically indicates clinically normal periradicular tissues (as long as there are no other signs or symptoms to suggest otherwise).
- **"Tender"**—a painful response when percussing the tooth; this typically indicates secondary acute apical periodontitis or a secondary acute apical abscess.
- **"Very tender"**—very painful response on percussion of the tooth; this typically indicates primary acute apical periodontitis or a primary acute apical abscess.
- **"Different sensation"**—the patient reports that the tooth feels "different" on percussion compared to how the adjacent teeth felt—it is not painful or tender; this typically indicates chronic apical periodontitis or a chronic apical abscess.
- **"Ankylosis sound"**—there is no pain but percussion produces a dull sound that is different from that produced when percussing the adjacent teeth—there is usually decreased mobility of the tooth also; this indicates the tooth has ankylosis and it may also have external replacement resorption (or possibly another form of resorption). A detailed history should help to determine why there is ankylosis.

All of these are "positive" responses. It is impossible to have a negative response to percussion. Hence, the test results for percussion should not be called "positive" or "negative." Instead, the earlier descriptors should be used when recording the results of a percussion test.

Palpation

Palpation of the soft and hard tissues overlying the tooth can provide information regarding the periradicular status of the tooth. In the normal, healthy situation, palpation does not cause any discomfort. However, if there is inflammation present, then palpation may produce discomfort or even pain. Pain is more likely when there is swelling of the mucosa overlying the tooth root.

Palpation is performed by gently feeling and pushing the mucosa overlying the teeth (Figure 8-26). It should be performed on both the labial/buccal and lingual/palatal aspects of the tooth. It should be performed over the periapical region as well as along the entire length of the tooth root (Figure 8-26). In most cases with apical periodontitis or an apical abscess, the labial/buccal aspect will have tenderness but this is not always the case and is dependent on the degree of inflammation and its spread toward the labial/buccal or lingual/palatal aspects. In addition to assessing whether palpation causes discomfort, the clinician should assess whether the region feels normal with respect to shape and size of any abnormality plus whether the region feels hard, soft, or fluctuant. The teeth and their surrounding tissues on the contralateral side of the mouth should also be visually examined and palpated as this can help to determine whether an unusual finding with palpation is a normal anatomical variation or not.

The absence of any discomfort during palpation does not necessarily mean that there is no disease present or that the tissues are healthy, for example, teeth with chronic apical periodontitis usually have no tenderness to palpation. Conversely, the presence of discomfort does not necessarily mean that a disease is present, for example, palpation over an anatomic structure such as the mental foramen may be uncomfortable for some patients.

The results of palpation tests should be graded and noted as one or more of the following:

- **"Normal"**—the same as the other teeth for that patient, with no tenderness or pain; this typically indicates clinically normal periradicular tissues (as long as there are no other signs or symptoms to suggest otherwise).

- **"Tender"**—painful response when palpating the mucosa overlying the tooth root and/or the periapical region; this typically indicates secondary acute apical periodontitis or a secondary acute apical abscess.
- **"Very tender"**—very painful response when palpating the mucosa overlying the tooth root and/or the periapical region; this typically indicates primary acute apical periodontitis or a primary acute apical abscess.
- **"Hard swelling or expansion"**—the tissues overlying the root and/or periapical region appear to be swollen or the cortical plate has expanded; the region feels hard on palpation.
- **"Soft swelling"**—the tissues overlying the root and/or periapical region are swollen; the region feels soft on palpation.
- **"Soft fluctuant swelling"**—the tissues overlying the root and/or periapical region are swollen and fluctuant; the region feels soft on palpation as though the swelling contains fluid.

Periodontal Probing

Periodontal probing is an essential part of the diagnostic process for pulp, root canal, and periradicular diseases. However, it is often not performed by clinicians especially if they do not think that the patient has generalized periodontal disease. However, the lack of periodontal probing may mean that an important piece of information is missed and this may in turn lead to misdiagnosis of the presenting complaint.

Periodontal probing is performed for two main reasons—first to assess the overall periodontal status of the dentition and in particular the individual tooth with the problem. The second reason is to check for the presence of a deep, narrow pocket that could be associated with an infected root canal system, a draining sinus, concurrent endodontic and periodontal diseases, or a crack in the tooth roots.

Since one of the main aims is to locate deep, narrow periodontal pockets, it is important to use a narrow periodontal probe. The ideal instrument for this is the WHO periodontal probe with a small ball-end (Figure 8-27). Such an instrument should be included in every examination kit.

When examining a tooth that is suspected as having pulp, root canal, or periradicular pathosis, that particular tooth plus several adjacent teeth should be periodontally probed as a minimum. Ideally the contralateral teeth should also be assessed as this will help to assess the overall periodontal

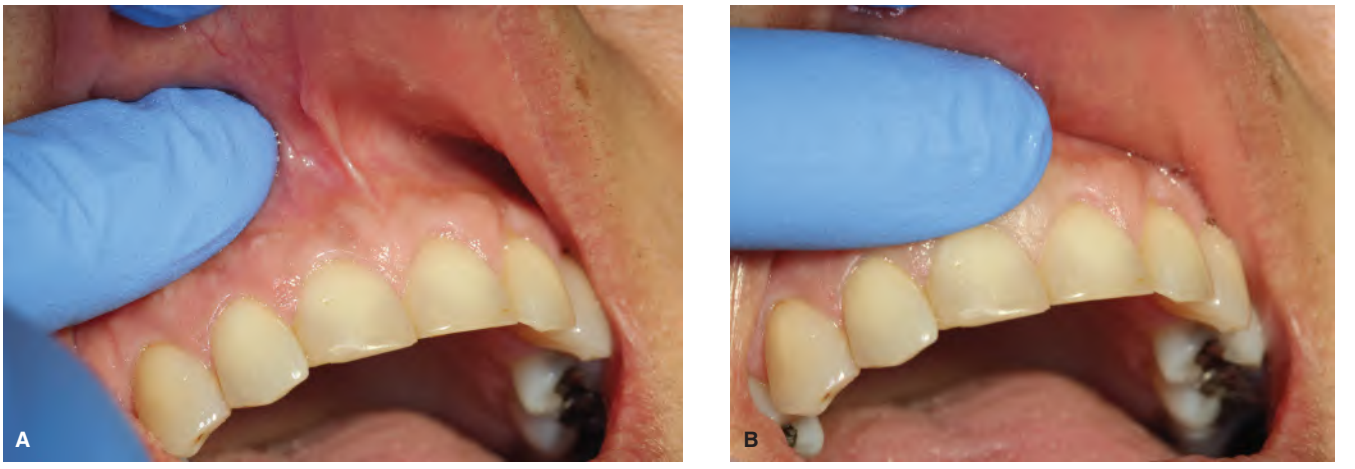

FIGURE 8-26 ***A.*** Palpating the periapical region of a tooth. ***B.*** Palpating the coronal third of the tooth root.

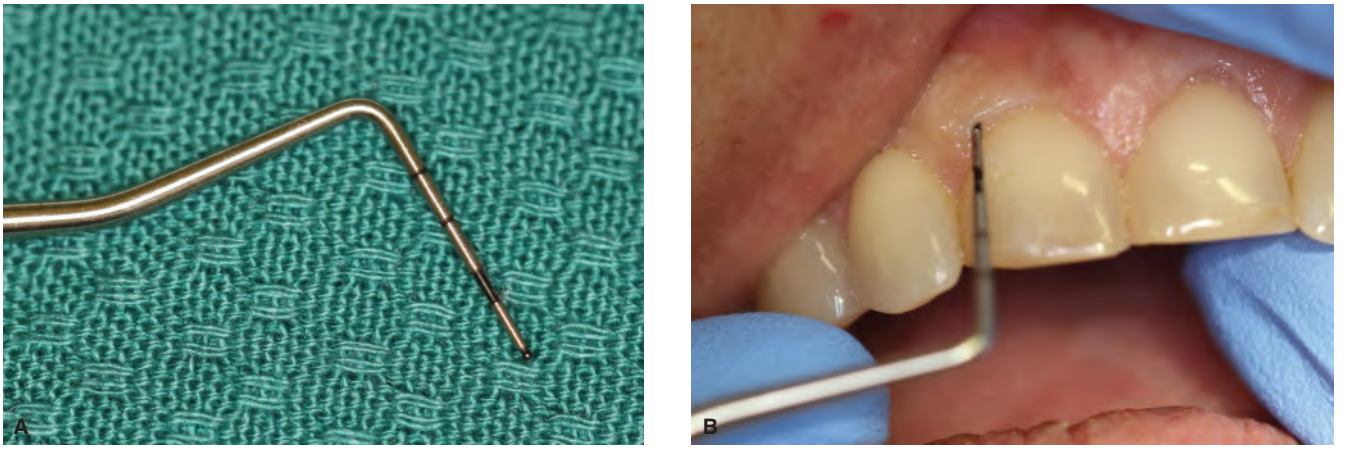

FIGURE 8-27 ***A.*** A WHO periodontal probe. ***B.*** Periodontal probing around a tooth; this should be performed in a step-wise manner around the entire circumference of the tooth.

status for that patient. Each tooth should be probed in a continuous, stepwise manner around the entire circumference of the tooth and not just at the typical six points (MB, B, DB, DLi, Li, and MLi) where pocket depths are recorded. If only these six points are probed, then a deep, narrow pocket situated between these points may be missed (Figure 8-27).

Care must be taken to probe the proximal surfaces (mesial, distal) of the teeth as these are the most common locations for deep, narrow pockets associated with cracks in teeth—since many cracks run in the mesiodistal direction. However, the proximal surfaces are particularly difficult to probe for deep, narrow pockets because of the presence of the adjacent teeth. This process can be further complicated by the presence of old restorations that may not be ideally contoured. Even in an ideal situation, the deep, narrow pockets associated with mesial or distal cracks are often in the centre of the proximal surface and it is usually impossible to angle a periodontal probe into such pockets when approaching from the buccal and lingual directions.

As a result of the difficulties associated with periodontal probing during a routine examination, teeth undergoing root canal treatment should have further periodontal probing done during the investigation phase of the treatment (see subsequently). In summary, investigation means removal of all restorations, caries and cracks from teeth. This is typically done under local anaesthesia and rubber dam isolation using a "cuff technique." Once the proximal aspects of the restorations have been removed, an ideal opportunity exists for re-evaluation of the proximal periodontal condition as the proximal surfaces can be directly probed without hindrance and also without causing discomfort for the patient as local anaesthetic has been administered.

The furcation of multirooted teeth should also be carefully probed from all directions since pocketing in these regions is sometimes a result of an infected root canal system where there are accessory canals running from the floor of the pulp chamber to the furcation region. Such teeth need to be distinguished from teeth with periodontal disease causing bone loss in the furcation.

The results of periodontal probing should be recorded to indicate that such probing was done and to provide information that can be compared with the findings of subsequent examinations after initial treatment, postoperatively, and when reviewing the treatment outcome.

The probing depths at the six points (MB, B, DB, DLi, Li, and MLi) assessed for periodontal disease should be recorded and a note made to indicate that no other pockets were found. Alternatively, if one or more pockets are found, then the exact location(s), the depth(s) and the nature of the pocket (narrow, broad) should be recorded in the patient's clinical record.

Mobility

The mobility of teeth should be assessed for two main reasons—first, it can provide information about the periodontal status of the tooth, and second, it indicates whether there has been bone loss which might be a result of an infected root canal system and involvement of the periradicular tissues. This information is required when assessing the prognosis of the tooth as well as when diagnosing the presenting condition.

Horizontal/lateral mobility implies movement in the buccolingual or mesiodistal direction. This can be assessed by using the handles of two instruments with one instrument on the labial/buccal surface and the other on the lingual/palatal surface (Figure 8-28); then push against the tooth with one instrument at a time. Movement can be detected by the instrument on the opposite side of the tooth. Repeat from the opposite direction and then do this several times to gauge how far the tooth moves in each direction.

Vertical mobility should also be tested and this is easily achieved by pushing the tooth from the occlusal or incisal direction using the end of the mirror handle. The depth of intrusion and subsequent extrusion of the tooth should be noted.

The Miller Index[49] is the most common system used to grade tooth mobility. There are various descriptions of this Index in the literature but the most common[50] is

- **Grade 0:** Normal (or physiological) mobility for the particular patient and when compared to that patient's other teeth.
- **Grade I:** Increased horizontal/lateral movement up to 1 mm.
- **Grade II:** Increased horizontal/lateral movement exceeding 1 mm.
- **Grade III:** Severe horizontal and vertical (depression) movement and/or rotation of the tooth.

Radiographs

Radiographic techniques and interpretation of the images will not be discussed in detail in this chapter. For more details, see Chapters 9 and 10. However, the following should be noted.

Radiography is an essential adjunct to, and should be an integral part of, every clinical examination when attempting

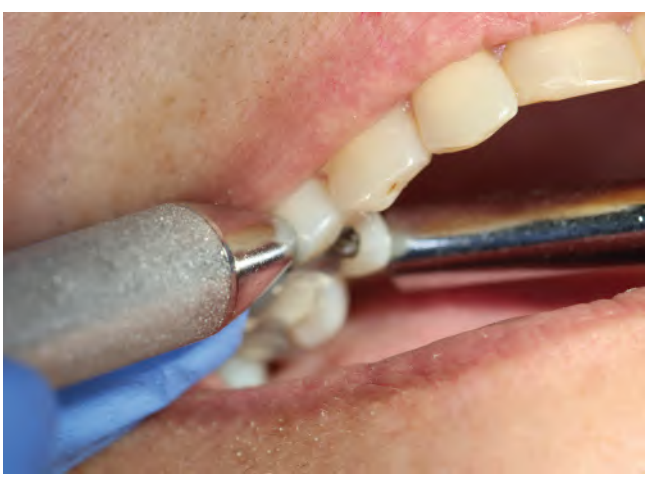

FIGURE 8-28 Testing the mobility of a tooth by concurrently using the handles of two instruments.

to assess the pulp, the root canal system and the periradicular tissues. Radiographs provide a wealth of information ranging from the causes of the various conditions, the anatomy of the involved tooth and its root canals, the periodontal status and the general state of the dentition in that region of the mouth (Figures 8-3 to 8-15, and 8-18). Radiographs are also essential when interpreting pulp sensibility test results since the lack of a response may be indicative of a tooth without a viable pulp (Figures 8-3 to 8-5, 8-8, 8-14, and 8-18), a tooth with PCC (Figure 8-8), a tooth with a root canal filling (Figures 8-6, 8-7, and 8-10 to 8-15) or a tooth that has had a pulpotomy performed in the past (Figure 8-12). These can only be determined by viewing a periapical radiograph of the tooth.

The most useful radiographic image for the diagnosis of pulp, root canal, and periradicular conditions is the periapical radiograph. Ideally, this should be taken using the modified parallel technique or the parallel technique (Figures 8-3 to 8-15, and 8-18). The former provides a more accurate image of the tooth since it reduces the inherent magnification (average 10%) associated with the parallel technique. The modified parallel technique involves using an increased vertical angle of approximately 15°, that is, the X-ray machine collimator is angled from a more apical direction by 15°.

Horizontal tube shift periapical radiographs are also useful at times to assess teeth with endodontic problems. The horizontal shift can be either from the mesial or distal direction, depending on which tooth is being assessed and what the possible problem is. A mesial shift is usually the best adjunct view for maxillary and mandibular central incisors and premolars, as well as for mandibular molars. Distal horizontal tube shifts are the most useful for maxillary and mandibular lateral incisors and canines, and for maxillary molars. Some teeth may even require both mesial and distal tube shifts; for example, maxillary molars, because of their typical anatomy with three divergent roots.

Bitewing radiographs are not sufficient on their own for the examination of teeth with pulp, root canal, and periradicular conditions since these radiographs do not show the entire tooth root and surrounding tissues. However, at times, bitewing radiographs are useful to assess whether a tooth has caries or open restoration margins, external invasive resorption, and bone loss due to periodontal disease. The angle from which bitewing radiographs are taken potentially provides a better view of the crown of the tooth and the most coronal part of the tooth root.

Panoramic radiographs do not provide sufficient "fine detail" for accurate assessment of pulp, root canal, and periradicular conditions. From an endodontic perspective, panoramic radiographs should only be considered as a screening method to provide an overview of the patient's oral and dental status. They may reveal areas of pathosis that were not evident prior to the radiograph being taken and they can also provide a better indication of the size of large lesions and their proximity to anatomical structures. A panoramic radiograph should be considered as a "screening radiograph" that can be used to direct the clinician to areas that require periapical radiographs for further assessment of the teeth.

Computed tomography (CT) scans can also be used as an adjunct to the assessment of teeth with pulp, root canal, and periradicular conditions. Such scans may be multislice CT or cone-beam CT (CBCT) scans. These provide a three-dimensional view of the teeth and surrounding tissues that may make them useful for some cases (e.g., where surgery is being considered). However, they are not required for the routine diagnosis of the vast majority of teeth with pulp, root canal, and periradicular conditions. Care also needs to be taken when assessing CBCT images as many teeth with clinically normal pulps may have widened PDL spaces or even periapical radiolucencies.[51] Such appearances may confuse the clinician and emphasizes the need for a thorough examination, and especially the use of pulp sensibility tests, before making a diagnosis.

Transillumination

Transillumination has been used as a diagnostic tool in medicine and dentistry for over 100 years. In 1916, Cameron[52] reported the use of the "Dentalamp," an instrument that he developed, to transilluminate teeth and surrounding structures to diagnose infection in the alveolar process and maxillary sinuses. He also advocated its use for the detection of subgingival calculus, caries in teeth (especially interproximal caries) and the extent of fillings in the crowns of teeth. Transillumination is also very useful during endodontic procedures, such as to help locate root canal orifices (especially where there has been calcification of the canals) and to observe the progress of root canal preparation. Transillumination is also an excellent and simple tool for assessing the progress and direction of a posthole preparation.

The concept of transillumination involves the use of an intense light source with concentrated white light rays projected as one beam of light. The intense light is deflected in different directions when the light rays reach tissues of different densities or of a different nature (e.g., fluid versus soft tissue, hard tissue versus soft tissue, etc.). Similarly, when a strong beam of light is shone through a tooth with a crack, the light will be deflected by the crack (Figure 8-29). This results in illumination of the ipsilateral side of the tooth only while the contralateral side remains dark. In addition, the crack becomes readily visible (Figure 8-30).

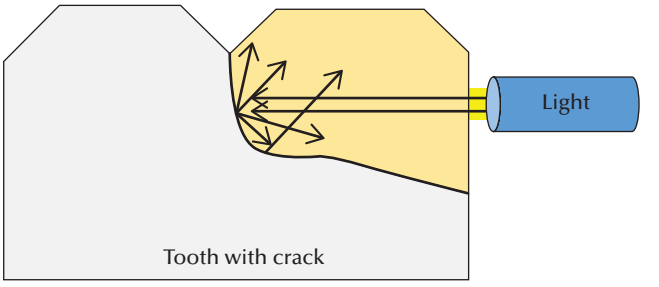

FIGURE 8-29 Diagrammatic representation of the effects of transillumination of a tooth with a crack. The light rays are deflected by the crack so the tooth structure on the other side of the tooth is not illuminated.

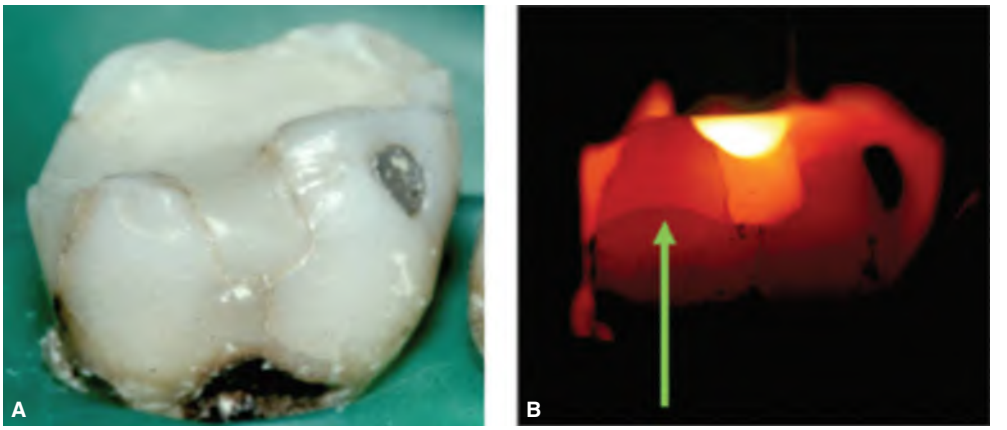

FIGURE 8-30 Transillumination. ***A.*** Visual examination under normal operating light. ***B.*** Examination of the same tooth using transillumination with a fibre-optic light source.

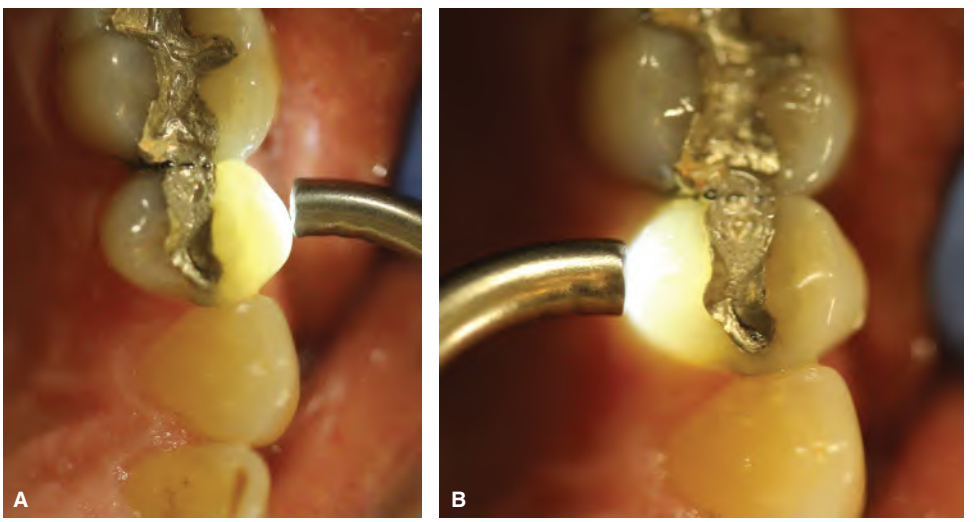

FIGURE 8-31 Transilluminating a maxillary first premolar tooth. ***A.*** Transilluminating from the buccal aspect. ***B.*** Transilluminating from the palatal aspect. The light should be moved around all surfaces and from different angles to fully assess the tooth for cracks in the enamel.

In the 1950s, the dental profession began to become aware of the effects of cracks in teeth.[53] Since then, many textbooks, especially older books,[54,55] and scientific articles[4,56–61] have advocated the use of transillumination to detect cracks. However, this technique has not been widely adopted or used regularly by most dentists despite being a very inexpensive, simple and reliable method for the detection of cracks, as well as for the other uses outlined earlier.

Several studies[62,63] have compared the use of transillumination to detect cracks in the apical third of teeth undergoing surgery or in an experimental model simulating the surgical treatment of root apices. Transillumination has been compared to other methods for detecting cracks such as the use of various dye solutions, magnification with microscopes and camera devices. Transillumination has consistently been reported to be the most reliable method with no added advantage from using a dye at the same time as transilluminating.

While these studies have assessed apical cracks, the results are consistent with a study by Jang[64] where cracks in enamel were assessed using transillumination, the normal dental operating light and a microscope. Transillumination revealed the highest number of cracks in the enamel of teeth. The normal operating light was better than the dental operating microscope which found the least number of cracks. The cracks found in these teeth using these methods were compared to the cracks seen on micro-CT scans of the same teeth. Transillumination had the highest sensitivity, the highest positive predictive value, the highest intra-observer reliability and the highest inter-observer reliability of the three techniques. Hence, transillumination is recommended as a routine examination procedure for all patients when assessing teeth for pulp, root canal, and periapical conditions as it can readily reveal cracks in teeth (Figure 8-31) and the cracks may be the pathway by which bacteria have entered the tooth to cause these conditions. Furthermore, transillumination should be performed when teeth are being investigated as the first stage of root canal treatment (see subsequently).

Several types of lights are commercially available. The most convenient light is the "diagnostic light probe" which is made by most companies that manufacture dental handpieces (Figure 8-32). These lights use the fiber-optic light systems within the dental unit or cart. A most convenient way to incorporate transillumination into everyday dental practice is to install an extra line/hose in the dental cart (Figure 8-32) so the light probe can be easily attached and used without having to remove a handpiece and swap with the light (especially when investigating teeth, as outlined subsequently). Other lights can also be purchased—some are battery operated (Figure 8-32) while others use the normal electrical power supply. The most ideal light has a small diameter tip (3.5–4 mm) as this concentrates the light on the tooth.

Biting Tests

The "biting test" can be a useful way to determine the tooth involved when a patient complains of pain associated with biting or chewing on a specific tooth. If the patient does not complain of such pain, then this test is not indicated. Percussion can also identify a tooth that is painful to biting or chewing but the biting test can sometimes identify a particular cusp that may be associated with the pain—such as when there is a crack undermining the cusp.

Pain associated with biting or chewing on a tooth can indicate several possible problems that vary in their management. Hence, it is important for the clinician to determine the nature of the pain caused by biting so the correct diagnosis can be established. In some cases, it can be difficult to distinguish between the different conditions that could cause pain to biting or chewing. The important criteria for each condition are:

- **Cusp flexure** (associated with a crack undermining the cusp)
  - ◊ The pain is usually sharp in nature and only momentary; it does not occur every time the patient bites on the tooth and typically only occurs when hard objects are bitten in a particular direction (with the direction of biting depending on the position and direction of the crack).
- **Apical periodontitis** (especially acute apical periodontitis)
  - ◊ The pain is not sharp although it can be severe (e.g., when primary acute apical periodontitis is present); the pain is present every time the patient bites or chews on the tooth.
- **Apical abscess** (especially an acute abscess)
  - ◊ The pain is not sharp although it can be severe (e.g., when a primary acute apical abscess is present); the pain is present every time the patient bites or chews on the tooth.
- **Lateral periodontitis** (associated with a crack, a fracture or a periodontal abscess).
  - ◊ The pain is not usually sharp but it can be sharp at times (e.g., when a root fracture is present); the pain is present every time the patient bites or chews on the tooth; the tooth will usually have a periodontal pocket and/or swelling on the lateral aspect of the tooth rather than apically; the tooth will have tenderness to palpation of the lateral aspect of the tooth rather than over the periapical region.

Many methods have been described for testing biting pain to identify a crack undermining a cusp but the most common, and generally considered to be the most useful, is the use of a specially-designed instrument such as the Tooth Slooth or the FracFinder (Figure 8-33). These instruments have a "pyramid-shape" on one side of the end of the instrument while the opposite side is flat (and may be serrated). The "pyramid" side has a concave tip which can be placed over one cusp at a time. The patient then bites on the flat or serrated surface with the opposing teeth (Figure 8-33). This allows the biting force to be placed on one cusp at a time in order to identify the cusp with the crack. The patient should be asked to bite down hard on the instrument and then to maintain pressure on it while the

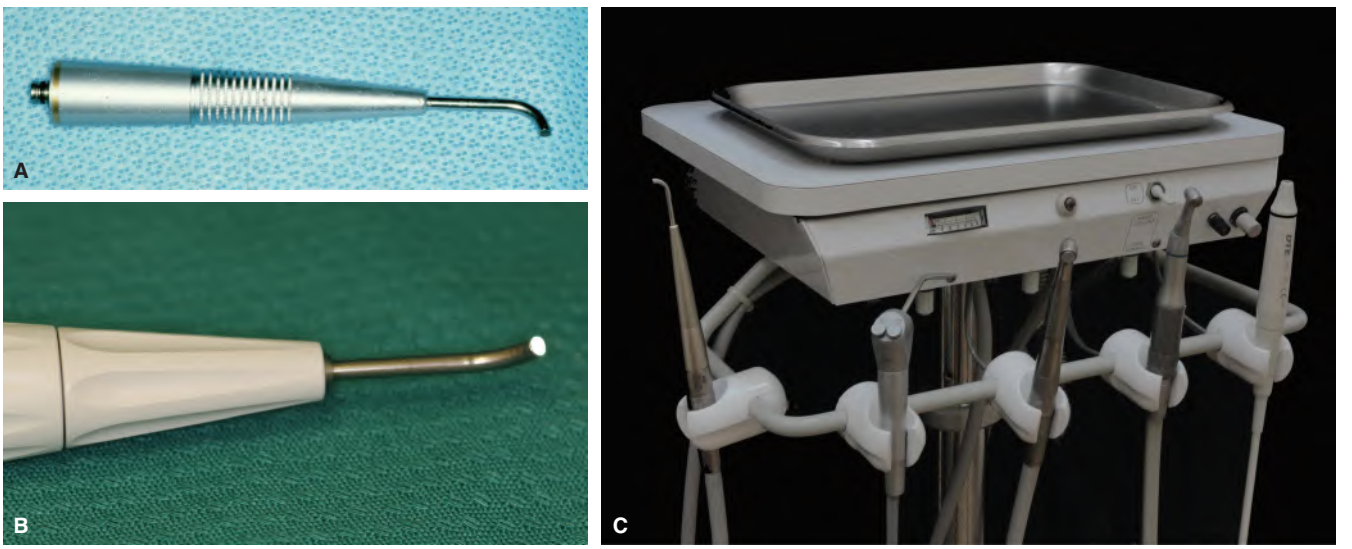

FIGURE 8-32 Examples of lights used to transilluminate teeth. ***A***. Light probe that attaches to the handpiece hose of a dental unit. ***B***. Battery-operated light. ***C***. A dental cart with an extra line/hose installed for the light probe.

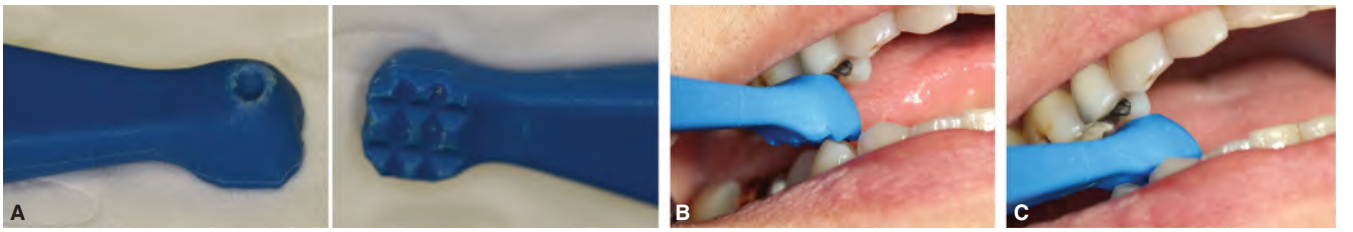

FIGURE 8-33 Performing a biting test with the FracFinder instrument. ***A.*** The left image shows "pyramid" side of the tip of the FracFinder instrument showing the concave depression that is placed over the cusp being tested; the right image shows the serrated surface on the opposite side of the tip onto which the patient bites with the opposing teeth. ***B.*** The FracFinder instrument being used on the buccal cusp. C. Testing the palatal cusp with the FracFinder instrument.

clinician manually moves it around in various directions to try and simulate the forces applied during chewing. The patient is then asked to stop biting on it by releasing the biting forces. Patients should be asked to identify whether they had any pain during the test procedure and, if so, when they felt the pain. The test is then repeated on each cusp (Figure 8-33) and on several teeth in the region of the patient's complaint. It can also be useful to repeat the test on any cusp(s) that gave an uncertain response or in order to confirm initial findings. The cusps of the teeth in the opposing arch should also be tested, as there may be referred pain.

In the past, much emphasis has been given to trying to identify teeth where the patient experiences pain on release of biting pressure with the belief that this was indicative of a crack undermining a cusp whereas pain on application of pressure did not indicate a crack. However, this concept is not supported by the literature as Abbott and Leow[65] reported that 68% of patients with cracks in teeth with reversible pulpitis had pain on application of pressure, 8% on release of pressure, 15% on both application and release of pressure and 9% did not have any pain on biting. Hence, pain on biting can occur any time and is not specific enough as a test on its own to indicate a crack in a tooth. It is just one of several symptoms that can occur and therefore the entire range of tests should be performed.

Biting pain associated with apical periodontitis and an apical abscess is easily reproduced and assessed by percussing on the tooth as this transmits the force of the percussion through the tooth to the PDL, and particularly the periapical PDL. Applying continuous pressure can also be useful at times and this is more indicative of pain associated with chewing rather than biting.

Biting pain associated with lateral periodontitis can be assessed through percussion in a vertical direction as well as from a lateral direction—such as from the labial/buccal or lingual/palatal. Palpation can also help locate the painful tissues. Lateral periodontitis is also likely to be associated with some increased mobility of the tooth.

Occlusal Analysis

The occlusion should be carefully checked to determine whether the affected tooth is in "traumatic occlusion." Excess occlusal forces or occlusal interferences can be either a cause of pulp inflammation[66,67] or they may be a result of periapical/periradicular conditions. They can also affect the healing of the periapical/periradicular tissues following trauma[68] to a tooth or after root canal treatment.[69]

Traumatic occlusion can cause either reversible or irreversible pulpitis in a tooth. Most cases will be reversible unless the traumatic occlusion has been present for a long time. A simple occlusal adjustment may be the only treatment required for many cases of reversible pulpitis. Hence, it is important to determine the cause of the pulpitis so appropriate treatment can be provided and to avoid unnecessarily removing pulps.

A tooth may be in traumatic occlusion as a result of apical periodontitis or an apical abscess. Primary acute apical periodontitis is an inflammatory condition with no space for the inflammatory cells and exudate since there has been no bone resorption—this in itself will lead to pain on biting but the inflammation may also cause the tooth to be extruded slightly from its normal position with the result that it may look longer than the adjacent teeth and it will be subjected to excess forces on biting and chewing. This can be quite painful for the patient. Likewise, a primary acute apical abscess has no space for the inflammatory reaction to occur and for the pus that is produced as part of the process of abscess formation. The resultant pain is usually very severe and often present even with just very light touching of the tooth (e.g., with the patient's tongue).

Chronic apical periodontitis, secondary acute apical periodontitis, a secondary acute apical abscess and other chronic periapical conditions are less likely to cause traumatic occlusion since bone has been resorbed to create space—this is evidenced by the presence of a periapical radiolucency. However, traumatic occlusion may be present in some cases or it may be result of periodontal disease and loss of bone support.

The occlusion should be carefully checked by examining the tooth for the presence of wear facets and by visually observing the tooth when the patient bites the teeth together. The patient should be asked to tap their teeth together several times in centric occlusion to see if there is any vertical movement of the affected tooth. If the tooth is depressed into its socket as the patient bites, then this indicates traumatic occlusion. The patient should then be asked to grind their teeth from side to side (lateral excursions)

and from front to back (protrusive movements)—if the tooth moves as the jaw moves, then this also indicates traumatic occlusion.

The occlusion can also be checked by placing a finger on the tooth while the patient taps and grinds the teeth in order to feel for fremitus or movement of the tooth. Finally, the occlusion should be checked for excessive contacts and "high spots" by using articulating paper (Figure 8-34) held in a pair of Miller's articulating forceps (Hu-Friedy Manufacturing Co., Chicago, IL, USA) while the patient bites and grinds the teeth laterally and protrusively.

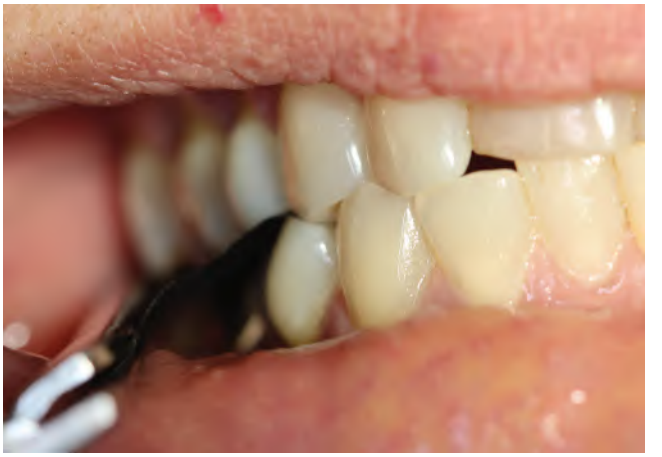

FIGURE 8-34 Assessing the occlusion with articulating paper held in a pair of Miller's articulating paper forceps.

Other Tests

Local Anesthetic Test

The principle of the local anesthetic test is to use a series of local anesthetic injections to anesthetize one small area at a time in an effort to localize the source of the patient's pain. This test should only be done when all the other tests have been performed (ideally several times) and the diagnosis is still uncertain. There are several limitations to this test and it is relatively uncommon that clinicians would need to perform this test for pulp or periapical conditions. It is unlikely to be required for the diagnosis of acute conditions since the symptoms are present and can usually be readily reproduced via the various tests outlined earlier. In acute pain cases, it is easy to stimulate the pain or to increase it by applying the stimulating factor (e.g., cold, heat, percussion, biting, etc.) to the tooth. However, in some cases where the patient has chronic pain, the local anaesthesia test might be helpful.

If considering the use of a local anesthetic test, it is essential that all other tests have been performed first as they cannot be done once local anaesthetic has been administered. If the local anesthetic test is to be helpful, then the clinician initially determines the general region of origin of the pain. Then, a small amount of local anesthetic solution can be injected into that region to see if the pain subsides. If the pain does not change, then another injection can be given in an adjacent region, and so on until the pain abates.

The most mesial tooth in the region should be anaesthetized first and then move distally with each subsequent injection. This works best in the maxilla where infiltration injections can be given but a large factor that restricts the usefulness of this test is the spread of anesthetic through the bone to potentially anaesthetize several teeth. In the mandible, infiltration injections are unreliable and therefore the local anesthetic test is also unreliable. PDL injections have been advocated but these injections are essentially intraosseous injections and the solution is known to spread to adjacent teeth. Hence, the local anesthetic test is not an ideal tool for localising the source of pain.

A thorough history and the full range of noninvasive diagnostics tests outlined earlier will usually lead the clinician to a diagnosis without the need for invasive tests such as the local anesthetic test. If a diagnosis cannot be established once all the tests have been performed, then the clinician should not attempt any irreversible treatment; instead the patient should be referred to a specialist practitioner for further assessment. Fortunately, most pulp, root canal, and periradicular conditions are readily diagnosed if the clinician collects the relevant information and has a good understanding of the disease processes.

Test Cavity

The concept of doing a so-called "test cavity" involves cutting a cavity in a tooth without the use of local anesthetic. This is essentially just another a form of pulp sensibility test as all it can do is indicate whether the pulp of the tooth is able to respond to a stimulus. However, this is an invasive procedure that damages the tooth structure and/or existing restoration. It is also very likely to make the patient quite apprehensive which may result in the patient perceiving pain even if the pulp is necrotic or the tooth is pulpless and infected—that is, false responses are very likely in this situation. Patients are unlikely to feel relaxed about such a procedure even when clinicians believe they have fully informed a patent of the nature of the test.

The procedure is invasive in that it involves cutting an unnecessary cavity into the tooth. This particularly applies if the pulp does respond to the procedure since the tooth now has a cavity that requires restoration. All restorations have a finite life span and every restoration has the potential to break down and allow bacterial penetration with the potential of eventual pulp disease. This can be avoided by not doing a "test cavity" in the first place.

The only potential results of a "test cavity" are that the patient feels pain or the patient does not feel pain. Neither of these is indicative of the status of the pulp as they do not indicate whether the tooth has a clinically normal pulp, reversible pulpitis or irreversible pulpitis if pain is felt. Alternatively if no pain is felt, then this does not indicate whether the pulp is necrotic and infected, pulpless and infected or even root filled and infected. Hence, the test is not specific enough in its outcome to lead to a diagnosis.

A further problem exists with teeth that have PCC. These teeth are unlikely to respond to cold pulp sensibility tests and they may or may not respond to an electric pulp test. Likewise, they may or may not respond to the "test cavity." If there is no response to the cavity preparation, yet there

is a viable pulp in the calcified canal, then unnecessary root canal treatment will be performed. The diagnosis of the state of calcified canals should be based on whether there are any periapical changes evident radiographically—if no changes are evident, then the pulp is likely to be clinically normal but just does not respond to pulp sensibility tests. Such a tooth does not require root canal treatment. Hence, the "test cavity" can be very misleading.

In addition to the abovementioned problems, a "test cavity" provides no additional information than what can be obtained with standard pulp sensibility tests such as a cold test or an electric pulp test—that is, it only indicates the pulp's ability to respond to a stimulus. In essence, the cutting of such a cavity without anesthetic is a barbaric procedure that should *not* be considered as a "test" and it simply causes pain and apprehension for the patient. This procedure really has no place in modern dentistry as better, simpler, noninvasive, and more reliable tests are available.

FINALIZING THE DIAGNOSES

All of the information obtained from the diagnostic process will lead to the definitive diagnoses and management plan in a stepwise manner, as outlined in Table 8-5. Usually, the examination and tests confirm the provisional diagnoses that the operator had formulated (based on the patient's history) prior to the examination. However, in some cases, the final diagnoses may vary from this.

There should always be more than one diagnosis whenever teeth with pulp, root canal, and periapical/periradicular conditions are being assessed since these aspects are interrelated. Pulp and root canal conditions are part of the same disease process, but at different stages of the process, and they cause periapical/periradicular conditions. Hence, all cases should have the pulp assessed if it is present, otherwise the root canal system must be assessed, and all cases must also have the periapical/periradicular tissues assessed. The state of each should be recorded even in the absence of disease as diagnosis also includes the assessment of health.

The clinical examination should also have identified the cause(s) of the disease processes. Hence, the four aspects of a complete diagnosis listed above—that is, the tooth involved, the state of the pulp (if present, otherwise the state of the root canal system), the state of the periapical/periradicular tissues, and the cause of the disease(s)—must be recorded in the patient's record.

MANAGEMENT PLANNING, PATIENT DISCUSSION, AND CONSENT FOR TREATMENT

Once the definitive diagnoses and cause(s) have been identified, the management options usually become obvious. Teeth with clinically normal pulps will not require any treatment but will usually need to be monitored and reassessed to ensure the pulp remains normal. Teeth with reversible pulpitis will normally require some form of conservative pulp therapy such as a direct pulp cap, a partial pulpotomy, a pulpotomy or partial pulpectomy. The actual treatment chosen will depend on

TABLE 8-5 Summary of the Examination and Diagnostic Processes for the Assessment of Pulp, Root Canal, and Periradicular Conditions, and Their Outcomes

Stage	Procedure		Outcome
History	Medical history Dental history Description of presenting complaint Details of previous treatment of tooth involved	→	• Provisional diagnoses of presenting conditions
Clinical examination	Extra-oral signs Intra-oral signs Individual tooth assessment, *e.g., mobility, periodontal probing, etc.* Restoration assessment	→	• Identify possible cause(s) • Provisional assessment of tooth status
Clinical tests	Pulp sensibility tests, *e.g., CO_2, EPT*	→	• Provisional diagnosis of the status of the pulp or root canal system
	Periradicular tests, *e.g., percussion, palpation*	→	• Provisional diagnosis of the periapical/periradicular status
Radiographic examination	Periapical radiograph(s) Other radiographs/images (if needed, *e.g., Bitewings, panoramic, CT, etc.*)	→	• Provisional diagnosis of the periapical status • Assess/identify/confirm cause(s)
Correlation of all findings	Combine the history, clinical examination, clinical tests and radiographic examination results	→	• Definitive diagnoses—tooth, pulp, or root canal system, and periapical/periradicular tissues + Cause(s) of the condition(s)
Management plan	Tooth Investigation, *i.e., remove all restorations, caries, cracks, etc.*	→	• Confirm the definitive diagnoses and cause(s)
	Reassess amount of tooth structure and the overall prognosis/longevity of tooth + restoration	→	• Finalize management plan and continue treatment if the canals can be negotiated and tooth is suitable for further restoration

(Adapted from Abbott[3,5] and Abbott and Yu.[11])

the restorative dental requirements of the tooth. Some cases of reversible pulpitis may even require root canal treatment if they are found to need some form of intra-radicular retention to help retain the restoration but this can only be determined when the tooth has been investigated as described subsequently.

Root canal treatment (or retreatment) will generally be the first choice of treatment to manage irreversible pulpitis and most other root canal and periapical/periradicular diseases (Tables 8-6 and 8-7) although some cases may require, or be more suitably managed with, periapical surgery. Furthermore, some teeth will require extraction as they are not suitable for root canal treatment and/or further restoration of the tooth. In a study by Al-Dhufairi and Abbott,[70] 11.8% of 5775 teeth with pulp, root canal, and periapical/periradicular problems were recommended for extraction following a thorough examination. They were recommended for extraction because there was insufficient tooth structure (8.3%) or they had significant cracks (3.5%) that rendered them unsuitable for further restoration.

Extraction is always an option for teeth with irreversible pulpitis and most other root canal and periapical/periradicular diseases, even if the tooth is suitable for further treatment, as extraction will lead to predictable resolution of the symptoms and healing. Some patients may choose to have the tooth extracted for a variety of reasons. As with all clinical procedures, the advantages and disadvantages of retaining the tooth versus extracting it should be discussed with the patient so he/she can make an informed choice.

The examination findings and the diagnoses must be discussed with the patient along with an outline of the management options. The patient needs to understand what has happened to the tooth, why it has happened and what treatment can be done to rectify the problems. The best approach to discussing this is to advise the patient of the general nature of the problem—such as whether it is an

TABLE 8-6 Summary of Endodontic Treatment Strategies for the Various Pulp and Root Canal Conditions

Clinically normal pulp		NIL treatment required
Reversible pulpitis—acute or chronic		Conservative pulp therapy then reassess healing response
Irreversible pulpitis—acute or chronic		Routine root canal treatment with anti-inflammatory medications then reassess healing response, or extraction
Pulp necrobiosis		Routine root canal treatment with anti-inflammatory and antimicrobial medications then reassess healing response, or extraction
Pulp necrosis	No signs of infection	No treatment required—monitor and reassess periapical tissues for signs of inflammation (which would indicate an infected root canal)
	Infected	Routine root canal treatment with anti-inflammatory and antimicrobial medications then reassess healing response, or extraction
Pulpless, infected root canal system		Routine root canal treatment with anti-inflammatory and antimicrobial medications then reassess healing response, or extraction
Pulp atrophy		NIL treatment required
Pulpal canal calcification		Treatment will be determined by the condition of the pulp/root canal system (e.g., irreversible pulpitis; pulpless, infected root canal system; etc.)
Pulp hyperplasia		Routine root canal treatment with anti-inflammatory medications then reassess healing response, or extraction
Previous root canal treatment		
No signs of infection		No treatment but monitor and reassess unless restoration being replaced ➔ then do root canal retreatment and reassess healing response
Infected root canal system		Root canal retreatment with anti-inflammatory and antimicrobial medications then reassess the need for periapical surgery, or extraction, if the canal is still infected or if there are signs of on-going periapical pathosis
Technical standard	Inadequate	No treatment but monitor unless the restoration is being replaced or the root canal system is infected ➔ then root canal retreatment and reassess healing response
	Adequate	No treatment but monitor unless the restoration is being replaced or the root canal system is infected ➔ then root canal retreatment and reassess healing response
Other findings	Perforation	Root canal retreatment and repair the perforation from an internal approach if possible; then reassess the need for periapical or periodontal surgery, or extraction—if there are signs of on-going periapical and/or periodontal pathosis
	Missed canal	Root canal retreatment then reassess the need for periapical surgery, or extraction, if the root canal system is still infected or if there are signs of on-going periapical pathosis

Note: recommendations for endodontic treatment and retreatment are dependent on the tooth having sufficient tooth structure remaining to enable it to be adequately restored again. (Adapted from Abbott and Yu.[11])

TABLE 8-7 Summary of Endodontic Treatment Strategies for the Various Periapical/Periradicular Conditions

Clinically normal periapical/periradicular tissues	NIL treatment required
Apical periodontitis • Primary acute • Secondary acute • Chronic	Routine root canal treatment with anti-inflammatory and antimicrobial medications then reassess healing response, or extraction
Apical abscess • Primary acute • Secondary acute • Chronic	Routine root canal treatment with anti-inflammatory and antimicrobial medications then reassess healing response, or extraction
Facial cellulitis	Immediate systemic antibiotics (intra-venous, intra-muscular), plus routine root canal treatment with anti-inflammatory and antimicrobial medications then reassess healing response, or extraction
Extra-radicular infection	Root canal treatment or retreatment with anti-inflammatory and antimicrobial medications then reassess the need for periapical surgery, or extraction, if the canal is still infected or if there are signs of on-going periapical pathosis
Foreign body reaction	Root canal treatment or retreatment with anti-inflammatory and antimicrobial medications then reassess the need for periapical surgery, or extraction, if there are signs of on-going periapical pathosis
Periapical cyst • Pocket • True	Root canal treatment or retreatment with anti-inflammatory and antimicrobial medications then reassess the need for periapical surgery, or extraction, if there are signs of on-going periapical pathosis
Periapical scar	NIL treatment required but regular radiographic review essential to monitor and reassess

Note: recommendations for endodontic treatment and retreatment are dependent on the tooth having sufficient tooth structure remaining to enable it to be adequately restored again. (Adapted from Abbott.[3])

inflammatory condition (e.g., pulpitis) or an infection (e.g., pulpless and infected root canal system) that also has an inflammatory component (e.g., chronic apical periodontitis). Simple, although not too simplistic, and meaningful terms should be used. For example, the pulp should not be called the "nerve" of the tooth as this word conjures an image or expectation of pain in the patient's mind when advised that it needs to be removed. In many cases, there will be no pulp tissue to remove anyway (e.g., pulpless, infected teeth) and therefore the clinician should not mislead the patient by saying the pulp or "nerve" needs to be removed.

An important part of the discussion is to inform the patient of the reasons why the tooth has the problem. Typically pulp, root canal, and periradicular conditions develop as a result of bacteria entering the tooth. Hence, the patient should be advised about this and the specific way that the bacteria have entered the tooth in question. The majority of cases are a result of restorations breaking down, caries and/or cracks in the tooth even when these may not be visible or entirely evident during the investigation. If the patient understands this, then he/she will be more likely to realize that the existing restoration(s), caries and cracks will need to be removed while the tooth is being investigated, as outlined subsequently.

The discussion should also include an outline of the alternative treatments that may be available for the particular problem. This will typically include extraction and possible replacement with a prosthesis. Some cases may involve a discussion about reversible pulpitis and conservative pulp treatment, while others may involve discussions about root canal retreatment and/or periapical surgery. The exact nature of the discussions will depend on the diagnoses, the condition of the involved tooth, as well as the particular patient, their overall oral status and their interest in retaining their teeth. While a discussion of all possible options for managing the problem is necessary, the patient should not be misled or given false expectations about a procedure that is unlikely to have a good outcome or to provide a predictable long-term result for the patient. For example, periapical surgery should not be offered if the root canal system is infected and the coronal restoration is unsatisfactory.

Once the patient has had a full explanation of the problem and the proposed treatment, they can then provide informed consent so the treatment can commence. However, patients should understand that the initial stage of treatment (i.e., the tooth investigation—see subsequently) is also an important part of the diagnostic process as the tooth cannot be fully assessed until the restorations, caries and cracks have been removed. The overall treatment plan cannot be finalized until this procedure has been performed.

TOOTH INVESTIGATION

Tooth investigation is the procedure whereby all existing restorations, caries and cracks are removed from the tooth (Figures 8-35 and 8-36) as the initial stage of root canal treatment.[4] Despite seeming to be more a part of the treatment procedure, it should be first considered as part of the diagnostic process since it provides valuable information about the tooth, the conditions being treated and the prognosis of the tooth. This is not a new concept and it has been advocated by many authors for many years[55,71,72] but it has not been regularly practiced by the profession. In 1985,

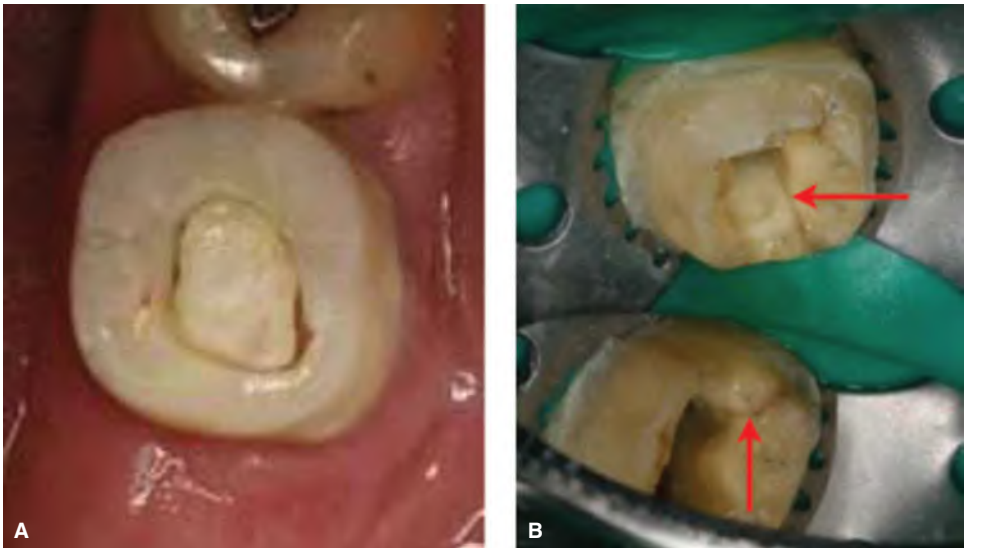

FIGURE 8-35 Investigation of a tooth. ***A.*** Preoperative view of the tooth with a full coverage crown restoration. A dentist had been attempting root canal treatment but the patient continued to have symptoms. ***B.*** After removing the crown restoration, a crack was evident (see arrows) This crack was allowing continual microbial entry into the tooth and therefore symptoms continued. The tooth was recommended for extraction due to the extent of the crack. (Courtesy of Dr. Edward Houston, Brisbane, Australia.)

Marshall et al.[55] stated that *"prior to any endodontic treatment, and after all coronal restorations and decay are removed, the remaining tooth structure should be ere-examined with a fiber-optic light for fractures and perforations. Teeth with vertical fractures and perforations may be untreatable."* Today, with better definitions, "fractures" could be interpreted to mean both cracks and fractures, and "untreatable" implies that they are not salvageable and require extraction. Marshall et al.[55] also stated that the suitability of a tooth for further restoration *"depends on the amount of sound tooth structure remaining"*—another concept that remains true today.

There are several aims of tooth investigation:

- To remove the cause(s) of the disease(s) that are present in the tooth and the surrounding tissues,
- To assess the remaining tooth structure to determine if the tooth is suitable for further restoration after the root canal treatment has been completed,
- To assess whether root canal treatment will be feasible (e.g., can the canals be located and negotiated, can the existing root filling material be removed in retreatment cases, etc.).
- To better assess the long-term prognosis of the tooth, and
- To allow construction of an interim restoration that will prevent bacterial penetration into the tooth during the root canal treatment and until a definitive restoration is placed.

It is well accepted that bacteria are the major cause of pulp, root canal, and periradicular conditions. However, the pathway of entry of the bacteria into the tooth is often ignored. In order to achieve the highest possible rate of favourable outcomes of the treatment of any disease, the first principle is to remove the cause(s) of the disease. This applies to root canal treatment as much as it does to any other treatment. Since the most common pathways of entry for bacteria are via caries, cracks and the breakdown of restoration margins, it is essential to first identify the cause(s) of the diseases and then to remove them as part of the treatment.

Abbott[4] has demonstrated that the standard visual clinical examination and periapical radiographs do not provide sufficient information to accurately assess the status of the teeth and the pathway(s) of bacterial penetration that commonly cause pulp, root canal, and periapical diseases. He examined a series of 245 teeth in 220 consecutive patients referred for endodontic treatment using the standard visual clinical examination procedures and tests plus periapical radiographs. In particular, evidence of restoration breakdown, caries, cracks, and fractures was noted. The examination procedure included a visual inspection with probing of restoration margins, pits, fissures and grooves, periodontal probing, percussion, palpation, and transillumination of the teeth with a fibre optic light shone from numerous different directions. The restorations were then removed from the teeth and the clinical examination was repeated with the same methods. Examination of the teeth without restorations in place revealed 2.5 times as many teeth with evidence of marginal breakdown of the restorations, 2.3 times as many teeth with caries and 2.6 times as many teeth with cracks than what could be seen preoperatively. Hence, the value of tooth investigation is clear in that it not only reveals the common cause(s) of the diseases but it removes them from the tooth.

Teeth with infected root canal systems must have a pathway of entry for the bacteria to invade the tooth. This same pathway is also a likely pathway of entry for nutrients from the oral cavity (food, drinks, etc.) to enter the tooth and help the bacteria to establish colonies so they can survive

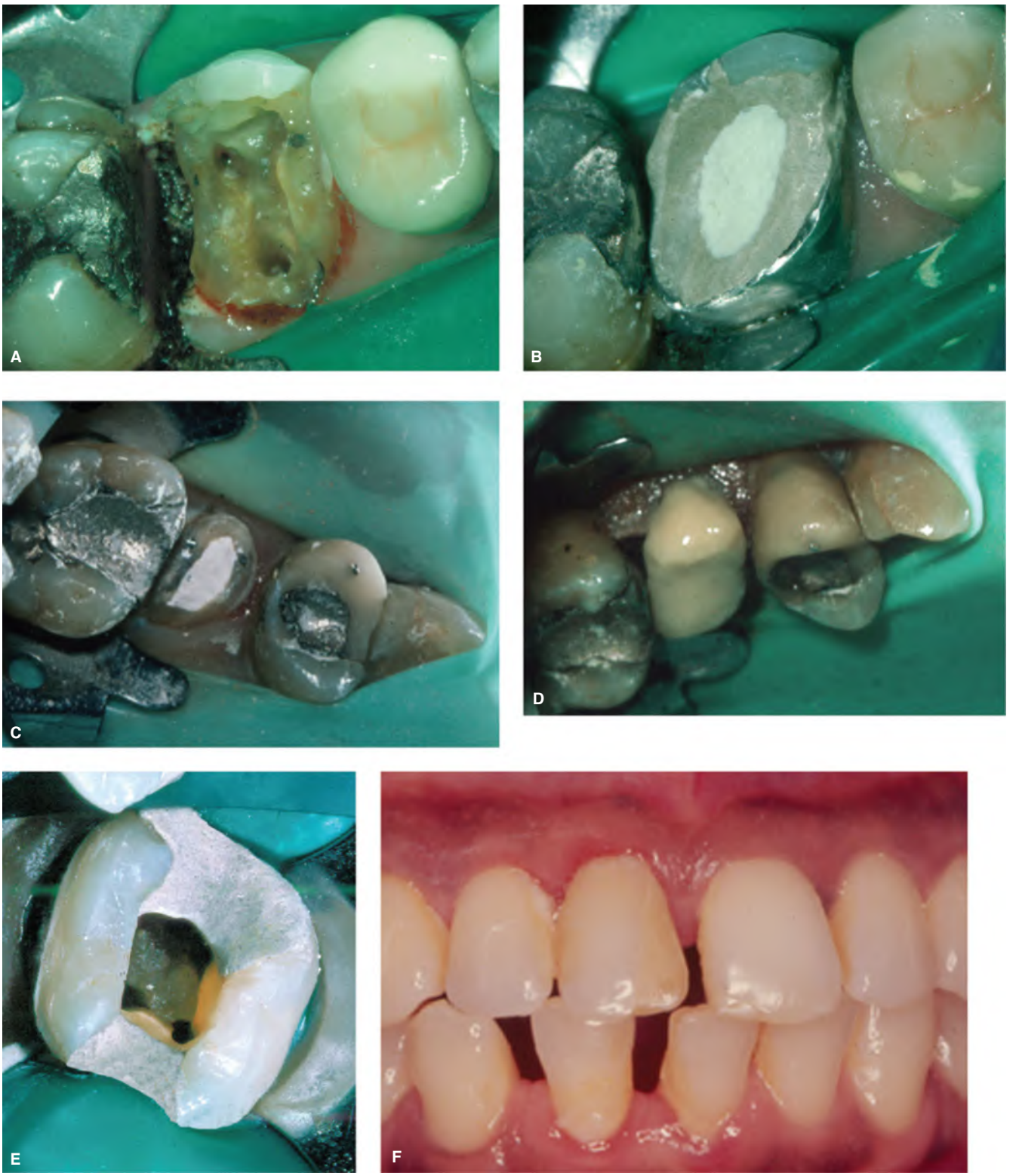

FIGURE 8-36 Interim restorations placed following investigation of the teeth. ***A.*** A maxillary molar after tooth investigation, and ***B.*** following placement of a stainless steel band and Ketac Silver interim restoration. ***C.*** A maxillary premolar after tooth investigation and placement of an intracanal medicament and Cavit in the pulp chamber, and ***D.*** Following placement of a stainless steel band and Ketac Fil interim restoration. The Ketac Fil has overlaid the buccal aspect of the band as a veneer to provide an esthetic interim restoration. ***E.*** Ketac Silver without a band with subsequent access cavity in a mandibular molar tooth. ***F.*** Ketac Fil placed as an interim restoration to restore the fractured mesioincisal portion of the right maxillary incisor tooth while root canal treatment was performed.

within the root canal system. However, the pathway of entry may not always be clinically evident as demonstrated by Kwang and Abbott[73] who used scanning electron microscopy to examine the fitting surfaces of 30 restorations that had been removed from teeth with infected root canal systems. In order to be included in the study, the restorations had to be "clinically satisfactory"—that is, they had no visible signs of marginal breakdown when examined—and the teeth had no caries or cracks that could have allowed bacteria to enter and infect the root canal system. All 30 restorations were completely lined by bacterial biofilms that consisted of a wide range of bacterial morphological types—cocci, filamentous rods, spirochaetes, and so forth. The results clearly demonstrate that it is not possible to accurately assess restored and infected teeth for the presence of bacteria under the restorations. This study further supports the concept of tooth investigation and the need to remove all restorations prior to commencing root canal treatment so all possible pathways of bacterial entry are removed.

Tooth investigation is also a valuable process that leads to better case selection since the remaining tooth structure itself can be examined and assessed rather than trying to evaluate the tooth with the restoration in place (Figures 8-35 and 8-36). Al-Dhufairi[70] has reported that a higher proportion of teeth were referred for extraction at the tooth investigation stage than at the initial examination stage. Al-Dhufairi reviewed the decisions made by 13 operators (specialist endodontists and postgraduate endodontic students) regarding 5775 teeth. When these teeth were examined using the standard clinical procedures outlined earlier, 11.8% of the teeth were referred back to the referring dentist for extraction—8.3% were recommended for extraction due to an obvious lack of tooth structure and 3.5% were recommended for extraction due to the presence of cracks. Both of these findings rendered the teeth unsuitable for root canal treatment and further restoration. Then, 4218 of the original set of teeth were investigated by removing the restorations, caries and cracks—this revealed that 11.9% had insufficient tooth structure and 7.3% had cracks which made them unsuitable for root canal treatment and further restoration. Overall, extraction was recommended for 19.2% of the teeth that were investigated—that is, approximately one in every five teeth were not suitable for root canal treatment and further restoration. If these teeth had not been investigated then they would have had an unrecognized poor, or hopeless, prognosis and the patient would have had inappropriate treatment with subsequent loss of the tooth within a short period of time.

A systematic review by Ng et al.[74] involving 14 studies with over three million teeth that had undergone root canal treatment reported a mean survival rate of just 86% over two to three years. This meant that 14% of these teeth had been extracted within a very short period of time following the treatment. This study did not account for teeth that may have been extracted after four to five years (or longer) and it did not include teeth that required further root canal treatment due to persistent periapical disease which is likely to be a result of not removing the cause of the disease or not having sufficient tooth structure to enable an adequate restoration to be placed. While this figure of 14% is slightly less than the 19.2% reported by Al-Dhufairi,[70] it is reasonably similar and supports the concept that tooth investigation should be done in order to improve case selection and only treat those teeth that have a good prognosis.

A further study by Ng et al.[75] assessed the reasons for extraction following root canal treatment. This was a prospective analysis over two to four years of 759 teeth that had root canal treatment and 858 teeth that had root canal retreatment. In both groups, approximately half of the teeth that were extracted had a "tooth structure" problem which led to either fracture of the tooth (28.6% and 29.3%, respectively) or "failure" of the restoration (22.9% and 22%, respectively). The other major factor leading to extraction was an "endodontic problem" in 28.6% and 39.0%, respectively, of the teeth. Analysis of other studies[76–79] reveals similar findings that approximately half the teeth requiring extraction within just a few years of having root canal treatment have either fractured or the restoration has not been adequate, both of which are "tooth structure" problems. The results of these studies are also highly suggestive of the teeth not being adequately assessed prior to the root canal treatment. This is a very likely consequence of not removing existing restorations, caries, and cracks.

When the concept of tooth investigation was applied to a series of 432 teeth undergoing root canal retreatment of infected teeth, Abbott[80] reported that 423 teeth (97.9%) had a favourable outcome of the treatment when assessed for up to five years or until periapical healing was confirmed radiographically. While it is recognized that there are many factors that can affect the outcome of root canal treatment, the ability to select only those teeth that had sufficient tooth structure and no obvious cracks is likely to be a major contributor to this result which is a much higher rate of favourable outcomes than reported in most studies regarding root canal retreatment of infected teeth.

The abovementioned studies highlight the need to investigate teeth as part of the diagnostic process. This allows better case selection and avoids patients having inappropriate and expensive treatment for no long-term gain. When presenting the concept of tooth investigation to patients, they should initially be advised of the reasons why the tooth requires investigation (i.e., to remove the causes of the diseases and to assess the remaining tooth structure). They should then be advised that there are two possible outcomes of the investigation:

- The tooth *is* suitable for root canal treatment and further restoration (in which case the root canal treatment can be continued), or
- The tooth is *not* suitable for root canal treatment and further restoration (in which case the tooth will require extraction).

A patient fully informed and forewarned of these possible outcomes is more likely to understand and accept the need for the restorations, and so forth to be removed (i.e., tooth investigation). This essential part of the diagnostic process then becomes the first stage of the endodontic management of the tooth if the tooth is proven to be suitable for treatment.

Apart from the reasons and advantages outlined earlier, tooth investigation also markedly simplifies the initial root canal treatment since it allows uninhibited access to the pulp chamber and root canals. As stated by Ingle in 1965,[71] *"it is much easier to complete the radicular preparation through an open cavity than through the restored crown. As a matter of fact, the more of the crown that is missing, the easier the canal preparation becomes."* This is still true today. Furthermore, Ingle stated in 1985[72] *"complete caries excavation is imperative both to diagnose the condition and to plan proper treatment."* Again, this is still true today, particularly to *"plan proper treatment"* which should not just be planning of the endodontic treatment but it should also include planning of the restoration.

The two key aspects to consider about the restoration are first should the tooth be restored, and second, can it be restored adequately with a good long-term prognosis. These factors can only be determined by assessing the tooth structure that remains (Figures 8-35 and 8-36). Unfortunately, in general, the dental profession has not followed this concept and have tended to cut access cavities through existing restorations. However, this approach ignores the cause(s) of the disease(s) that are not removed—this in turn leads to continuation of the disease process as bacteria can continually enter the tooth and root canal system. This approach also hinders the ability of the operator to assess the long-term prognosis of the tooth as the amount and quality of remaining tooth structure cannot be seen and therefore cannot be assessed.

In summary, removal of all existing restorations, caries and cracks is essential since these are the most likely pathways of entry for the bacteria causing the pulp, root canal, and periapical conditions. This approach also allows the diagnoses to be confirmed, allows assessment of the amount and quality of the remaining tooth structure, allows assessment of the tooth's suitability for further restoration, and improves the prognosis for healing and longevity of the tooth. That is, better and appropriate case selection becomes possible when the tooth is investigated in this manner.

Once a tooth has been investigated, a comprehensive interim restoration will be required. This can usually be achieved with relatively simple techniques[81] such as by using glass ionomer cements with or without stainless steel bands to help retain the restorative material (Figure 8-36), especially at subsequent appointments when access is gained to the pulp chamber and root canals.

RECORD KEEPING

The final stage of the "Diagnostic Process" is to thoroughly record all the details of the history and clinical examination, the test results, a radiographic report, the definitive diagnoses, the cause(s) of the disease(s), the management options, the discussions with the patient, the patient's consent, the anesthetic used, the findings of the investigation procedure, the procedures provided during the investigation stage, the materials used (e.g., irrigants, medicaments, restoration, etc.), other relevant details (e.g., number and location of canals, files used, etc.), recommendations for further restoration,[4,5] final advice regarding retention or extraction of the tooth, any postoperative medications prescribed or advised, and the future treatment needs.[82] In summary, a through clinical record is required! This may be facilitated through the use of specifically designed paper forms or computerized screens that help to ensure that details are not omitted.

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Chapter 9

Imaging Devices and Techniques

The success of endodontic treatment is dependent on the identification of teeth requiring such treatment, and then the recognition of the root canal system, so that it can be properly cleaned, shaped, and obturated. Endodontic treatment relies on a series of images made at different stages of the treatment: preoperatively, periodically during instrumentation and obturation, and postoperatively. These images provide important clues about hard and soft tissues of the teeth, including the pulp chamber and root canals. They also facilitate the estimation of root canal length and appropriateness of obturation. Furthermore, they aid in detecting pathoses, provide useful information of the periradicular region, and assist in evaluating posttreatment healing.

Imaging technology has gone through many advances in the past several years. This chapter describes the most pertinent imaging technologies related to endodontics, traditional, and advanced that have impacted the way endodontic treatment is being performed.

A. Analog Radiography

Richard E. Walton, Manuel R. Gomez

Radiographs are the "eyes" of the dentist when performing many procedures. They are essential for diagnosis and treatment planning, determining anatomy, managing treatments, and assessing outcome. Now considered essential, the use of the X-ray was not available to the dental profession until early in the last century.

No single scientific development has contributed as greatly to improved dental health as the discovery of the amazing properties of cathode rays by the German professor Wilhelm Konrad Roentgen in 1895. The significant possibilities of their application to dentistry were seized upon 14 days after Roentgen's announcement. Dr. Otto Walkoff took the first dental radiograph for himself.[1] In the United States, within five months, Dr. William James described Roentgen's apparatus and displayed several radiographs. Three months later, Dr. C. Edmund Kells gave the first clinic in the USA on the use of the X-ray for dental purposes. Three years later (1899), Kells was using the X-ray to determine tooth length during root canal therapy. "I was attempting to fill the root canal of an upper central incisor," Kells later said. "It occurred to me to place a lead wire in this root canal and then take a radiogram to see whether it extended to the end of the root or not. The lead wire was shown very plainly in the root canal."

One year later (1900), Dr. Weston A. Price "called attention to incomplete root canal fillings as evidenced in radiographs." By 1901, radiographs were used to check the adequacy of root canal fillings.[2] Price is credited with developing the bisecting angle technique, whereas Kells described what today is called the paralleling technique, made popular some 40 years later by Dr. Gordon Fitzgerald.

Although these early attempts were rarely of diagnostic quality, they were the beginning of a new era. For the first time, dentists could visualize hidden disease as well as see the accumulation of past dental treatment; therapy done without knowledge of what lay beneath the gingiva. Yet even today, with all of the technical refinements, the sleekness of operation, and the reduction of hazards, a discouraging segment of our profession continues to deprive their patients by failing to use radiography to its full potential. Dentists often under- or overuse, under- or overinterpret radiographs or fail to use careful and appropriate techniques.

EVOLUTION OF RADIOGRAPHY

Endodontics evolved and upgraded as a dental discipline throughout the 20th and into the 21st centuries. Much of the improvement was because of procedural advances as well as technical innovations. This section is focused on traditional (analog) radiography. Other sections that follow in the chapter will include newer imaging methodologies. These technologies have many advantages and also some shortcomings and limitations. In many parts of the world, including the United States, these newer techniques are neither practical nor available nor affordable.

Importantly, traditional analog techniques and procedures, utilized for decades, are still effective. In fact, in many dental offices and clinics this is the only technique utilized. Therefore, not only analog radiograph is useful but also most of the analog aspects are applicable to digital and somewhat to cone beam computed tomography (CBCT). Therefore, this section reviews these basic analog methods and applications.

APPLICATION OF RADIOGRAPHY TO ENDODONTICS

In endodontics, radiographs have the following several essential functions:

1. Aid in diagnosis of hard tissue alterations in the teeth and periapical structures and proximity of anatomic structures.
2. Determine the number, location, shape, size, and direction of roots and root canals.[3]
3. Assess anatomy, size, and alterations in the pulp chamber.
4. Detect procedural errors such as perforations, ledges, transportation, and instrument separation.
5. Locate root tips prior to surgery.
6. Estimate and confirm the length of canals.
7. Localize hard-to-find, or disclose unsuspected, pulp canals by examining the position of an instrument within the root (Figure 9A-1).
8. Aid in locating a pulp space markedly calcified and/or receded (Figure 9A-2).
9. Determine the relative position of structures in the facial–lingual dimension.
10. Confirm the position and adaptation of master cones.
11. Aid in the evaluation of obturation.
12. Facilitate the examination of soft tissues for tooth fragments and other foreign bodies following traumatic injuries.
13. Aid in localizing a hard-to-find apex during root-end surgery.
14. Confirm, following root-end surgery and before suturing, that all tooth fragments and excess filling material have been removed from the apical region and the surgical flap (Figure 9A-3).
15. Evaluate, in follow-up films, the outcome of treatment.

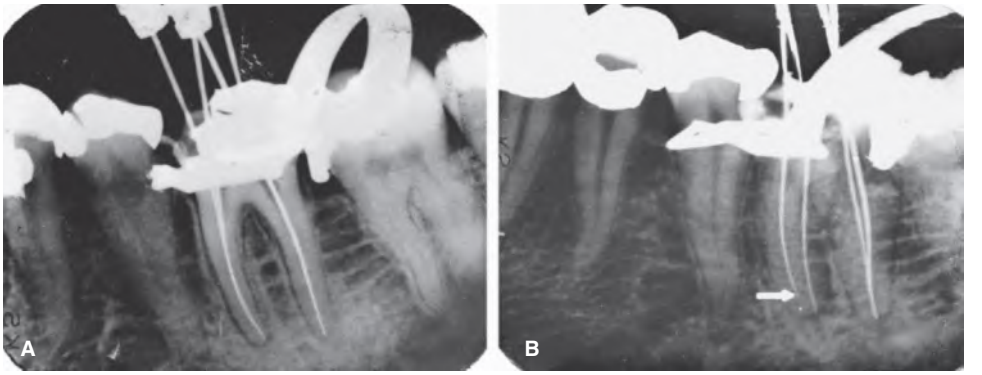

FIGURE 9A-1 Disclosing canals by radiography. ***A.*** Right angle horizontal projection reveals four superimposed files. ***B.*** Horizontal angulation varied 30° mesially reveals that there are three canals (two files in a broad distal canal). The file is short of working length in the mesiolingual canal (arrow).

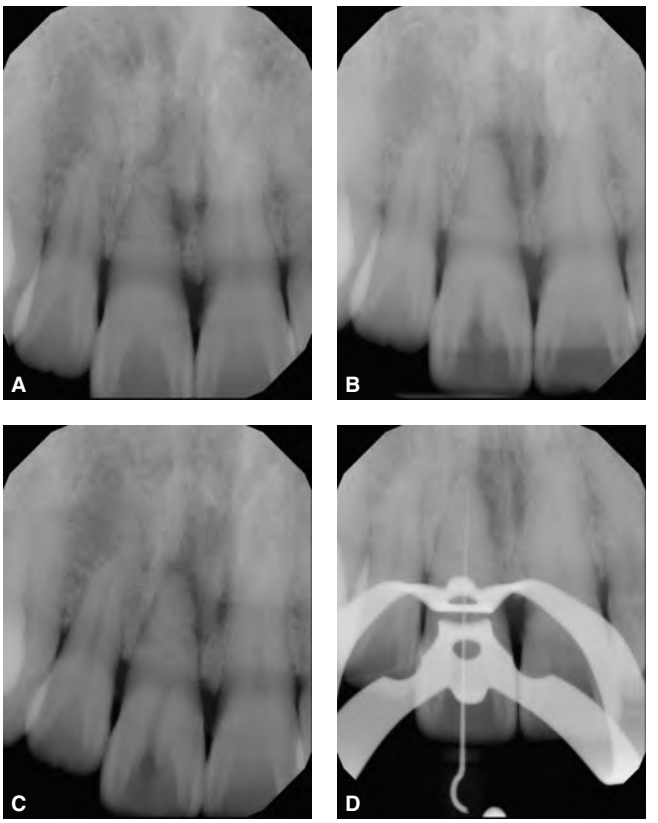

FIGURE 9A-2 Locating a canal. ***A.*** Calcific metamorphosis and apical pathosis. ***B.*** Access without rubber dam to aid in orientation. Preparation is slightly off-center to the distal. ***C.*** Access is redirected to the mesial. ***D.*** the canal is located with a fine file.

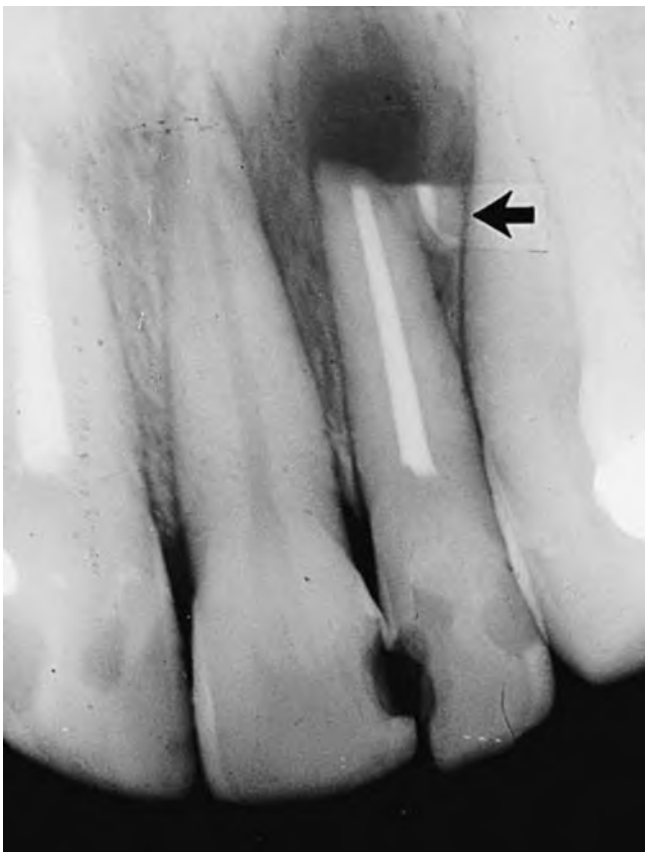

FIGURE 9A-3 Radiograph taken after suturing and developed after patient left the office. The patient had to return to the office for removal of the root tip.

Limitations of Radiographs

Radiographs could be termed the "Great Pretender." They are obviously essential, but are frequently overused and/or misinterpreted. A radiograph is but a two-dimensional shadow. They are suggestive only and are not the singular final evidence in judging a clinical problem. There must be correlation with other subjective and objective findings. The greatest fault with the radiograph relates to its physical state. As with any shadow, these dimensions are easily distorted through improper technique, anatomic limitations, or processing errors. In addition, the buccal–lingual dimension is absent on a single film and is frequently overlooked, although techniques are available to define the third dimension. These techniques are later described in detail.

Radiographs are not infallible. Various states of pulpal pathosis are indistinguishable in an X-ray shadow.

This factor must be considered in evaluating teeth that become symptomatic but show no radiographic changes; thus, the lack of radiolucency should not be interpreted as an absence of a bone resorbing process. Neither healthy nor necrotic pulps cast an unusual image. Correspondingly, the status of infection in hard or soft tissue is not detectable other than by inference. Only microbial culture can determine this. Furthermore, periapical soft tissue lesions cannot be accurately diagnosed by radiographs; they require histologic verification.[4] Chronic inflammatory tissue cannot, for example, be differentiated from healed, fibrous, "scar" tissue, nor can a differential diagnosis of periapical radiolucencies be solely made on the basis of size, shape, and density of the adjacent bone.[3–7] A common misconception is that an inflammatory lesion is present only when there is at least a perceptible "thickening" of the periodontal ligament space. In fact, investigators have demonstrated that lesions of the *medullary bone* often go undetected unless there is marked resorption or until the resorption has eroded a portion of the *cortical plate*.[8–11]

The fallibilities and inherent errors in radiographic interpretation were clearly demonstrated by Goldman et al.,[12] who submitted recall radiographs of endodontic treatments, for clinical evaluation, to a group of radiologists and endodontists. They assessed success and failure by observation of radiodensities. There was *more disagreement than agreement* among the examiners.

Importantly, the radiograph is only an adjunctive tool and can be misleading. Information obtained from proper interpretation of the radiograph is not always absolute and must be always integrated with information gathered from a detailed medical and dental history, clinical examination, and pulp testing procedures. For more details, see Chapter 10.

The techniques outlined in the following sections have proved to be successful and predictable. If followed, they will greatly simplify difficulties in root canal treatment.

TECHNOLOGY SYSTEMS

Alternative imaging techniques have been introduced over the years to overcome the existing limitations of analog intraoral radiographs. CBCT has proved to be particularly useful for diagnosis, treatment planning, and anatomic surveys.[13,14] CBCT, magnetic resonance imaging and ultrasound will be discussed later in this chapter.

There are basically two radiographic approaches. The traditional is the X-ray exposure of film that is chemically processed to produce an image. The newer digital systems rely on an electronic detection of an X-ray-generated image that is then electronically processed and reproduced on a computer screen. Overall, the resulting image is similar in interpretative quality to the traditional radiograph.[15,16] Advantages of digital radiography include reduced radiation, speed of obtaining the image, enhancement of the image, computer storage, transmissibility, and a system that does not require chemical processing.[17] Disadvantages are cost and more difficulty in placing the sensor. However, the advantages outweigh the disadvantages. As costs decrease, sensor design improves and technology progresses, use of the digital system has increased considerably and is becoming the standard.

Either digital or film type receptors can be used to capture the image when exposing an intraoral radiograph. Both digital and film type receptors are available in American National Standard Institute sizes 0, 1, 2, and 4. The receptor size is selected considering the specific area of interest. Usually, the size 1 receptor is chosen for evaluation of anterior teeth, while the size 2 receptor is used in the posterior region, specifically from the distal of the canine posteriorly. The size 4 receptor is used exclusively for the occlusal radiograph. The size 0 receptor is indicated for pediatric patients or in situations where anatomic considerations limit the ability of adequate placement of a larger receptor. For full discussion on digital radiography, see Chapter 9-B.

Traditional Machines

Two basic types of X-ray machines are commonly used in dental offices. One type has a range of kilovoltage and two milliamperage settings with which the long (16-in) cone is frequently used. The other type offers only 1 kV and milliamperage setting and only the short (8-in) cone. Either type provides adequate radiographs. However, each has advantages that, under different circumstances, will yield a better result. The long-cone system is superior for diagnostic radiographs, whereas the flexible short-cone machine is more appropriate for treatment or "working" films. However, either the "long cone" or the "short cone" is satisfactory if used properly.

The long-cone machine is preferred for exposing diagnostic, final, and follow-up radiographs, because of the clarity and minimum distortion inherent in the long-cone parallel technique.[18,19]

The short-cone machine with a small, easily manipulated head, saves time, energy, and frustration, and may therefore be preferred when taking working radiographs.

Short Cone and Long Cone

The positioning-indicating device is sometimes referred to as the cone. Whether using digital or film, the length of the cone does affect the size of the image. A longer cone decreases magnification and gives better detail. The shorter cone has greater divergence of the X-ray beam causing excessive interproximal overlapping.

Increasing the focal spot distance from the object using a long cone, decreases the magnification and fuzziness of an image. There is also less divergence of the X-ray beam. The short cone has a greater divergence of the X-ray beam causing excessive overlapping and magnification, if there is any distance between the tooth and the film (Figure 9A-4). The length of the cone does not affect the amount of scatter radiation because the diameter of both cones is the same.

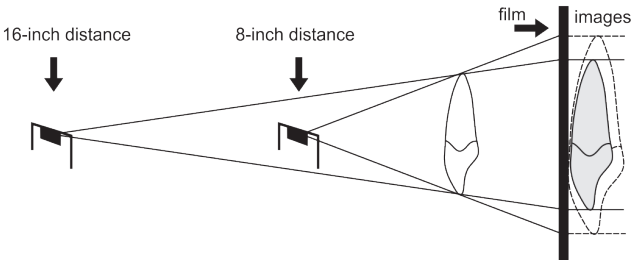

FIGURE 9A-4 Effect of changing the focal length. Increasing the focal spot distance from the object using a long cone decreases the magnification. It also decreases the fuzziness of the image. Another advantage is less divergence of the X-ray beam and therefore less magnification of the image.

Film Speed

Film speed is a term that refers to how efficiently the light-sensitive agents in a film emulsion react to energy (e.g., e-rays or light) exposure.

The American National Standards Institute and the International Organization for Standardization have established standards for film speed.[20] Film speeds available for dental radiography are D-speed, E-speed, and F-speed, with D-speed being the slowest and F-speed the fastest. The use of faster film speed can result in up to a 50% decrease in exposure to the patient without compromising diagnostic quality.[3,21] Film of a speed slower than E-speed should not be used for dental radiographs.[21,22]

The limited use of E or F-speed films compared with D-speed films is often due to misconceptions about price, clinical film quality, and processing. A comparison of one manufacturer's D-speed film to E-speed or F-speed film shows no significant clinical differences in quality between the films. Using E-speed or F-speed film for endodontic application provides a similar mean-correct diagnosis, comparable sensitometric properties (i.e., technical aspects of film imaging) and similar clarity of film compared to D-speed film. E-speed or F-speed film is processed without significant changes in processing practices to those that are used for D-speed film.[23,24] F-speed film is beginning to be more and more popular in dental offices.

The standard periapical-size film is used for most situations. In addition, every office should have 3 by 2 1/4-in *occlusal* film available for use when:

1. Periapical lesions are so extensive that they cannot be demonstrated in their entirety on one perirapical film;
2. there is interest in, or involvement of, the nasal cavity, sinuses, or roof or floor of the mouth;
3. trauma or inflammation prohibits normal jaw opening required to place and hold a periapical film;
4. a disabled person is unable to hold a periapical film by the usual means;
5. detection of fractures of the anterior portion of the maxilla or mandible is needed; and
6. very young children are being examined.

Intraoral Film Placement

Film placed parallel to the long axis of the teeth and exposed by cathode rays at a right angle to the surface of the film yields the most accurate images, with no foreshortening or elongation[25,26] (Figure 9A-5). If this principle is applied, it is unnecessary to memorize fixed cone angulations. Also, a patient need not be returned to an upright position for each exposure.

To achieve this parallel orientation it is often necessary to position the film away from the tooth, toward the middle of the oral cavity. This technique permits an accurate reproduction of the tooth's dimension and additionally reduces the possibility of superimposing the zygomatic process over the apices of maxillary molars that often occurs with distal-angulated films.

Because of the complicating presence of the dental dam, the methods for placement of *working* films differ somewhat from the methods for placement of diagnostic, final, and follow-up films.

Diagnostic Radiographs

These must be the best radiographs possible. The tooth should be centered on the film; this gives the least distortion. In addition, at least 3 mm of bone must be visible beyond the apex.

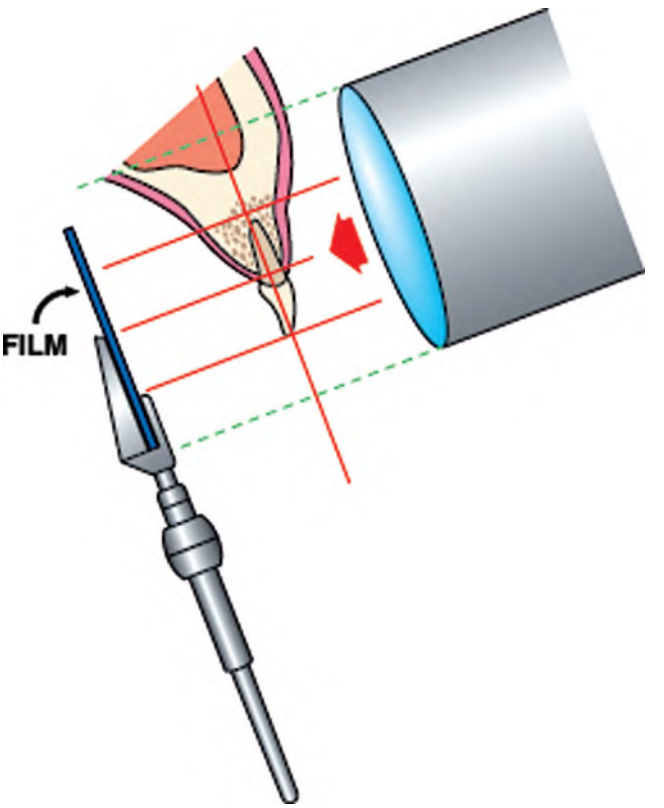

FIGURE 9A-5 Radiographic parallelism. The long axis of the film, the long axis of the tooth, and the leading edge of the cone are parallel and perpendicular to the X-ray central beam. (Reproduced with permission from Goerig AC. In: Besner E, Michanowicz A, Michanowicz J. editors. *Practical endodontics*. Mosby, St. Louis, MO, 1993; p. 56).

Failure to capture this area may result in misdiagnosis. There are advantages to parallelism; this permits more accurate visualization of structures as well as reproducibility. This facilitates comparison of follow-up radiographs.

The bitewing radiograph is often used as a supplement for diagnosis. It provides less image distortion because of its parallel placement. It is a single image depicting the maxillary and mandibular crowns, the interproximal contacts, the height of the bone crest, anatomy and size of the pulp chamber, presence of pulp stones, calcifications or resorptions, recurrent caries, and status of restorations. The bitewing is very helpful assessing restorability.

Bitewing radiographs are usually made with the size 2 receptor in a horizontal orientation. Horizontal bitewings may fail to reveal the alveolar crests when the crest has receded. Reorienting the receptor into a vertical position will then capture the alveolar crests. During treatment, a bitewing radiograph is useful to check the depth of access or the proximity of the furcation in cases with a receded pulp chamber. In this case, the dental dam should be removed temporarily to avoid overlapping of the clamp on the cervical area of the tooth. With large radiolucent lesions, an occlusal or a panoramic radiograph will assess the size and extent, as well as aiding in the planning of a proper treatment strategy.

There are a number of devices on the market that improve film placement and parallelism. The *Rinn XCP* (Dentsply/Rinn, Elgin, IL, USA) virtually guarantees distortion-free films but cannot be used with the dental dam in place. Special endodontic film holders are designed specifically to ensure parallelism yet avoid dental dam clamps while allowing space for files protruding from the tooth (Figure 9A-5). A disadvantage to this system is that the paralleling bar interferes with the cone when varying the horizontal cone angulations. Finger retention of the film should not be used, because the film is often bent and therefore, distorted. A *straight hemostat* is a good film holder and additionally serves as a cone-positioning device (Figure 9A-6).

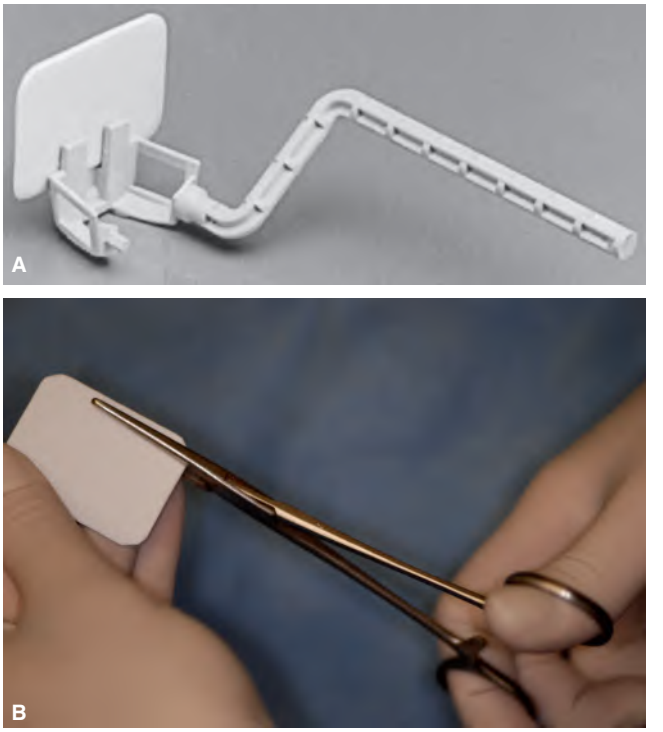

FIGURE 9A-6 Devices for holding "working" films. ***A.*** The Universal Rinn Endo Ray plastic film holder is designed for horizontal posterior films or anterior vertical films, maxilla or mandible, right or left. Here it is set for a maxillary posterior view. The "cupped out" area accommodates the tooth, clamp, and extruding endodontic files. (Photo courtesy of Rinn Corp., U.S.A.) ***B.*** A hemostat is used for grasping the film and as a cone positioning and orientation device.

Working Radiographs

One difficulty in root canal therapy is the clumsy, aggravating method of taking treatment radiographs with the dental dam in place. The dental dam frame should not be removed for access in film placement because doing so allows saliva entry to contaminate the operating field. A film-placement technique is used so that the *dental dam frame need not be removed*. Use of a radiolucent frame such as the *Star VisiFrame* (Dentaleze/Star, USA) will ensure that apices are not obscured.

With the dental dam in place, a *hemostat-held* film has the following significant advantages:

1. The film placement is easier when the opening is restricted by the denta dam and frame.
2. The patient may close somewhat with the film in place, a particular advantage in *mandibular posterior areas* where closing relaxes the mylohyoid muscle, permitting the film to be positioned farther apically.
3. The handle of the hemostat is a guide to align the cone in the proper vertical and horizontal angulation (Figure 9A-6).
4. There is less risk of distortion of the radiograph caused by too much finger pressure bending the film.
5. Patients can hold a hemostat handle more securely with less possibility of film displacement.
6. Any movement can be detected by the shift of the handles and corrected before exposure.

The *identifying dimple* is always placed at the incisal or occlusal edge to prevent its obscuring an important apical structure.

Cone Positioning

It is often a mistake to rely on only one film. Additional exposures taken from varied horizontal or vertical projections give better visualization of the third dimension (Figure 9A-7).

Vertical Angulation

Ordinarily, it is preferable to align the cone so the beam strikes the film at a right angle. This alignment ensures a fairly accurate vertical image. Elongation of an image, however, *may be corrected by increasing the vertical angle* of the

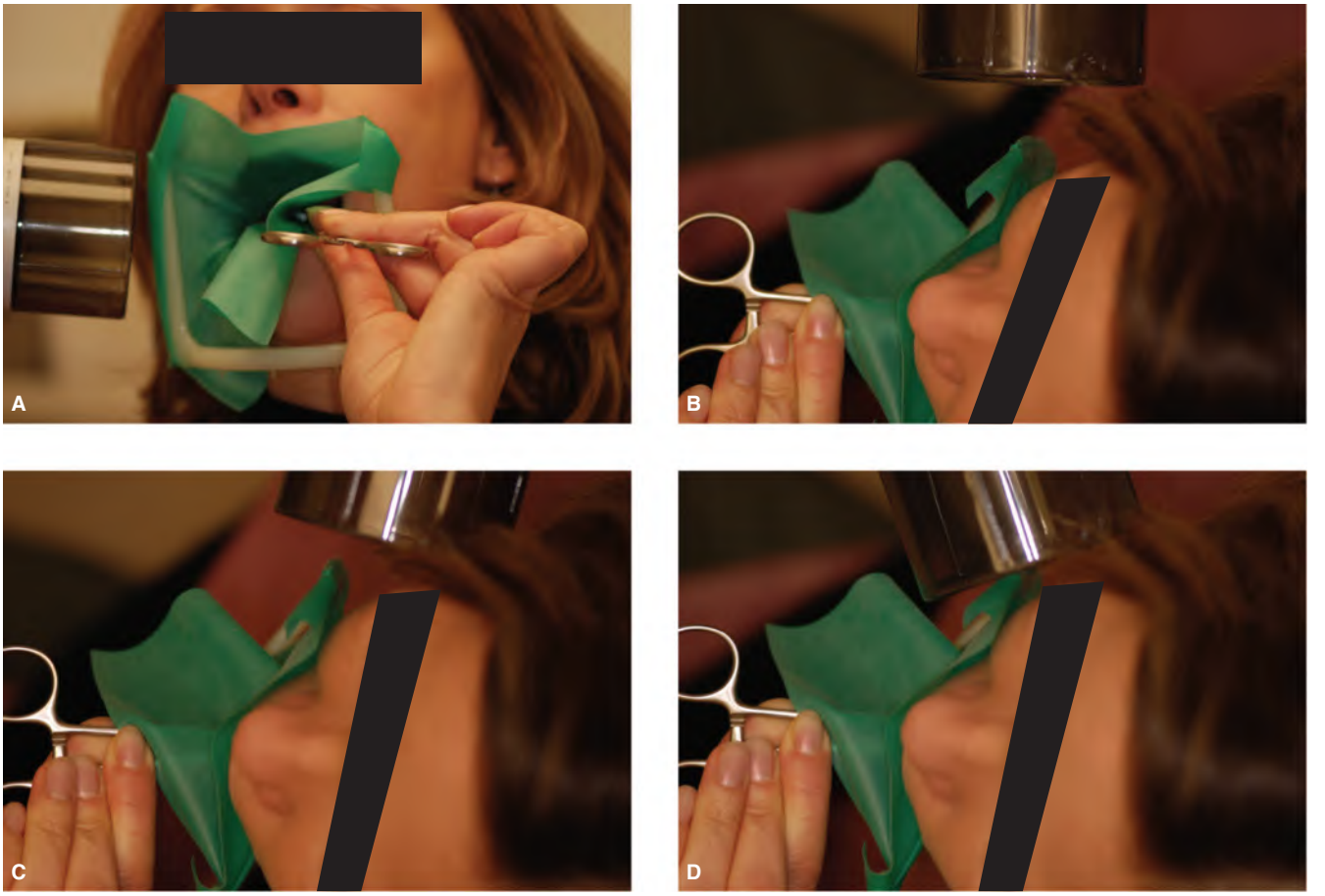

FIGURE 9A-7 "Working" film properly placed and held under dental dam with hemostat. ***A.*** Vertical angle. Cone is aligned so that central beam is parallel with the handle. ***B.*** Cone is aligned at right angle to handle and film. ***C.*** Varying horizontal angle to the distal 20–25°. Vertical angle is maintained. ***D.*** Cone is shifted 20–25° to the mesial.

central ray. Conversely, *foreshortening is corrected by decreasing* the vertical angle of the central ray.

Frequently, an impinging palatal vault prevents parallel alignment of the film and the teeth. However, if the film angle is no greater than 20° in relation to the long axis of the teeth, and the beam is directed at a right angle to the film, no distortion occurs, although there is a less effective orientation of structures.[26] The resulting radiograph is still adequate.

Vertical angulations are divided into positive (+) and negative (-) angles depending on the X-ray beam direction in reference to the horizontal plane. A positive (+) angle indicates the downward direction of the beam, while a negative angle (-) refers to an upward beam direction.

Horizontal Angulation

Walton[27] introduced an important refinement in dental radiography that has materially improved the endodontic interpretive film. He demonstrated a simple technique whereby the *third dimension* may be readily visualized. Specifically, the anatomy of superimposed structures, the roots and pulp canals, may be better defined. The horizontal plane of the buccal surfaces of the teeth determines the horizontal angulation.

The basic technique is to vary the *horizontal angulation* of the central ray of the X-ray beam. By this method, overlying canals may be separated, and by applying Clark's rule,[28] the separate canals may then be identified. Clark's rule states that "the most distant object from the cone (lingual/palatal) moves toward the direction of the cone." Stated in another way, using a helpful mnemonic, Clark's rule has been referred to as the Same Lingual, Opposite Buccal (SLOB) rule: the object that moves in the ***S***ame direction as the cone is located toward the Lingual. The object that moves in the Opposite direction from the cone is located toward the Buccal. The SLOB rule, simply stated, is "The lingual object follows the tube head." Goerig and Neaverth[29] applied the SLOB rule to determine, from a single film, from which direction a radiograph was taken: mesial, straight on, or distal. By knowing the direction, the lingual is differentiated from the buccal (Figure 9A-8). Stated more simply, Ingle's rule is *MBD*: If "shot" from the Mesial, the Buccal root will be to the Distal.

Horizontal Cone Angulations

For working and/or interpretive diagnostic radiographs, the following cone angles are preferred:

1. Maxillary anterior teeth (straight facial).
2. Maxillary premolars and molars (mesial angle).

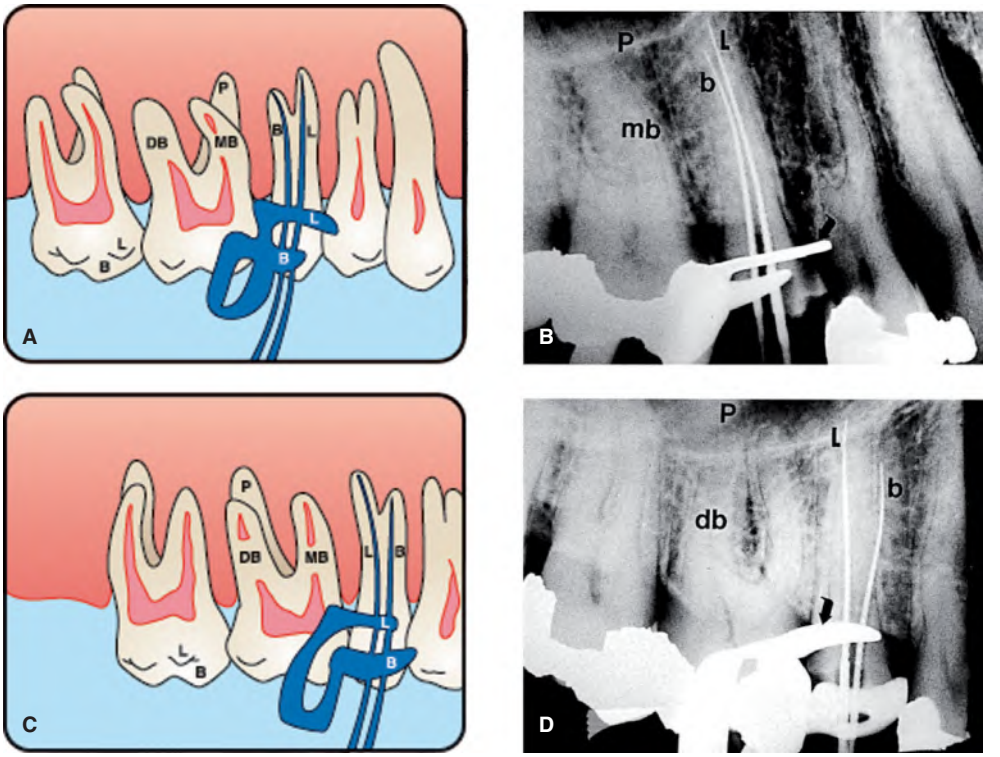

FIGURE 9A-8 Applying the Same Lingual, Opposite Buccal (SLOB) rule to determine the cone angle. ***A & B.*** Clues that the cone is oriented from the mesial. The mesial–buccal (MB) root lies over the palatal (P) root, that is, the lingual (palatal) root has moved mesially; the lingual arm of the dental dam clamp (arrow) has moved mesially. The canine is visible. Once it has been determined that the radiograph was taken from the mesial, the lingual root (toward the mesial) of the premolar is defined. ***C & D.*** Radiograph of the same teeth taken from the distal. Clues are reversed. The canine is not visible. The lingual premolar canal is now toward the distal. (Reproduced with permission from Goerig AC. In: Besner E, Michanowicz A, Michanowicz J. editors. *Practical Endodontics*. Mosby, St. Louis, MO, 1993; p. 54).

3. Mandibular incisor teeth (distal angle).
4. Mandibular canines (mesial angle).
5. Mandibular premolars (mesial).
6. Mandibular molars (distal).

A helpful mnemonic for the working mandibular angulations: MDDM.

All teeth require specific film placement and cone positioning for working films (Figure 9A-9).

Mandibular Molars

As previously emphasized, the film must be positioned parallel to the lower arch. The *standard* horizontal X-ray projection then is at a right angle to the film (perpendicular), as shown in Figure 9A-10. The two mesial canals are superimposed one upon the other and appear as a single line.

Through the *Walton projection (SLOB)*, however, the roots will "open up." This is done by directing the central beam 20° to 30° from either the distal or the mesial (Figure 9A-11A). In Figure 9A-11B (black arrows), the two canals in each root can now be readily discerned. The contrast gained by varying the horizontal projection is seen in a clinical case with four canals (Figure 9A-1). Figure 9A-1A taken at a right angle clearly shows the four instruments superimposed on one another. Figure 9A-1B, on the other hand, made from a 30° variance in horizontal projection, emphasizes the third dimension: the separation of the instruments in the canals. By applying Ingle's rule (MBD: project the central beam from the *mesial*), the *buccal* canals are toward the *distal*, the *lingual* canals toward the *mesial*.

Another point concerns a frequent mistake in "reading" periapical radiographs. It can best be illustrated by cross-sectional drawings of molar root structure (Figure 9A-12). Roots containing two canals are often hourglass shaped. When an X-ray beam passes directly through this structure (center image), the buccal and lingual portions of the root are in the same path (arrows). These will change with distal and mesial projection. Because a double thickness of tooth structure is penetrated by the X-rays, it is seen in the film as a radiopaque root outline in close contact with the *lamina dura*. This is readily apparent on the radiograph (Figure 9A-13).

By aiming the cone 20° from the mesial, however, the central beam passes through the hourglass-shaped root at an angle (Figure 9A-14). In this case, the two thicknesses of the root are projected separately onto the film. Because less tooth structure is penetrated by the X-ray, the image on the film is less dense. A radiolucent line is clearly seen (open arrow). This radiolucent line can be erroneously interpreted as a canal. By following up the length of the line, instead of

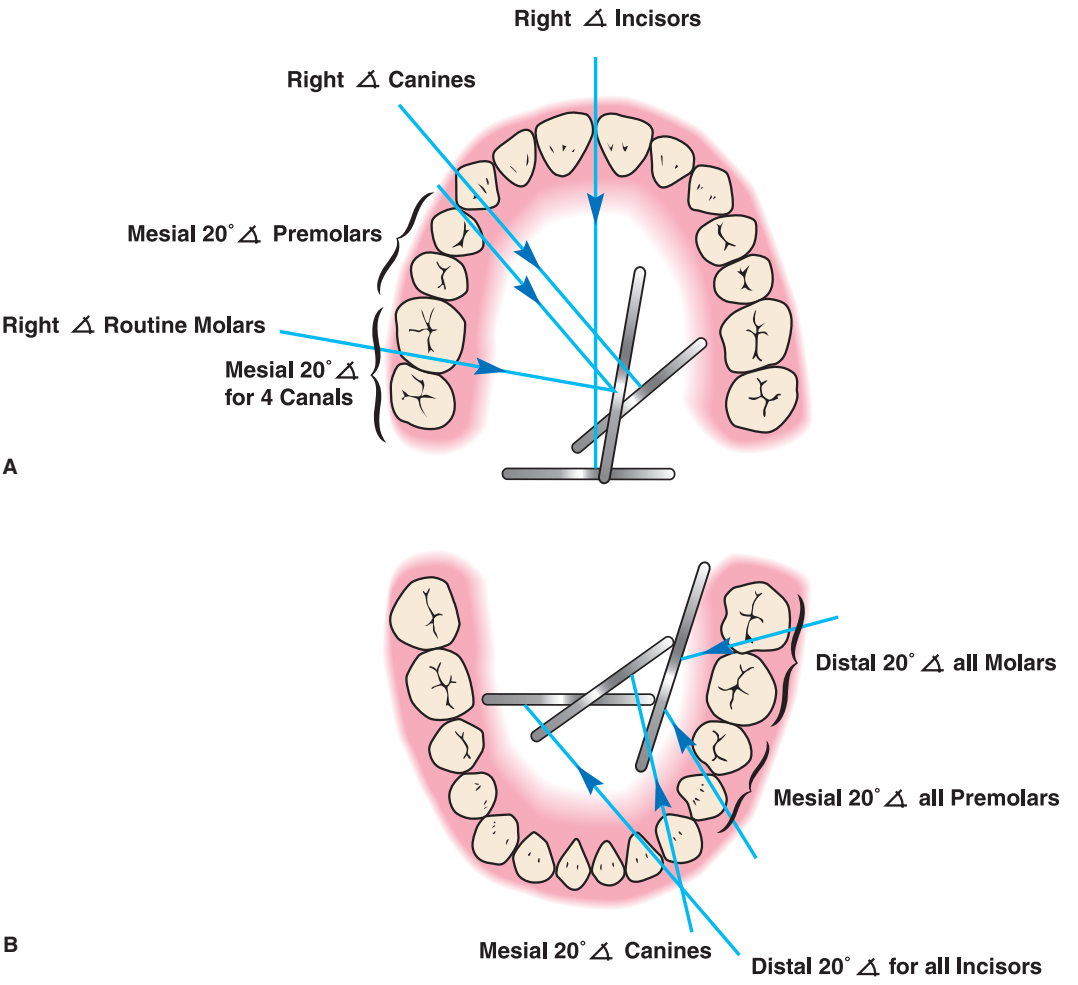

FIGURE 9A-9 ***A.*** Maxillary film/central beam (arrows) position. ***B.*** Mandibular film/central beam position (arrows). (Reproduced from Torabinejad M, Walton R. *Endodontics: Principles and Practice*, 5th ed. Philadelphia, PA: Saunders and Co., 2015, pp. 214–215).

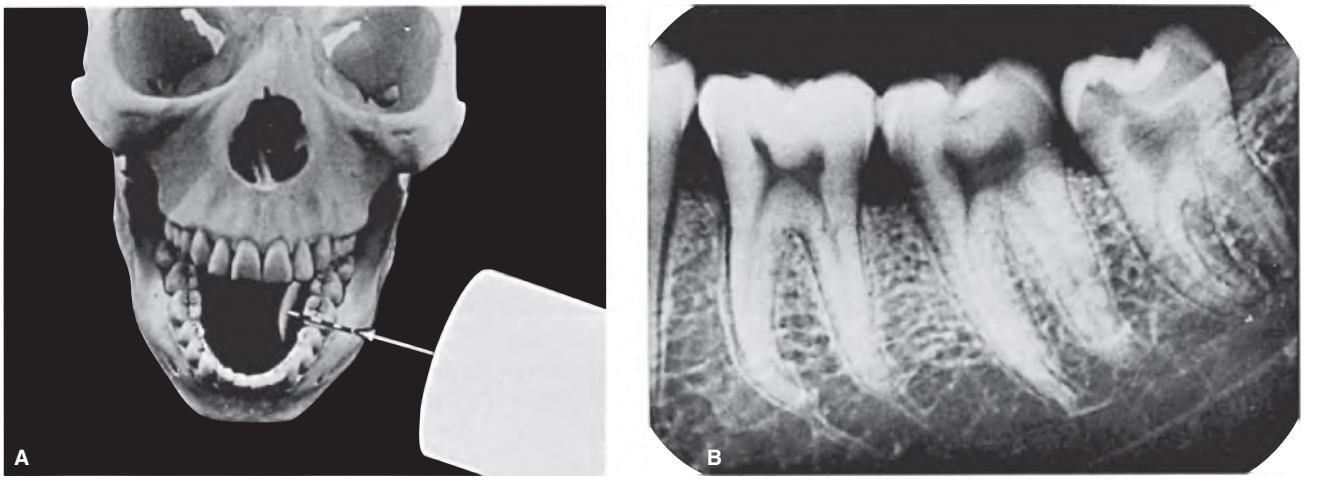

FIGURE 9A-10 Mandibular molars. ***A.*** Central ray directed at right angle to film positioned parallel to arch. ***B.*** Limited information is gleaned from the radiograph because of superimposition of structures and canals. (Reproduced with permission from Walton RE.[27])

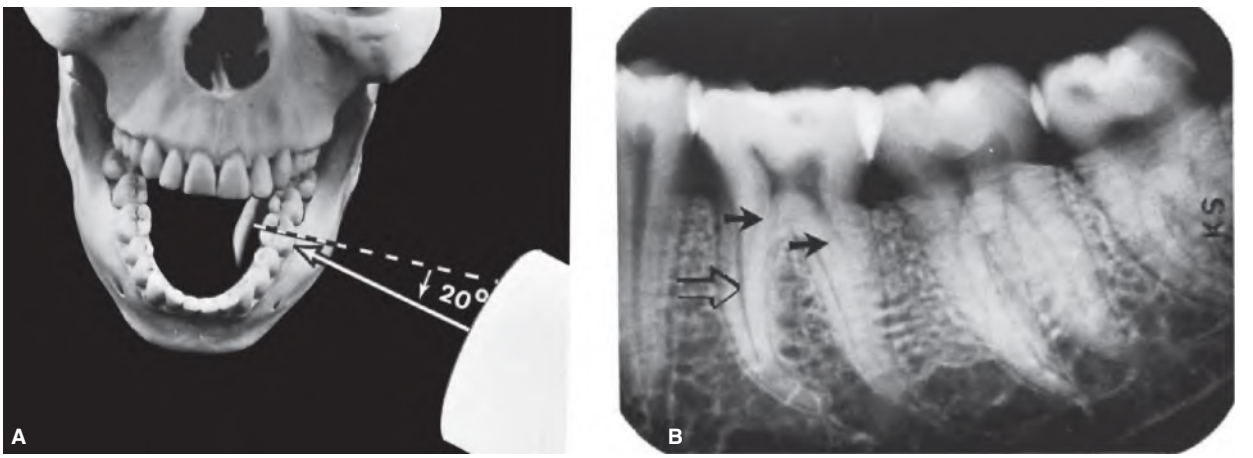

FIGURE 9A-11 Mandibular molars. ***A.*** Central ray directed at 20° mesially to film positioned parallel to arch. ***B.*** Two canals are now visible in both roots of the first molar (black arrows). Open arrow indicates confusing root outlines. (Reproduced with permission from Walton RE.[27])

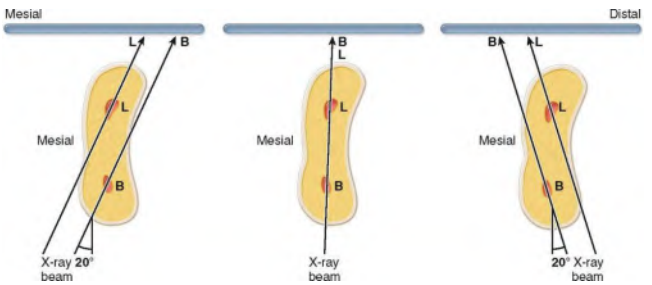

FIGURE 9A-12 Images vary and "move" when the horizontal cone changes position. Center: X-ray beam passing directly (straight-on) through a root containing two canals. Mesial and distal shifts: the lingual object moves in the same direction as the cone; the buccal object moves in the opposite direction (SLOB rule). (Courtesy of Dr. Ace. Goerig, Olympia, WA, U.S.A.) (Reproduced with permission from Endodontics: Principles and Practice 5th ed. Eds: Torabinejad M, Walton R, Fouad A. Elsevier, St Louis, 2015; p. 215).

entering the pulp chamber, the line can be traced to the gingival surface of the root; this is the PDL space. This simple interpretive error can easily lead to gross mistakes.

Mandibular Premolars

The importance of varying the horizontal angulation when radiographing mandibular premolars is demonstrated in Figure 9A-15A. The central beam is directed at a right angle to the film. What appears to be a single straight canal is discernible in each premolar (Figure 9A-15B). There is an indication, however, in the image of the first premolar that the canal might bifurcate at the point of the abrupt change ("fast break") in density (arrow).

Directing the central ray 20° from the mesial in the *first premolar* (Figure 9A-16A) causes the bifurcation to separate into two canals (Figure 9A-16B). The tapering outline of the tooth, seen in both projections, would indicate, on the other hand, that the two canals undoubtedly rejoin to form a common

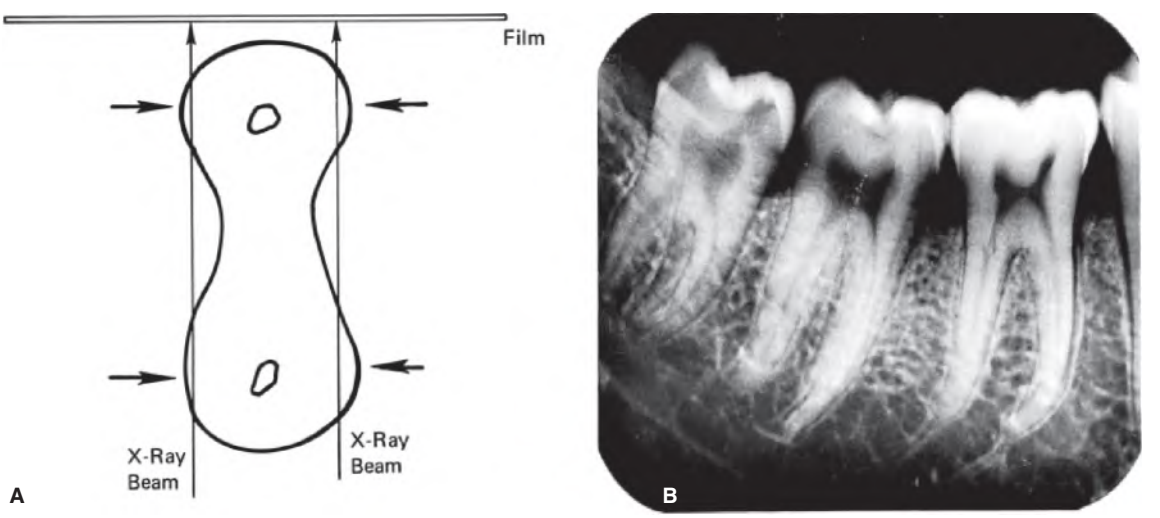

FIGURE 9A-13 X-ray beam passing directly through two thicknesses of root structure presents intensified image on film. Note radiopaque root outline inside lamina dura. (Reproduced with permission from Walton RE.[27])

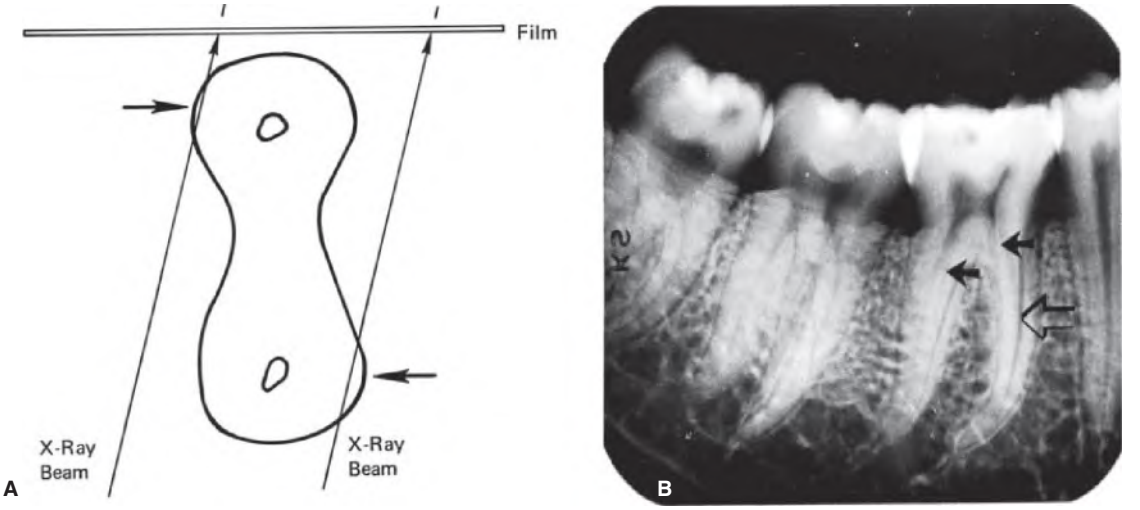

FIGURE 9A-14 X-ray beam aimed 20° passes through single thicknesses of hourglass root, leaving less dense impression on film. Radiolucent line is apparent (open arrow) and may be confused with root canal. Note that it emerges at gingival, not into pulp chamber. Black arrows indicate regular canals. (Reproduced with permission of Walton RE.[27])

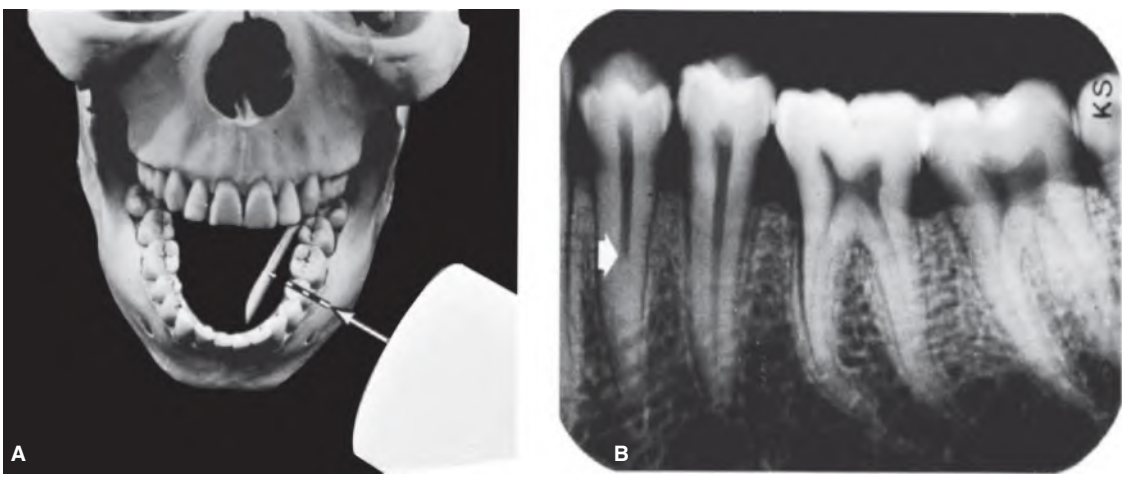

FIGURE 9A-15 Mandibular premolars. ***A.*** Central ray directed at right angle to film positioned parallel to arch. ***B.*** Radiograph reveals one canal in each premolar, although abrupt change in density (*arrow*) may indicate bifurcation. (Reproduced with permission from Walton RE.[27])

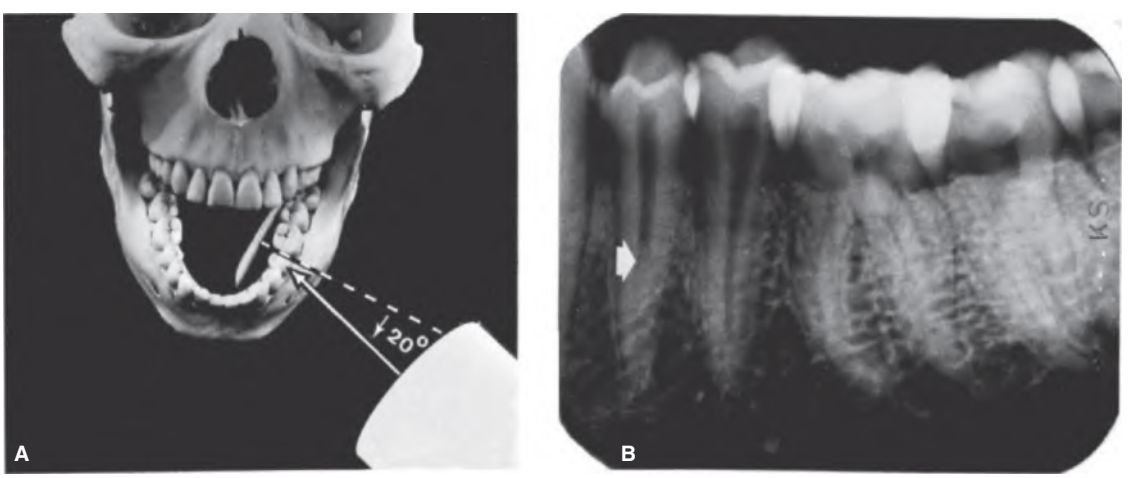

FIGURE 9A-16 Mandibular premolars. ***A.*** Central ray directed at 20° mesially to film, parallel to arch. ***B.*** In first premolar, two canals that are clearly visible (*arrow*) probably reunite, as indicated by sharply tapered root. (Reproduced with permission from Walton RE.[27])

canal at the apex. In both the right-angle and 20° variance projections, the *second premolar* appears as a single canal.

Maxillary Molars

Maxillary molars are consistently the most difficult to radiograph because of (1) their more complicated root and pulp anatomy, (2) the frequent superimposition of portions of the roots on each other, (3) the superimposition of bony structures (sinus floor, zygomatic process) on root structures, and (4) the shape and depth of the palate. Each of these, singly or in combination, can be a major impediment.

As is true of the mandible, the complex root anatomy and superimpositions may be dealt with *by varying the horizontal* angulations. Film placement must be parallel to the median raphe, not to the posterior maxillary arch.

The standard right-angle projection for a maxillary first molar, illustrated in Figure 9A-17A, produces the image seen in Figure 9A-17B, wherein the zygomatic process is superimposed on the apex of the palatal root (arrow) and the distobuccal root appears to overlie the palatal root. The sinus floor is also superimposed on the apices of both the first and second molars. When the horizontal angulation is varied by 20° to the mesial (Figure 9A-18A), the zygomatic process is "moved" to the distal of the first molar and the distobuccal root is cleared of the palatal root (Figure 9A-18B, arrows). The mesial projection also better "opens" the mesiobuccal root to demonstrate both canals.

The *opposite projection* can also be used to isolate the mesiobuccal root of the first molar, that is, the central ray may be projected from *20° distal* to the right angle (Figure 9A-19A). Although this projection distorts the shape of the mesiobuccal root, it also isolates it (Figure 9A-19B), so that the canal is readily discernible (arrow). In addition, the zygomatic process is moved completely away from any root structure, including the second molar. The same technique illustrated here for the maxillary first molar can be applied to

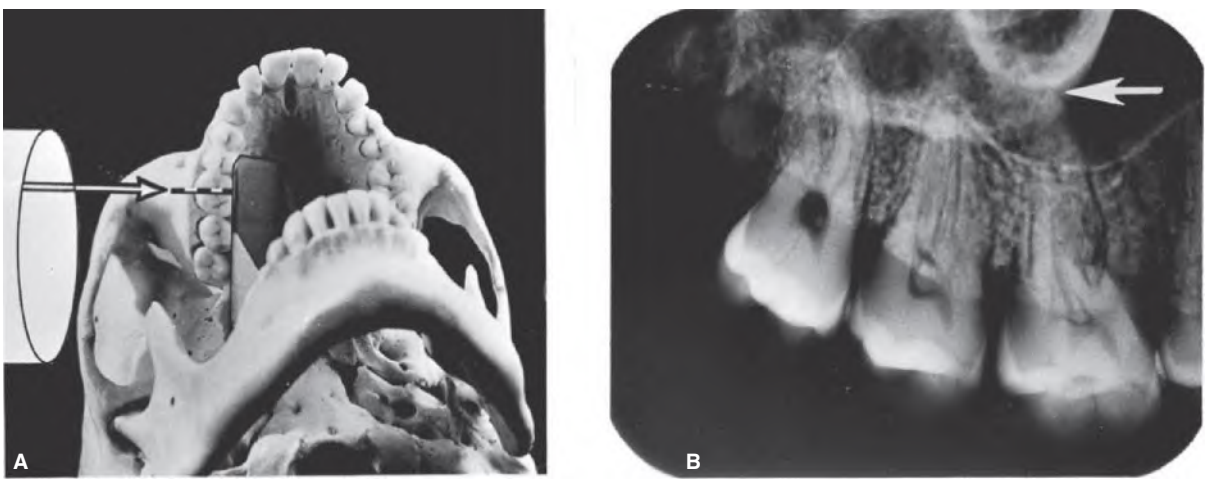

FIGURE 9A-17 Maxillary molars. ***A.*** Central ray is directed through maxillary molar at right angle to inferior border of film. Arrow and dotted line passing through malar process indicate it will superimpose over first molar. ***B.*** Superimposition of first molar roots, sinus floor, and malar process (white arrow) confuse the diagnosis. (Reproduced with permission from Walton RE.[27])

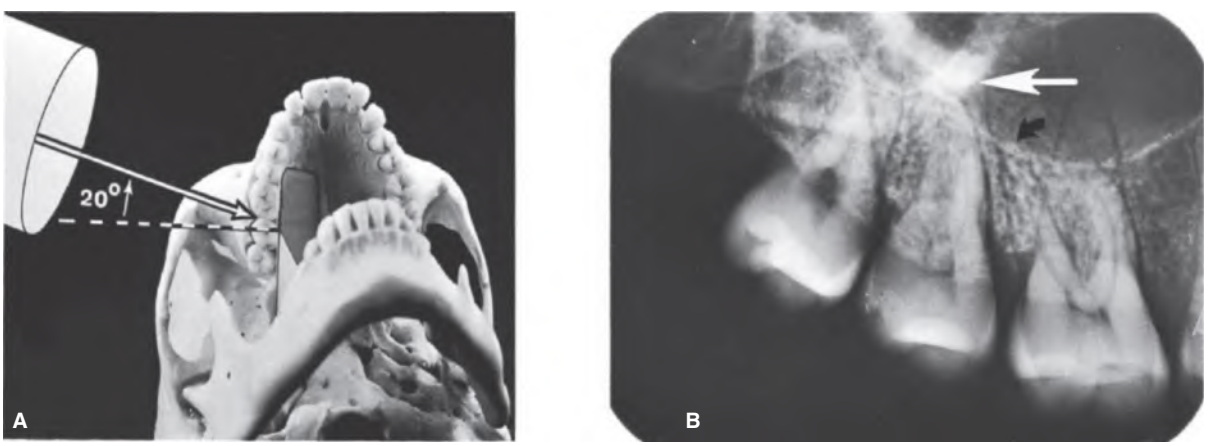

FIGURE 9A-18 Maxillary molars. ***A.*** Central ray directed at 20° mesially skirts malar process, projecting it distally. ***B.*** Distobuccal root is cleared of palatal root and malar process is projected far distally (white arrow). Between right angle and 20° projection, all three roots are clearly seen. (Reproduced with permission from Walton RE.[27])

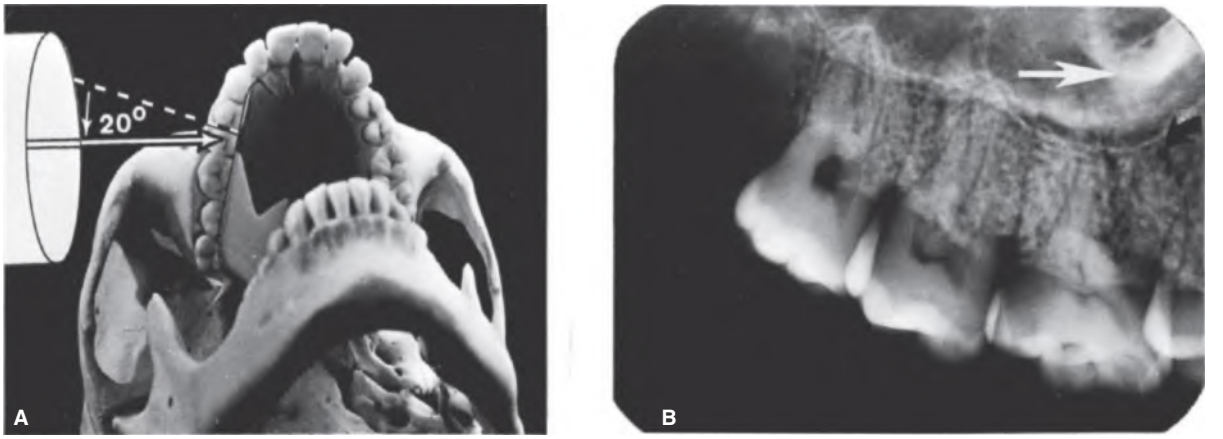

FIGURE 9A-19 Maxillary molars. ***A.*** Central beam projected 20° from the distal. ***B.*** Mesiobuccal root of the first molar is isolated (*black arrow*) and second and third molars are cleared of malar process, that is projected forward (*white arrow*). Sinus floor may be "lowered" or "raised" by changing vertical angulation. (Reproduced with permission from Walton RE.[27])

the second or third molars by directing the central beam at a horizontal variance through those teeth.

Maxillary Premolars

Variance in the horizontal projection has great value in maxillary premolar radiography, particularly for the *first* premolar, that generally has two roots and canals, but sometimes three. The clinical efficacy of the Walton (SLOB) technique is well illustrated in Figure 9A-20. The right-angle horizontal projection produces the single canal image seen in Figure 9A-20A. By varying the angulation by 20°, however, the two canals are separated (Figure 9A-20B), giving an unobstructed view of the obturation quality in both canals.

Mandibular Anterior Teeth

Aberrations in canal anatomy in the *mandibular* anterior teeth are not uncommon. Variance of the horizontal X-ray projections in this region will bring out the differences. Figure 9A-21A illustrates the standard X-ray projection bisecting the film held parallel to the arch. The incisor teeth appear to have single canals. However, a deep single canal is seen in the distorted canine image (Figure 9A-21B).

By varying the film placement and projecting directly through the canine, as seen in Figure 9A-22A (about 30° variance for the incisors), separate canals appear in the incisors (Figure 9A-22B, arrow); the canals join at the apex. This would be expected. However, when viewing the tapered incisor roots seen in both horizontal projections, the roots are far too narrow to support two separate canals and foramina. Once again, the abrupt change in canal radiodensity in the premolars (arrow) indicates a canal bifurcation.

Maxillary Anterior Teeth

Although canal or root aberrations appear less frequently in the maxillary anterior teeth, root curvature in the maxillary lateral incisors is a particularly vexing problem. Grady and Clausen[30] have shown, for example, how difficult it is to determine when foramina exit to the labial or lingual. Their radiographs of extracted teeth matched with photographs of instrument perforation short of the apex are a warning to all (Figure 9A-23).

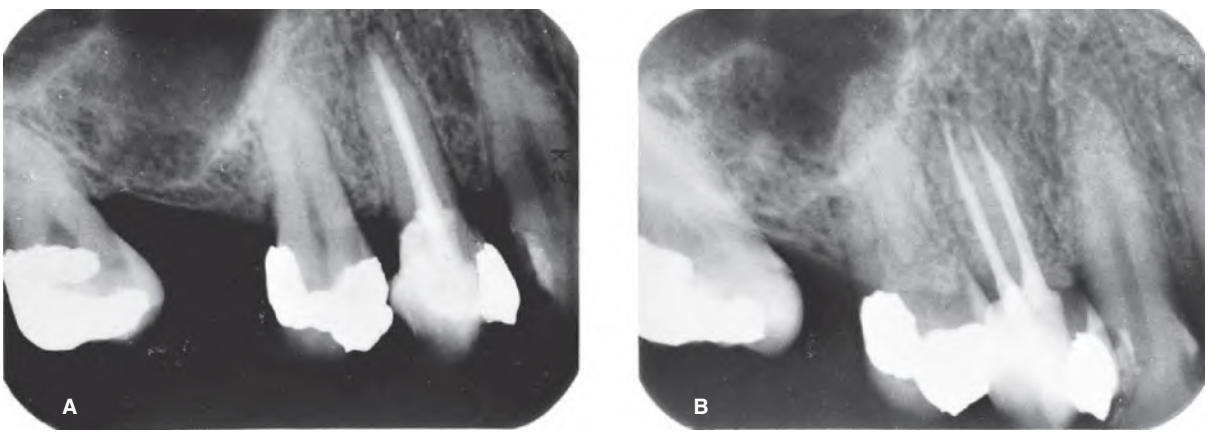

FIGURE 9A-20 Maxillary premolars. ***A.*** Horizontal right-angled projection produces illusion that that maxillary first premolar has only one canal. ***B.*** Varying horizontal projection by 20° from the mesial separates two canals. Buccal canal is distal (MBD). (Reproduced with permission from Walton RE.[27])

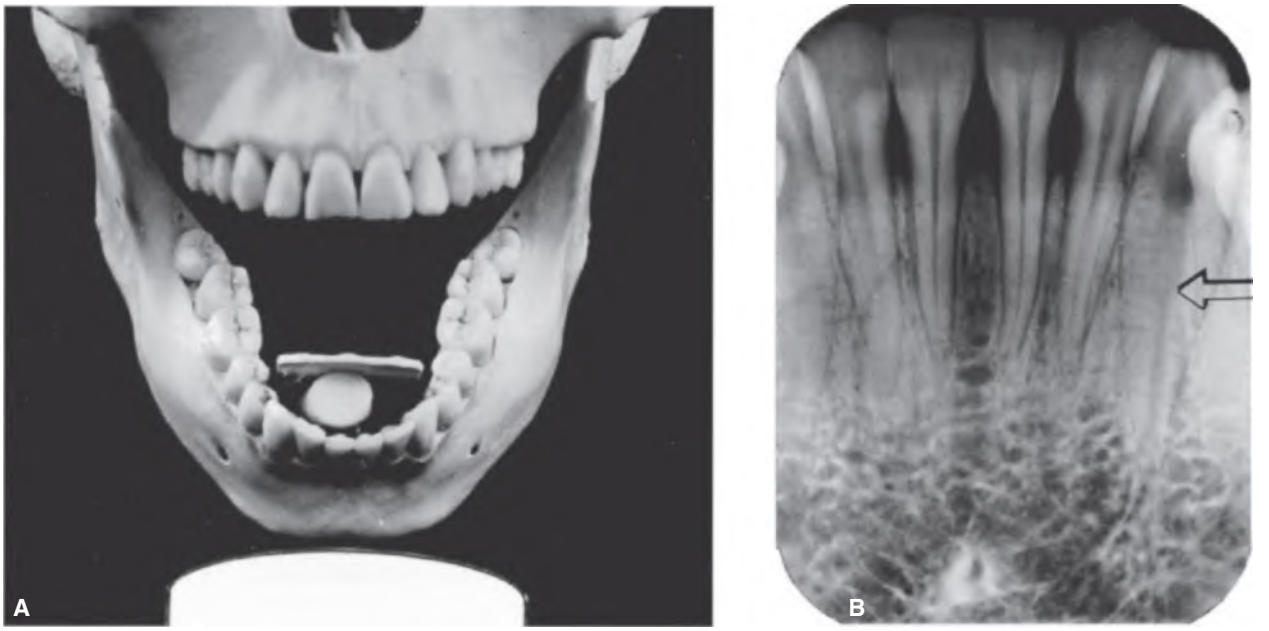

FIGURE 9A-21 Mandibular anterior teeth. ***A.*** Film placement for modified parallel technique. Horizontal central beam projection at right angle to film. ***B.*** Single canals seen in central incisors with only suggestion of two canals in lateral incisor. In distorted image of canine, note deep labiolingual canal dimension (arrow). (Reproduced with permission from Walton RE.[27])

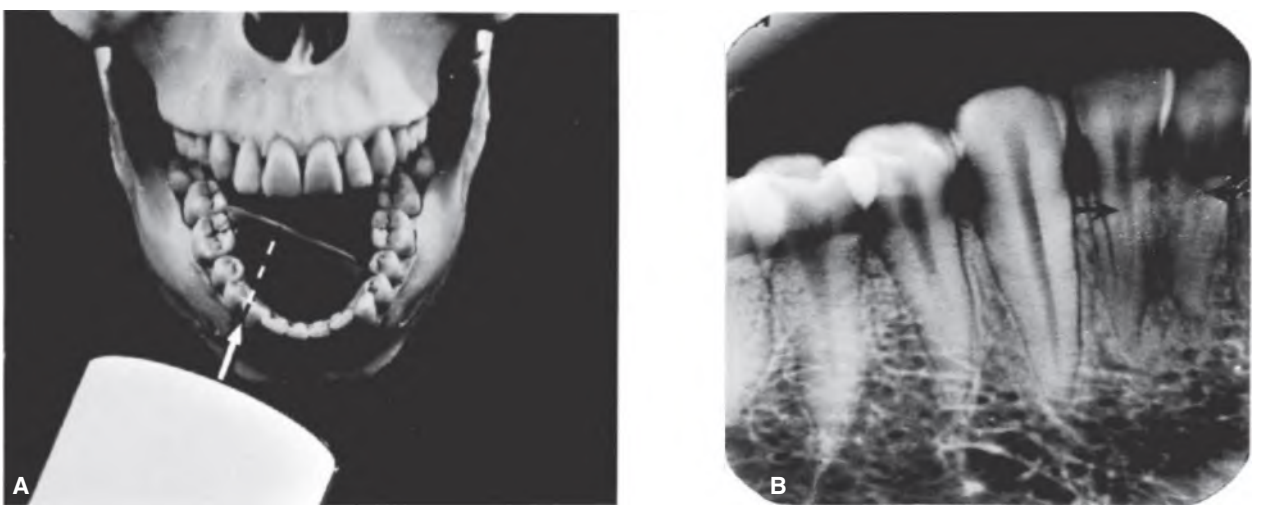

FIGURE 9A-22 Mandibular canine. ***A.*** Film is positioned for canine radiograph. Horizontally, central beam is projected at right angle to the film. ***B.*** Canine image is single straight canal, but incisor image reveals bifurcated canals that reunite in narrow tapered root (arrows). Note "bonus" image of bifurcated canals on first premolar. (Reproduced with permission from Walton RE.[27])

Extraoral Film Placement

This is useful for patients who cannot accommodate or tolerate intraoral film placement, usually because of gagging or trismus. Acceptable diagnostic and working films are possible.[31] This requires special positioning of cone and film (Figure 9A-24).

Processing

Another deterrent has been the length of time required to process films. Old, weakened solutions greatly increase the time required for processing. Moreover, adherence to the manufacturer's recommended temperature and time (68°F for five to seven minutes) for developing and clearing has hampered "on-the-spot" processing and viewing.

Ingle et al.[32] demonstrated, in a well-controlled blind study, the effects of varied processing temperatures. A processing temperature of 92°F yielded, in less than one minute, the most acceptable radiographs. At 92°F, using Kodak developer and fixer mixed to company specifications, *development required only 30 seconds* and fixation required 25 to 35 seconds, with no loss of quality. By comparison, at 70°F temperature, it required 5 minutes' developing time and 10 minutes' fixing time for Ultraspeed film. Ektaspeed is slightly better: 72 to 80°F for 2 1/2 to 4 minutes' developing and 2 to 4 minutes' fixing time. All films need to be *final washed* for at least 30 minutes.

For rapid processing, small quart-size tanks are adequate and economical. Frequent change in solutions is recommended.

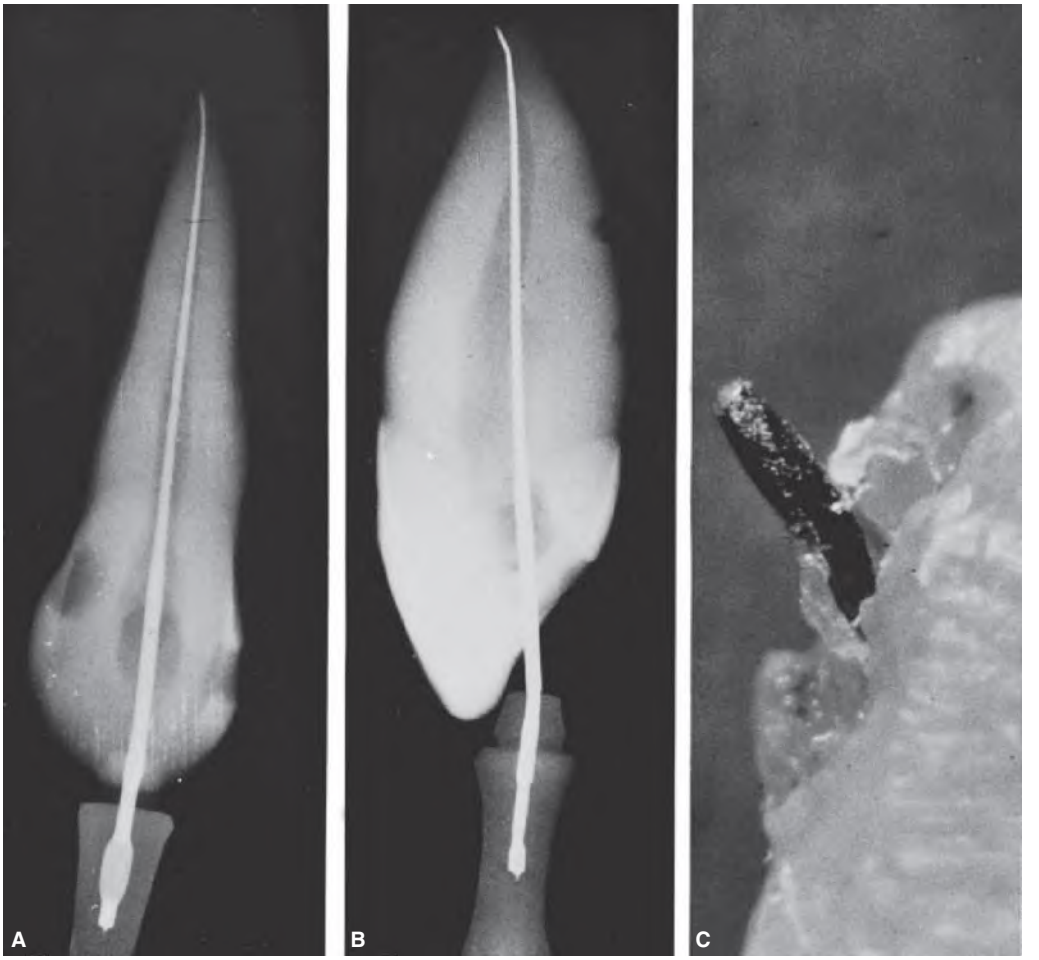

FIGURE 9A-23 ***A.*** Labiolingual projection through canine shows instrument apparently at apex with slight distal curvature. ***B.*** Mesiodistal projection reveals instrument actually emerging from the labial, short of apex. ***C.*** Instrument perforating foramen to labial well short of radiographic root end. (Photos courtesy of Drs. John R. Grady (Cleveland, OH, U.S.A) and Howard Clausen (North Dartmouth, MA, U.S.A).)

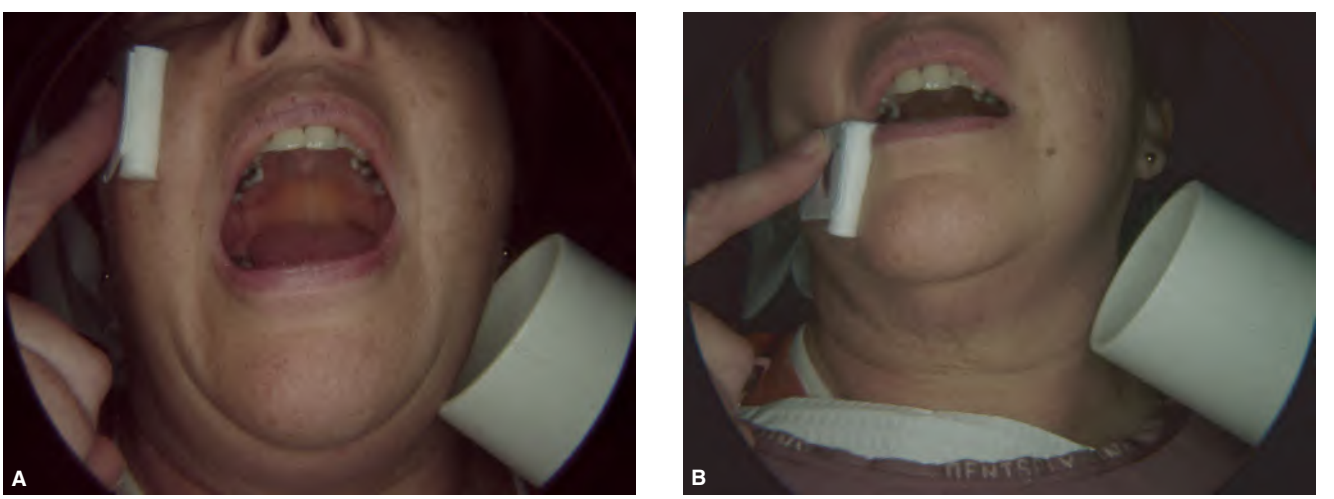

FIGURE 9A-24 Extraoral film placement. Useful for patients who cannot accommodate or tolerate an intraoral film. The film or sensor is placed on the cheek. Increasing the exposure time is necessary—approximately double. ***A.*** Maxillary posterior: The cone is positioned a negative 45° to the occlusal plane. ***B.*** Mandibular posterior: The cone is positioned a negative 35°.

Rapid-Processing Solutions

Concentrated chemicals, such as *Kodak's Rapid Access* developing and fixing solutions, have become very popular in endodontic practice. Although more expensive, they save measurable time, requiring only 15 seconds' developing and 15 seconds' clearing time in the fixer at room temperature.[33] These rapidly processed films will fade or discolor with time.[34] This change can be prevented, after viewing, by returning the wet film for a few minutes of fixation, followed by washing for 30 minutes, and then drying. The films will then retain their quality indefinitely.

TABLE-TOP DEVELOPING

For quick turnaround and ease of processing, combining rapid-speed solutions with a *table-top processing hood* greatly improves radiographic reporting, particularly working films. These hoods are often used right in the operatory. The film is passed through light-proof cuffs; hand movement is seen through the red Plexiglas cover. The rapid solutions and rinse water can be in small cups.

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Chapter 9B

Digital Radiography

Allan G. Farman, Martin D. Levin, Ramya Ramamurthy, Lars G. Hollender

Many acronyms have come about as a result of the development of technology in radiography and the need for terms to describe concepts. The following glossary of the acronyms used in this chapter will assist the reader in understanding these new concepts.

GLOSSARY OF ACRONYMS

AEC: Automatic Exposure Compensation is an enhancement feature that compensates for image variations due to exposure.

ALARA: Acronym for "As Low As Reasonably Achievable," an important principle in radiation protection.

CBCT: Cone-Beam Computed Tomography is a 3-D imaging device that integrates a series of transmission images generally taken over an arc of 180° or greater.

CBVCT: Cone-Beam Volumetric Computed Tomography (synonym of CBCT).

CCD: A Charge-Coupled Device is an image sensor consisting of an integrated circuit containing an array of linked or coupled, energy-sensitive capacitors.

CDR: Computed Dental Radiography. Digital intraoral sensor manufactured by Schick Technologies (A Division of Sirona AG).

CMOS: Complementary Metal–Oxide–Semiconductor. This represents a major class of integrated circuits including image sensors.

CMOS-APS: CMOS Active Pixel System.

DICOM: Digital Imaging and Communication in Medicine. The internationally referenced standard for improving the likelihood of interoperability of diagnostic images by setting file format standards and transmission protocols.

dpi: Dots per inch.

H&D: Hurter and Driffield. Descriptor applied to a curve relating optical density to exposure for film photography and radiography.

IP: Imaging Plate. The sensor plate used for photostimulable phosphor (PSP) imaging.

ISO: International Standards Organization.

MB2: For maxillary molar, mesiobuccal root second canal.

PSP: PhotoStimulable Phosphor. An imaging system using IPs that store energy from transmitted X-rays. The PSP IP is processed using laser scanning.

RVG: RadioVisioGraphy. Radiographic system proprietary to Kodak Dental Imaging: Term applied to the first commercial dental digital detector.

SNR: Signal-to-Noise Ratio.

TACT: Tuned Aperture Computed Tomography. A system developed by Richard Webber that integrates a series of transmission images taken from varying beam geometry to tomosynthesize a limited 3D perception.

Radiography is an important adjunct to endodontic diagnosis and image-guided treatment. The use of digital technologies can facilitate the process by speeding image acquisition and display. The invention of digital intraoral radiography has "endodontic roots." In the 1970s, Mouyen, then a dental student at the University of Toulouse, France, tells the story that because the distance between the endodontic clinic and the film processors several floors below spurred the idea of finding a way to create instant X-ray images for "operative" purposes. Mouyen realized that the chip in his video camera could detect light and that X-rays could be converted to light by scintillators such as the screens used in extraoral radiography cassettes. In other words, almost 90 years after the discovery of the X-ray, available technology could indeed permit the placement of a "fluoroscope" in the patient's mouth.

A few years following graduation as a dentist, Mouyen completed the DERSO, a French equivalent to the PhD in engineering, and went on to develop a prototype digital radiographic system that he named RadioVisioGraphy (RVG).[1] The RVG patent was licensed by a French Company, Trophy Radiologie, and digital imaging for operative radiology in dentistry was born (Figure 9B-1). In 1989, in a special issue of the "National Geographic" magazine, celebrating the bicentennial of the French Revolution, the RVG was included among 10 major French inventions, alongside such inventions as the Train Grand Vitesse (TVG), the Concorde, and the Arienne space rocket. Trophy Radiologie was acquired by Kodak Dental Imaging, and Kodak Dental Imaging was subsequently acquired by Carestream Dental.

Since 1989, there have been eight new generations of RVG, and there are now many alternate solid-state systems (Figure 9B-2) that can produce almost instant images electronically. Although these images are essentially "instant," most are not "direct" as the solid-state systems, for the most part, employ a scintillator to convert X-rays to light, and it is the analog light signal that is then converted to a digital image.

Furthermore, it is possible to replace silver halide film by photostimulable phosphor (PSP) plates that interact directly with X-rays but require the added step of subsequent processing by laser scanning to convert the latent image to one that can be viewed on a computer monitor screen.

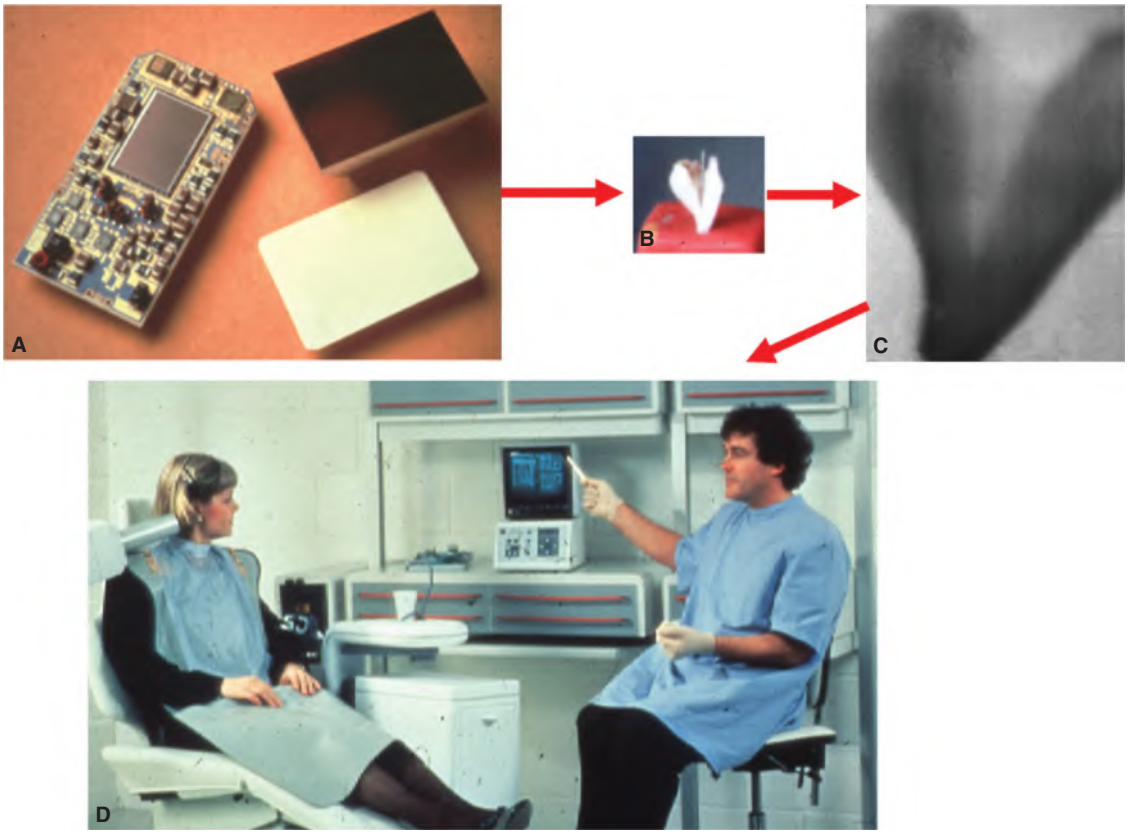

FIGURE 9B-1 ***A.*** components of the RadioVisioGraphy (RVG)-32000 showing the solid-state electronics board with charge-coupled device (CCD) chip, the scintillator, and the fiber-optic plate used to reduce the size of the image to the size of the CCD. The fiber optics were made of tungsten glass that protected the CCD from direct exposure to X-rays; ***B.*** Primary tooth of Mouyen's daughter; ***C.*** The first "clinical" RVG image achieved in Dr. Mouyen's living room of this tooth; ***D.*** Commercial version of the RVG with Mouyen himself in the blue gown.

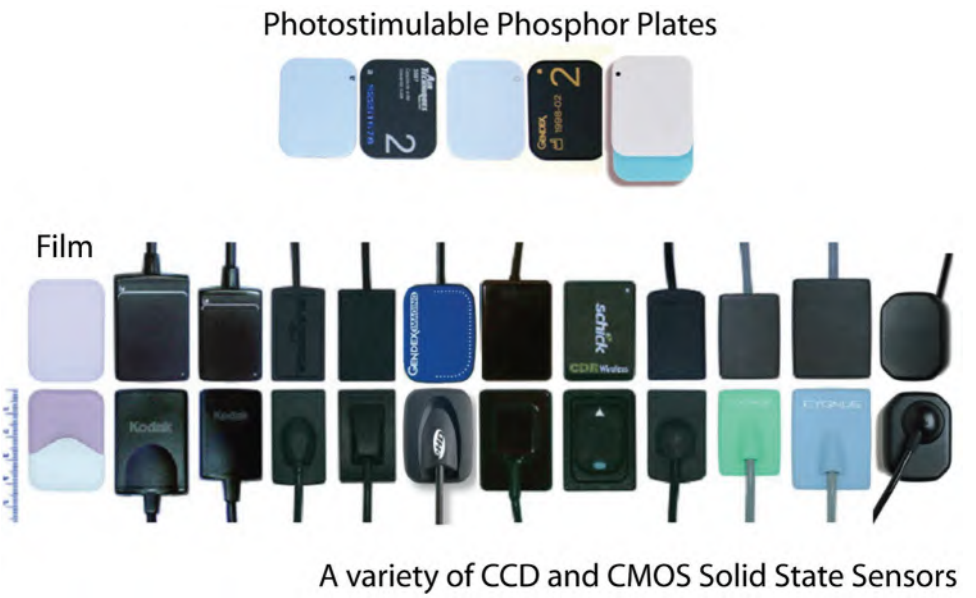

FIGURE 9B-2 Demonstration of the diversity of available intraoral X-ray sensors.

DIGITAL INTRAORAL X-RAY IMAGING OPTIONS

Digitization Using Secondary Capture of Analog Film Images

2-D Imaging

Conventional X-ray films consist of a polyester sheet covered on both sides by a thin layer of silver halide emulsion that absorbs X-ray photons resulting in a latent image. When the latent image is developed using developing chemicals, the silver halide crystals are converted into metallic silver (silver is not black but gives such an impression in granular from within the processed radiograph). The unexposed crystals are later removed from the emulsion during the fixing process. This procedure, including the unpacking of the film in a light safe environment, usually takes several minutes and can produce a visible image of high diagnostic quality even when automatic processing is utilized, as long as optimal procedures are employed. Unfortunately, optimal procedures are not always employed in dental practices. To digitize the analog film image adds another time-consuming step, and the extra step also leads to the possibility of impaired image quality. For more details, see Chapter 9A.

Once a visible film-based image has been produced, it is possible to use a flatbed scanner with a transparency adaptor to digitize the image by secondary capture. Flat bed scanners are preferred over cameras as the latter is more prone to optical distortion and added "noise" from light reflected from the surface of the radiograph film. For scanning, the parameters should be set at high-definition grayscale images and scanned at a minimum of 300 dots per inch (dpi) using a transparency adaptor and acquisition software parameters set to "transparency." Secondary capture images are dependent on the person making the input and associate such information as right versus left, patient name, procedure date, and so forth. This can be subject to "human error."

Conventional X-ray film has survived around 120 years in dental radiography. It obviously is capable of providing useful diagnostic information. However, disadvantages of analog X-ray film include the following:

1. Inefficiency as photon detectors, therefore requiring a relatively high radiation exposure.
2. Film packet is thin and can be felt by the patient to "cut into the tissues."
3. Film is not rigid and hence can bend resulting in a distorted radiographic image.
4. Produce static images with no availability of post-image treatments other than varying the brightness of the viewbox illumination.
5. Exposure errors and suboptimal processing conditions can result in poor image quality that is not immediately obvious.
6. Film processing is relatively time consuming.
7. Costs for maintaining a darkroom.
8. Silver in spent processing chemicals has the potential to cause environmental pollution.
9. Duplicate radiograph in a double film packet is of very slightly lower density compared with the top film. Physicochemical duplication of film radiographs results in copies of higher contrast and less detail.
10. Archiving radiographic film radiographs in physical patient files requires extra storage space in the office. Retrieving radiographs can take time.
11. Intraoral film radiographs not appropriately mounted and labeled cannot in themselves be associated for certainty with a given patient or time of exposure. Although one cannot exclude the possibility that digital radiographs may be attributed to the wrong patient, the use of a Digital Imaging and Communications in Medicine (DICOM) file format that is appropriately populated makes this less likely to occur.

DIGITAL RADIOGRAPHY

Figure 9B-2 represents some choices in intraoral digital X-ray systems for dentistry. Several of these systems may share an identical detector; however, they generally differ in electronics and the application software supplied. There is increasing acceptance of filmless X-ray alternatives in dentistry. As of 2015, at least half of the dentists in the United States were using digital X-ray detectors. This represented a fivefold increased penetration of the technology over the past few years. The adoption of digital imaging technologies by endodontists is undoubtedly much greater than is the case for general dental practice. With the continuous addition of improvements and sophistications in the already existing digital systems and introduction of new systems, the diagnostic quality of digital intraoral systems is now generally considered equal to conventional X-ray film even for tasks where film is known to do well such as in endodontic assessment and detection of proximal dental caries.[2]

Digital systems utilize computer technology in the capture, display, enhancement, and storage of radiographic images. Computers work on the binary number system consisting of two digits (0 and 1) to represent data. These two characters are called bits (binary digits), and they form "words" of eight or more bits in length called bytes. The total number of possible bytes for 8-bit language is $2^8 = 256$. The analog-to-digital converter transforms analog data to digital data based on binary number system. The strength of the output signal is measured and assigned a number from 0 (black or white depending on designation) to 255 (white or black—opposite of "0") according to the intensity of the electric signal. These numeric assignments translate into 256 shades of gray in an 8-bit system. A digital image consists of a number of pixels (picture elements), and each pixel is represented by a number corresponding to its gray level. The pixel is the smallest picture element of the image, and the resolution of an image is directly related to the pixel size among other factors. Images of 12, 14, or 16 bits will have a greater range of pixel values

and also consume greater memory space of the computer. For instance, a 12-bit image can have a range up to 4096 gray values, and window and level adjustments can permit selection of the gray levels to be displayed on the monitor as the latter is usually restricted to 256 gray levels at one time.

The two major technologies presently used in intraoral digital X-ray systems are as follows:

1. Solid-state detectors (Figures 9B-3 and 9B-4)
   a. Charge-coupled device (CCD)
   b. Complementary metal–oxide–semiconductor (CMOS)

Solid-state detectors, CCD and CMOS can be indirect detectors using a scintillating screen such as cesium iodide or gadolinium oxysulfide, or (less commonly) can use direct conversion of X-ray photons to electrons (e.g., Cadmium–Telluride technology).

2. Storage phosphor detectors (Figures 9B-5 and 9B-6)
   a. PSP

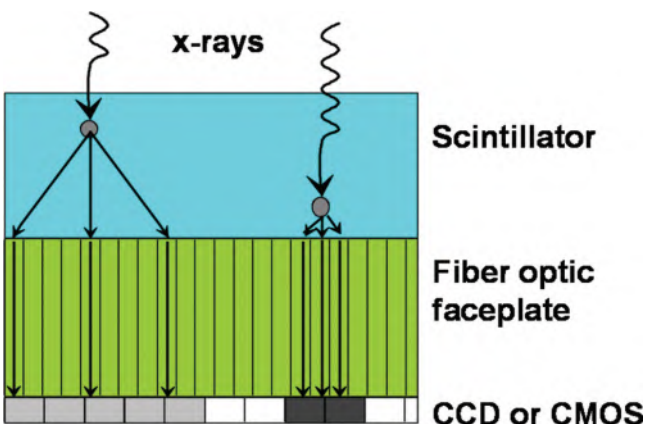

FIGURE 9B-3 Indirect solid-state X-ray sensor using a scintillator to convert X-rays to light photons. The image represents a small section of such a sensor.

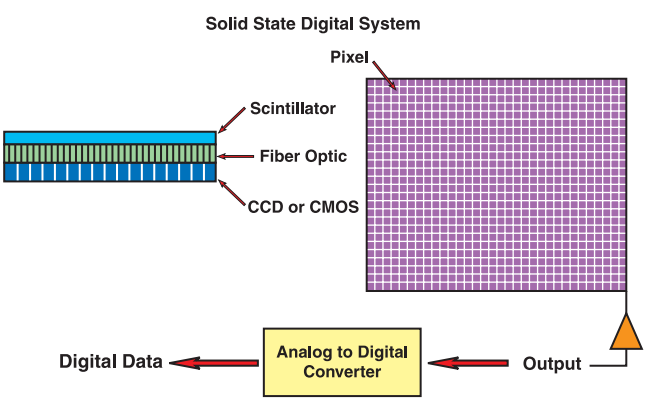

FIGURE 9B-4 Solid-state sensor process. With the charge-coupled device (CCD), the pixel wells are read out in sequence, whereas with the complementary metal–oxide–semiconductor (CMOS), each pixel can be read independently. CMOS sensors are employed more commonly than CCD technology.

FIGURE 9B-5 Example of a photostimulable phosphor (PSP) laser scanner (Scan-X; Air Techniques, Melville, NY, USA). Detail shows loading area for the individual PSPs. Scanners come in a variety of sizes and configurations from the various vendors of this technology.

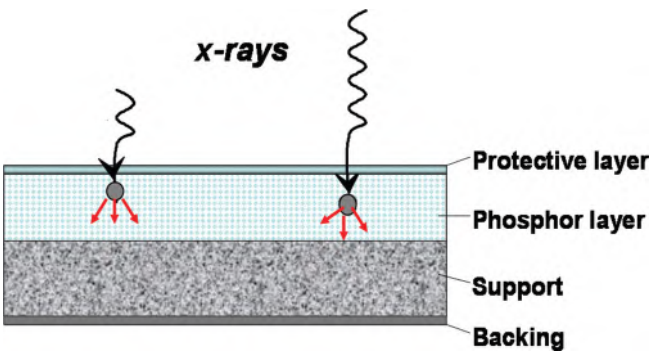

FIGURE 9B-6 The intraoral photostimulable phosphor (PSP) is composed of a phosphor layer, a thin protective layer, support, and backing. X-ray energy is stored during exposure and subsequently released as photoluminescence on application of energy from a scanning laser. The emitted light is photomultiplied and then converted to an electronic signal that is digitized. (Note that this figure is of the PSP during exposure only and represents only a small section of a PSP.)

WORKING PRINCIPLES OF DIGITAL INTRAORAL SYSTEMS[3]

Solid-State Systems

Complementary Metal–Oxide–Semiconductor Active Pixel Technology (CMOS-APS)

CMOS chips are commonly used in digital cameras, video cameras, and computers. Externally, CMOS detectors appear similar to CCDs, but the former use an active pixel technology, that is, each CMOS-APS has an active

transistor built into each pixel and provides a reduction by a factor of up to 100 in the system power (translating into electric voltage) required when compared with CCDs. This has permitted the introduction of wireless radio frequency (RF) transmission of the acquired image. Other advantages of the CMOS-APS include design integration and a potential for relatively low cost manufacture that, however, has not so far translated into cost savings for the consumer. In addition, the APS system eliminates the need for charge transfer between adjacent pixel wells extending the exposure latitude by suppressing "pixel blooming." Most intraoral solid-state digital X-ray sensors now employ CMOS technology. This technology is also used in some panoramic and CBCT systems.

Charge-Coupled Device

The CCD is composed of an electronic circuit embedded in several thin layers of silicon. The silicon chip usually is composed of an array of light sensitive pixels (picture elements), and each pixel consists of a small electron well into which the X-ray or light energy is deposited upon exposure. Each silicon atom in the detector chip is covalent with another silicon atom. When light photons strike the silicon and the energy exceeds the strength of the covalent bond, an electron hole pair is formed. Alternatively, electrons can be produced by a coating layer when direct technology is used instead of a scintillator layer. Either way, an electric charge is established by release of electrons. The electric charge in each "pixel" well is proportional to the incident X-ray or photon energy. Charge coupling is a process by which the electrons from one well are transferred to another in a sequential manner, and this transfer concept has been compared to a "bucket brigade." The charge of each pixel is converted from an analog electric signal representing the energy absorbed by the solid-state chip to a digital signal representing the discrete numeric pixel values for image display on a computer monitor. The CCD is still used in some digital solid-state intraoral and panoramic dental radiography systems, but less frequently now than CMOS technology.

STORAGE PHOSPHOR DETECTORS

Photostimulable Phosphor

The PSP imaging plate (IP) (Figure 9B-7) works on the principle of radiation-induced emission of photostimulated luminance. PSPs generally contain barium fluorohalide crystals with small amounts of bivalent europium atoms as an activator. When a storage phosphor IP is exposed to X-radiation, the europium atoms in the phosphor crystalline lattice are ionized liberating a valence electron. This results in the formation of electron vacancy. The valence electrons are exited to the level of conduction band where they travel freely until trapped by so-called Farbzentren centers present in halide crystals to form metastable electrons with an energy level slightly lower than the conduction band but greater than that of the valence band. These trapped metastable electrons constitute the latent image and their number is proportional to the number of incident X-rays. When the latent image is exposed to the red light of solid-state laser, the metastable electrons are again exited to reach high-energy conduction band where they recombine with Eu^{3+} atoms and return to low-energy valence bond ($Eu^{3+} + e^- = Eu^{2+}$). This results in the liberation of energy, emitted as blue light. The light is registered by a photo multiplier tube and converted into an analog electric output signal that is digitized, resulting in a digital image. Each pixel has a numeric value that is proportional to the amount of light emitted from the corresponding area of the PSP IP.

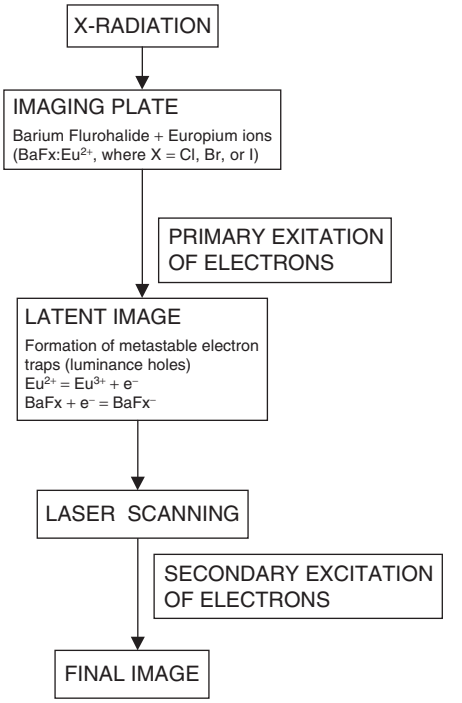

FIGURE 9B-7 Flow diagram to demonstrate the sequence involved in the formation of a photostimulable phosphor (PSP) image.

Advantages of Digital Intraoral X-Ray Imaging

Potential advantages of digital X-ray over analog film imaging include the following:

1. Reduced time between exposure and image formation when using solid-state detectors.
2. Reduced radiation dose per image sometimes possible.
3. Multiple exposures, from various angles, both vertical and horizontal, may be made without moving the sensor once positioned.
4. Elimination of chemical processing and disposal of spent chemicals.

5. Images can be duplicated any number of time without any loss of image quality (each is a perfect clone of the original).
6. Images can be stored and retrieved electronically.
7. Images can be transmitted electronically for referrals and other purposes.
8. Dynamic nature of the image with the ready option of post-imaging enhancements.
9. Digital systems also have measurement tools that, given a suitably positioned fiducial reference (e.g., an endodontic instrument of known length placed in a root canal), can accurately measure, for example, the root canal working length.
10. Reusable detector reduces expenditure on consumables.
11. With DICOM image file usage, digital images can provide greater security regarding radiographic image integrity and tags include such information as patient name, date of exposure, and laterality.
12. Wired detectors have an advantage when working on special needs patients as the wire makes swallowing or ingesting the detector unlikely.

Potential Disadvantages of Digital Intraoral Imaging over Analog Film

There are also some potentially negative issues that should be considered:

1. Relatively high initial investment cost if the practice has previously sunk costs in film-processing facilities.
2. Issues related to infection control as the intraoral detectors cannot be autoclaved.
3. Solid-state intraoral detectors are somewhat thicker and more rigid (however, this can also be an advantage in preventing disproportionate distortion from film bending).
4. Packaged PSP IPs are thinner than prepackaged analog intraoral X-ray films and may not be held firmly in position in film holders.
5. As most current versions of CCD and CMOS detectors are wired, this could create patient psychological discomfort. It might also be a drawback for the inexperienced operator, requiring adjustments in technique and patience through the learning curve of a new methodology.
6. Competency using software may take time to master. The learning curve may be longer or shorter depending on the computer literacy of the operator.
7. With PSPs, the intraoral (IPs) have been prone to mechanical degradation necessitating replacement of plates to sustain image quality.
8. Although unlikely to occur given reasonable diligence, mishandling can cause mechanical damage with high replacement costs for CCD and CMOS detectors.

Examples of Digital Intraoral Images Used in Endodontics

It is now appropriate to illustrate some examples of digital radiographic images used in the practice of endodontics so one might get a better appreciation of attainable image quality (Figures 9B-8 to 9B-10). It should, however, be remembered that the printed page is static and does not permit the modifications to image density and contrast that are available when the digital images are viewed on the computer monitor.

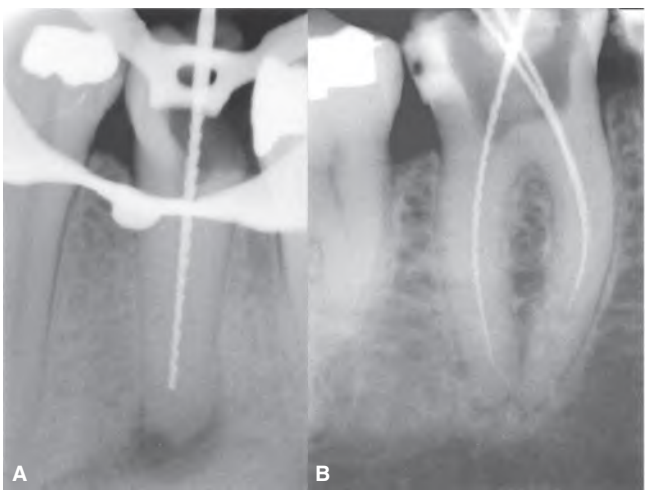

FIGURE 9B-8 ***A.*** endodontic instruments in a mandibular second premolar tooth with a radiolucent periapical pathosis; ***B.*** mandibular molar with slight widening of the apical periodontal ligament space of the distal root. These images were made using the DentX Eva (CMOS) system (AFP/DentX, Elmsford, NY, USA).

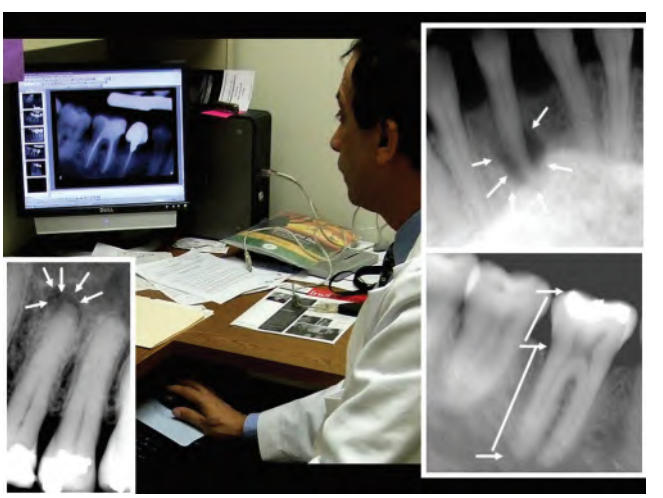

FIGURE 9B-9 Endodontic evaluations using the Kodak RVG-6000 (Kodak Dental Imaging, Marne-la-Vallée, France) CMOS system. These details clearly demonstrate the ability of solid-state sensors to demonstrate periapical lesions and tooth length (even with long root length in rhizodontia).

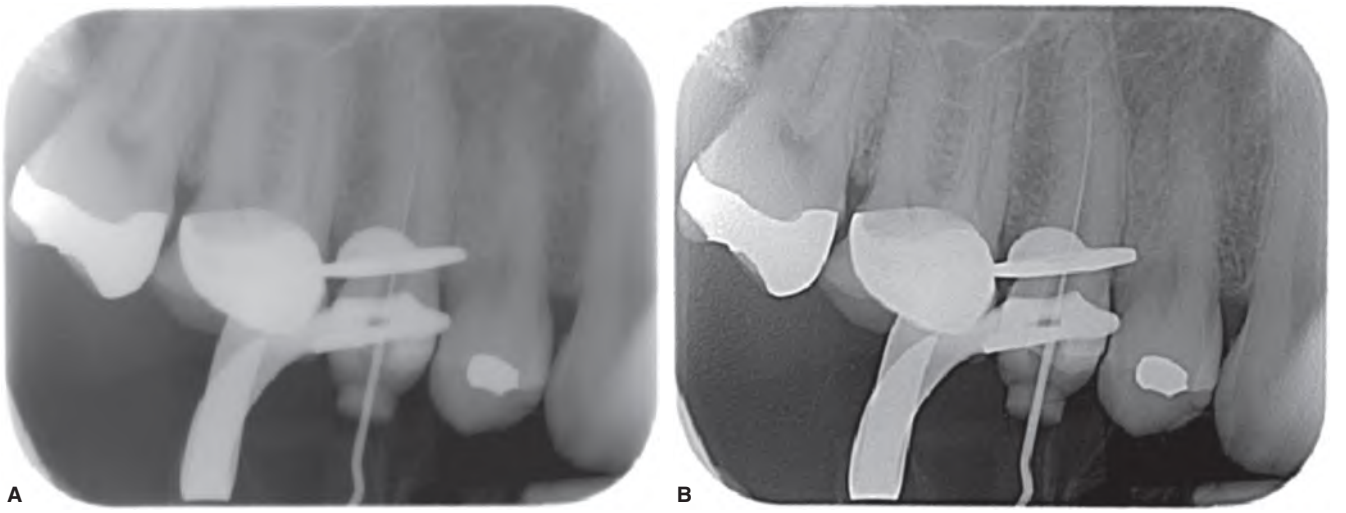

FIGURE 9B-10 Dörr Vista-Scan photostimulable phosphor image of endodontic instrument in place in tooth: ***A.*** without enhancement; ***B.*** with high-pass sharpness filter applied.

DIGITAL VERSUS CONVENTIONAL: WHICH IS BETTER?

With the introduction of digital imaging systems in dentistry, a question arises: "How effective are these systems when compared with film for specific diagnostic tasks in endodontic practice?" The reported studies are rather consistent and have shown that the diagnostic ability of the digital image is equivalent to film-based radiographs as referenced in the subsequent sections of this chapter. The following discussion concentrates on investigations concerning diagnostic tasks in endodontology.

DETECTION OF PERIAPICAL LESIONS

Accurate assessment of the periradicular status is crucial in diagnosis, treatment, and evaluation of healing in endodontic therapy. However, from some reported studies, it is believed that periapical lesion cannot be detected until a comparatively advanced bone destruction, that is, not until the lesion erodes the junction between cortical and cancellous bone.[4,5] Alternatively, it has been postulated that changes in the lamina dura are key to the detection of periapical lesions.[6,7] Several studies have evaluated the efficacy of digital images in detecting periradicular bone changes. Most of the studies, concluded that digital imaging did not increase the rate of detection of lesions.[6,8–11] Paurazas et al.[10] for instance, found no difference in diagnostic accuracy between film radiographs, CCD, and CMOS-APS systems in detecting periradicular lesions. However, the use of CMOS APS system has been supported because of perceived functional advantages over CCD systems such as "low cost," reduced electric power requirement, and suggested increased life time of the product. Digital imaging was preferred over film-based imaging as the former, according to one report, requires 50% less radiation than the latter.

Yokota et al.[8] studied mechanically created lesions in human cadaver specimens using film radiographs and an early vintage RVG CCD X-ray detector. It was concluded that (1) when no lesion exists, conventional radiograph was more accurate than the vintage RVG; (2) when the lesion is enlarged to involve lamina dura and medullary bone, the vintage RVG was superior; and (3) when the lesion involved the cortical bone, there was no difference between a conventional radiograph and vintage RVG. This study was in agreement with a similar study conducted by Tirrell et al.[12]

It has been reported that digital images have been found to be more accurate than conventional radiographs when image enhancements are performed.[13] In another study, image processing was found to be of limited value on diagnostic quality, but altering the basic functions such as contrast and brightness were preferred for the detection of periapical lesions.[14] However, Wallace et al.[15] concluded that enhanced digital images perform inferior to film-based images in periapical lesion detection. This was attributed to the observers' lack of familiarity with digital images and lack of experience with the image enhancement procedures.

Other image-processing functions such as color enhancement and reverse imaging have been found to be of limited diagnostic value in the detection of periapical lesions.[16] In this context, it should be noted that many times the enhancement leads to worse performance, although the subjective impression is that of improvement. It should be remembered that when enhancements are applied, they generally hide information, thereby making certain features stand out. Also many studies have used rather underexposed films or digital

detectors, before the enhancement of the digital images leading to improved performance of the digital system. One has to caution the readers about these inherent fallacies and also point out the great variation sometimes reported among observers in periapical radiographic diagnosis. When high variability between observers is present, one can predict that the studies concerned will result in no proven differences between the modalities compared. Woolhiser et al.[17] even reported great variation in the seemingly straightforward task of estimation of root length, with variations for the same observation being as great as 1.5 to 8 mm. Nevertheless, they did conclude that observers using enhanced digital images outperformed the same observers using analog film radiographs.

EVALUATION OF ASSESSMENT OF ROOT CANAL LENGTH

A too short working length can result in infected tissue being left in the root canal, and this can then continue to damage the periapical tissue. Hence, a correct estimation of working length is crucial for endodontic success. The radiographic apex does not always coincide with true apex (apical foramen). Even though, the International Standards Organization recommended smallest file size for a working length radiograph is #15, files smaller than #15 are required for initial instrumentation in fine, curved, and calcified canals to prevent root perforation or ledge formation within the canal. The problem encountered with small-sized files is that the instrument tip will not always be clearly visible on a radiograph. Comparative studies have been conducted between film radiographs and digital detectors to evaluate their accuracy in estimating endodontic length and perceived clarity in visualizing fine endodontic files. It has been reported that digital systems closely approximate film radiographs in the accuracy they provide for practitioners in estimating root length.[18,19] However, digital images were found to be inferior to film images when #10 rather than #15 files are used.[20,21]

Cederberg et al.[22] compared the difference in interpretation of the position of endodontic file tips between PSP imaging (Digora; Soredex, Helsinki, Finland) and intraoral radiographic film (Ektaspeed Plus; Kodak, Rochester, NY) for a limited number of patients (n = 13). Film radiographs were viewed using a 7× measuring magnifier. PSP was reported to outperform film radiographs in that a smaller difference was found between the file tip and root apex with digital imaging.

Eikenberg and Vandre[19] sectioned human skulls into 15 sextants. Teeth were then removed and 45 canals were instrumented to their apical foramina. Endodontic files were glued in place at random distances from the apical foramina. Image geometry was maintained by a custom-mounting jig. Images were captured with two types of analog film and a solid-state digital radiographic system (Dexis). Digital images were read on a conventional color monitor (cathode ray tube) and a laptop screen (active-matrix liquid crystal display). Fifteen dentists measured the distance from the file tip to the apical foramen of the tooth. Results showed that the measurement error was significantly less for the digital images than for the film radiographs.

Martinez-Lozano et al.[23] evaluated the diagnostic efficacy of an electronic system for the determination of working length, in comparison with two radiologic methods (conventional film and digital radiography). The study sample consisted of 28 root canals in 20 human mandibular teeth. A comparison was made between the working length measurements obtained by the radiologic methods and an apex locator, using as "gold standard" the observation of the actual file position within the root following selective grinding of the root tissue. It was concluded that none of the techniques was totally satisfactory in establishing the true working length. No statistical differences were proven comparing the techniques investigated.

DETECTION OF ROOT CANAL ANATOMY

Success of endodontic treatment depends on the identification of all the canals in the tooth that can then be cleaned, shaped, and obturated. The ability to locate all the canals is crucial in determining the eventual success of the therapy, and radiographs are considered one of the important tools apart from clinical examination. Reported studies are contradictory to one another. Naoum et al.[24] compared conventional with PSP images to visualize the root canal system contrasted with radiographic contrast medium. The image clarity on conventional radiographs was found to be better than with Digora PSP images.

Nance et al.[25] studied maxillary and mandibular molars using conventional D-speed film radiographs versus tuned aperture computed tomography (TACT) in the identification of root canals. TACT, using arrays of several digital images to tomosynthesize the third dimension, was reported to be superior to conventional film radiographs in the detection of root canal in molars. There was a reported 36% detection rate of the (elusive) second mesiobuccal canal (MB2) in maxillary molars and 80% detection rate of the mesiolingual canal in the mandibular molar using the TACT system. By contrast, with the film-based images, there was a somewhat unbelievable 0% detection of MB2 canals. This highly skewed result might be caused by using a standard paralleling technique for making conventional images, leading to overlap of canals. In a similar study by Barton et al.[26] parallax analog and parallax digital images were compared with TACT in the ability to detect MB2 canals in maxillary first molars. With TACT, there was 37.9% chance of locating the MB2 canal, which is consistent with the results

of Nance's study. However, in this study, the success rate of detecting MB2 canals using film-based images was approximately equal to that using TACT, and TACT did not significantly affect the rate of detection of MB2 canals. It was also observed that the distance between first mesiobuccal and the MB2 canal is quite small and perhaps too minute for current TACT applications.

Digital Subtraction Radiography

Given radiographs taken in precisely the same position and with the same beam geometry and exposure parameters, images can be subtracted to show changes over time. This technique has been largely conducted for research purposes in view of the difficulties experienced in practice in achieving images with reproducible projection geometry over time.

Digital subtraction has been performed for endodontic tasks, and digital detectors have been used for this purpose (Figure 9B-11).[27–29] Yoshioka et al.[27] evaluated the efficacy of digital subtraction radiography using an early version of the RVG system in a follow-up study of endodontically treated teeth. The intra-image variation of the original image caused by dark current and sensitivity variations among pixels in this vintage digital system was corrected pixel-to-pixel. The inter-image variation was further corrected using a copper step-wedge attached to the detector. Standardized images were obtained using the same beam geometry at patient follow-up. Pixel values at the regions of interest, positioned on the periapical lesion, increased after endodontic treatment, and this change continued during an observation period of up to 545 days. It was concluded that subtraction radiology is possible for the purpose of endodontic follow-up.

Nicopoulou-Karayianni et al.[28] also evaluated the effect of root canal treatment on periapical lesions by conventional and subtracted digital radiographic images of patients. Better observer agreement was achieved by digital subtraction radiography during the evaluation than without digital subtraction at follow-ups 3 to 12 months after treatment.

Mikrogeorgis et al.[29] evaluated the suitability of digital radiograph registration and subtraction in the assessment of the progress of chronic apical periodontitis. Ninety cases of teeth with chronic apical periodontitis were studied. In each case, a preoperative radiograph was made, root canals were prepared, and a calcium hydroxide paste was placed in the root canals. Radiographic control and replacement of calcium hydroxide paste took place at 15-day intervals before final obturation. The root canals were obturated 1.5 months after the first appointment, and recall radiographs were made 2 and 6 weeks, 3, 6, and 12 months after the obturation. Digital radiography using a solid-state detector was employed. Changes to the periapical tissue structure were detectable using digital subtraction, even during short time intervals.

Digital subtraction is effective but also time consuming. From 2014, the CDT procedure codes used in the United

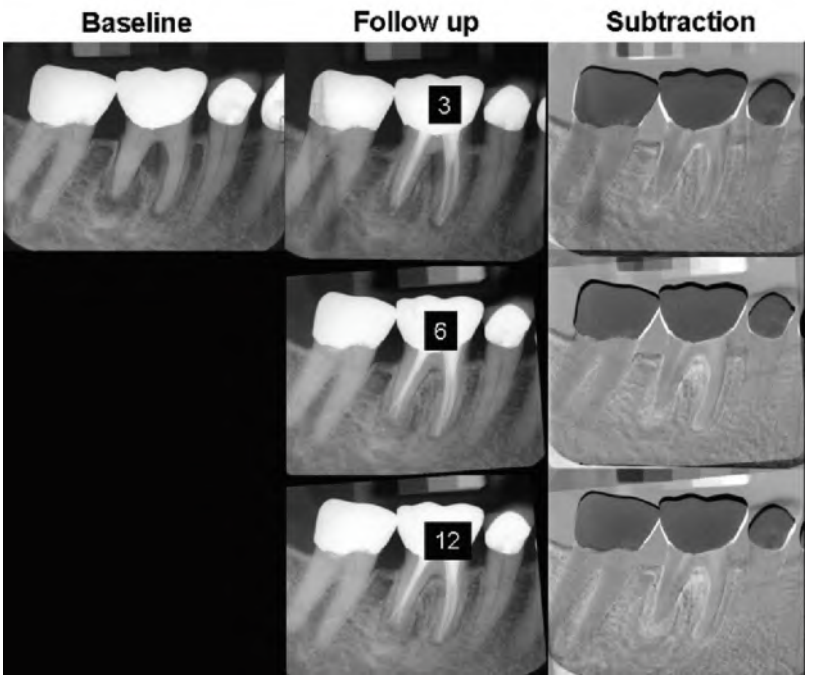

FIGURE 9B-11 Digital subtraction comparing baseline (column 1) to follow-up images made at 3, 6, and 12 months following endodontic therapy (column 2). Column 3 indicates subtraction of respective follow-up images from the baseline radiograph. Progressive remineralization of the radiolucency at the apex of the distal root of the first molar is evident.

States has included an image post-processing code for digital subtraction.

Radiation Dose for 2-D Imaging

The *A*s *L*ow *A*s *R*easonably *A*chievable (ALARA) principle advocates the use of the smallest possible radiation dose that can still produce an image without compromising diagnostic quality. Conventional direct exposure emulsion X-ray films are relatively poor detectors of radiation and can require relatively high radiation dosages to produce images of optimal diagnostic value.

By contrast, digital detectors can be more quantum efficient and, therefore, require lower radiation exposure. On the other hand, intraoral X-ray films are now available in higher speed than heretofore and this has greatly increased film's quantum efficiency to radiation. Hence, with the use of high-speed films, it is possible to obtain high-contrast images at relatively low exposure. Underexposing X-ray film still tends to increase radiographic "mottle" and graininess of the images, giving an impression of a lack of sharpness in the image.[30] This finding was supported by Sheaffer et al.[31] who found that underexposed radiographs, in an attempt to minimize radiation dosage, result in inferior images irrespective of film speed.

With the invention of increasingly more efficient digital X-ray detector technologies, with an added advantage of contrast enhancement as well as reported reduction in radiation dose, it might be possible to achieve diagnostically efficient images at lower exposures than with conventional film. Velders et al.[32] found that it was possible to achieve a dose reduction of approximately 95% compared with Ektaspeed film when digital systems were used for dental radiographic imaging. One should remember, however, that the use of such low dosages can impair image quality because of a reduced signal-to-noise ratio (SNR).

Although the possible dose reduction for thinner objects has, however, been questioned for a PSP detector, in a study by Huda et al.[33] the low-contrast discrimination of PSPs was found to be superior to that of X-ray films when using the same exposure time. By way of contrast, in a study by Berkhout et al.[34] it was found that the PSPs required 10 times more radiation than the minimally acceptable dose to produce images of preferred quality. Further, these authors state that PSPs require more radiation than E-speed films and that dose reduction with digital technology can be achieved only when using solid-state systems. PSP systems have a wide dynamic range, much larger than CCD systems and films. PSPs are said theoretically to have an exposure latitude ratio of approximately 10,000:1.[33] Hence, they should be capable of producing usable images over a wide range of exposures.[33–38] The actual exposure latitude for PSP systems is dependent on the acquisition software and scanning electronics. It does not exceed 25:1 for PSP intraoral radiography when the relative exposure latitude is determined using clear discrimination of the enamel–dentin junction as the lower limit and pixel blooming or unacceptable levels of cervical burnout as the upper limit.[39]

The effective latitude range for solid-state detectors can exceed 20:1 for some CMOS systems. However, intraoral X-ray systems using a CCD generally have narrower recording latitudes (<10:1), because high exposure results in pixel blooming.[39]

Dynamic range, also known as latitude, corresponds to the range of exposures that will produce images within the useful density range. Radiographic modalities, that produce diagnostic images over a wide range of exposures, are said to have a wide dynamic range and vice versa. The Hurter and Driffield (H&D) curve was invented to characterize photographic emulsion some 5 years before the discovery of the X-ray but was extended to include X-ray film radiographs (Figure 9B-12). Using pixel values rather than film density, the H&D curve concept can also be applied to digital imaging. The straight-line portion of the curve for film corresponds to the dynamic range. This curve demonstrates, for conventional film, the relationship between exposure (log number of X-rays) and the optical density (darkness) of an image detector. The scale of useful densities for film radiographs ranges approximately from 0.5, that produces low density or light images, to 3.0, that results in darker images of high density. However, outside this range, images tend to lose diagnostic detail. For films, this curve takes a form of stretched letter S appearance, with the top horizontal portion of the curve called "shoulder" that corresponds to higher exposure and the lower portion called "toe" that corresponds to lower exposure. Exposure changes in these portions of the curve have little effect on density. Even little changes in the straight-line and vertical portion of the curve significantly affect image density. Thus, the more vertical the straight-line portion of the curve, the smaller is the latitude. This explains the relatively wider dynamic range of digital receptors, especially storage phosphors. The curve can be linear and wider with no shoulder or toe when compared with film images (Figure 9B-12).

Many digital systems have an option of using an "image equalize" function, and this is commonly the automated default setting. Equalization is a method of enhancing the

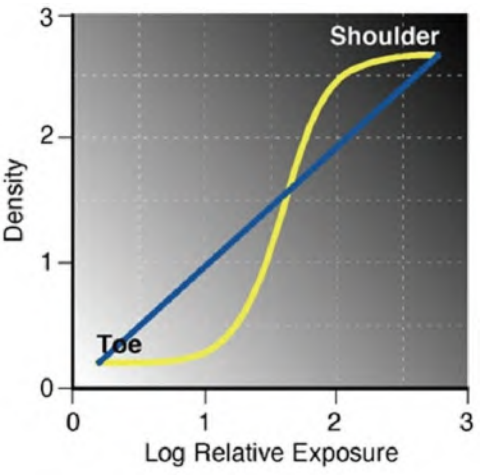

FIGURE 9B-12 Hurter and Driffield curve relating radiograph density to exposure.

contrast in a nonlinear fashion by stretching the pixel values disproportionately to obtain images of optimum image quality. In a study by Hayakawa et al.[40] equalization exposure compensation for computed dental radiography was found to be useful only for underexposed images but not for overexposed images. They also found that this compensation for underexposure did not significantly change the SNR. Furthermore, other studies have found that lower exposures, when severe, can in fact result in increased background noise and thus negatively affect the detection of small and low-contrast details within an object or tissue.[41]

Similar to equalize function in PSP images, the fourth generation RVG had an option of automatic exposure compensation (AEC) that compensated for exposure errors by stretching the pixel value range to increase the contrast of structures within the images. It was found that both under- and overexposed images can be compensated using this algorithm. Because it can compensate for overexposure, it might lead to the risk of practitioners overexposing their patients. Moreover, it was also found that AEC reduces the SNR and produces disproportionate pixel values relative to exposure.[40] Newer versions, such as the RVG-6200, have dose indicator that warns the dentist in cases of exposure errors.

There is evidence that dentists, particularly endodontists, routinely prefer and view high-contrast and relatively dense images when performing endodontic tasks.[31] Likewise, detection of occlusal caries was found to be more accurate when using darker radiographs.[42] This is probably because higher exposure allows easier discrimination between small and low-contrast objects. Moving the exposure difference up on the H&D curve increased contrast results. The smaller the change in structure, the greater the contrast required to differentiate between adjacent structures. One of the methods employed to achieve required contrast is increasing the exposure. Furthermore, if there is superimposition of structure by the images of other objects, the signal of the latter has to be handled as additional noise. Thus, the dose has to be increased in order to notice the structure in the image. This is proposed by Thoms,[43] who believes that attempting a dose reduction will result in images having a low SNR and thereby reducing the visibility of small structures. However, higher exposures also negatively affect the diagnostic accuracy. Whereas images with lower exposures resulted in more false negatives, overexposed images resulted in higher false-positive responses.[44] With these rather contrasting studies in the literature and unanswered questions, there is confusion regarding the exposure time that is actually required to produce images of diagnostic quality and more so in the detection of low-contrast objects. Questions that still need to be addressed are as follows: (1) What is the optimal exposure time or range of exposures required by each of the intraoral radiographic systems that are presently used? (2) Is high radiation exposure really necessary for endodontic tasks? (3) What are the effects of radiation dose on the detection of low-contrast objects such as fine root canals? Many of these questions have not yet been fully answered even for analog film radiography.

While endodontics is not frequently used on children, the American Association of Endodontists has joined the Image Gently Alliance that demands especial care when taking images on children using x-radiation. Children are thought to be especially affected by untoward effects of ionizing radiation due to the more rapid turnover of cells and the longer life expectancy. The aim is to child-size dose by appropriate selection of radiographs based upon inspection and professional judgment rather than routine, use of the fastest receptors consistent with diagnostic images, appropriately collimating the beam as much as possible, and use of a thyroid shield.[45,46]

SUMMARY

Digital imaging systems, using solid-state detectors, have definite advantages over analog film in terms of immediacy of image display and seem to be equal to film imaging for most tested diagnostic purposes. There is still a need, however, for further scientific validation of the best exposure strategies for imaging optimizing specific diagnostic tasks including those used for endodontics.

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Chapter 9C

Cone Beam Computed Tomography

Martin D. Levin, Allan G. Farman

Cone beam computed tomography (CBCT) is a mature technology that has a definite place in endodontic evaluation under certain circumstances. CBCT allows for the undistorted three-dimensional visualization of the dentition and the maxillofacial skeleton without the compression of anatomic features. CBCT may be used if a patient's chief complaint, medical history, clinical examination, and intraoral radiographic assessment demonstrate the need for additional information.

Imaging is achieved by use of a rotating gantry to which an X-ray source and detector are fixed. A divergent pyramidal or cone-shaped beam of x-radiation is directed through the center of the area of interest onto an area X-ray detector on the opposite side. The X-ray source and detector rotate around a fulcrum fixed within the center of the region of interest. During the rotation, hundreds of sequential planar projection images of the field of view (FOV) are generally 180° or greater. (Figure 9C-1). This procedure varies from a multislice CT (MSCT) that uses a fan-shaped X-ray beam in a helical progression to acquire image slices of the FOV and then stacks the slices to obtain a 3-D representation. Each slice requires a separate scan and separate 2-D reconstruction (Figure 9C-2) Because CBCT exposure incorporates the entire FOV, only one rotational sequence of the gantry is necessary to acquire enough data for image reconstruction for most maxillofacial purposes.

CBCT was initially developed for angiography,[1] but more recent medical applications have included radiotherapy guidance and mammography.[2,3] CBCT is less expensive in components than MSCT and provides a reduction of image unsharpness that might be caused by the translation of the patient during MSCT. Additional advantages of CBCT are faster exposure time, reducing likelihood of patient movement, and a reduction in radiation dose. However, its main disadvantage is a limitation in image quality related to noise and contrast resolution because of increased scattered radiation resulting in lower signal strength relative to back ground noise. While CBCT is excellent for hard tissue (bone window) imaging, contrast between soft tissues is generally poor. Fortunately, hard tissue discrimination is paramount in endodontic assessments. Further, CBCT can achieve the high resolution needed for endodontics while using a relatively low radiation dose.

It has only been since the late 1990s that sufficiently inexpensive powerful computers and X-ray tubes capable of

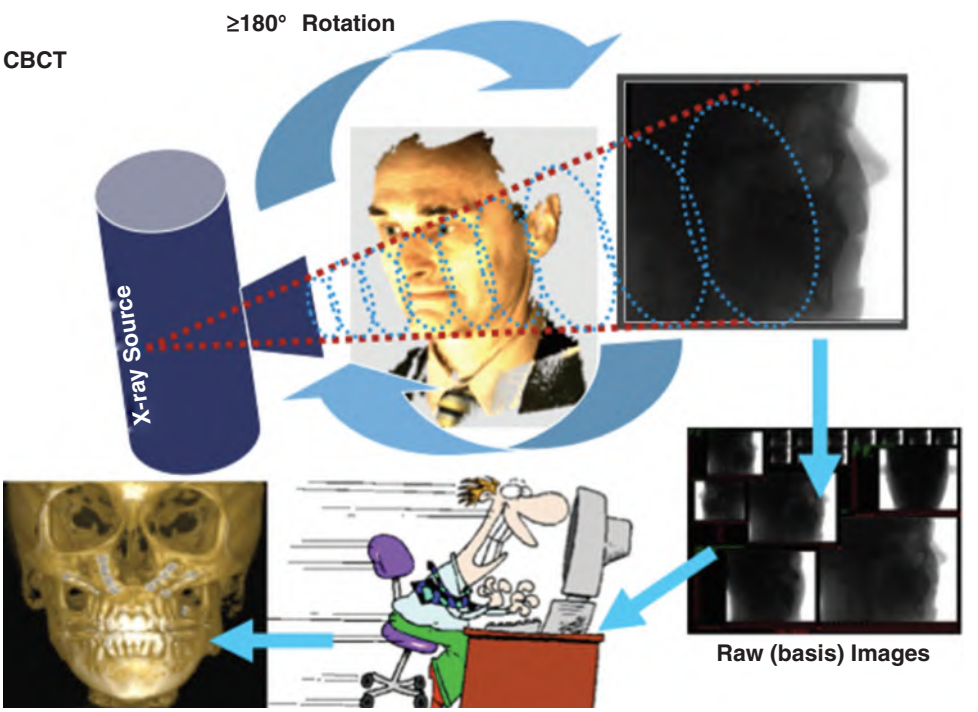


FIGURE 9C-1 Schematic illustration demonstrating CBCT operation. A collection of 2-D transmission images are integrated via back-projection filtering to provide image data that can be viewed in 3-D or as single slices in any plane.

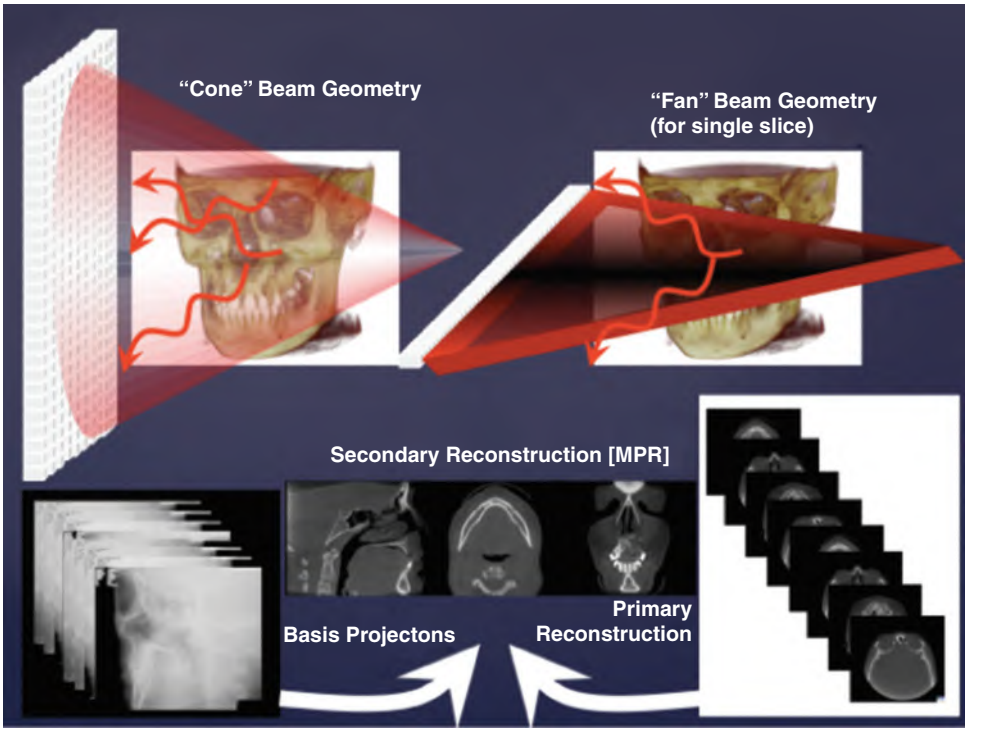


FIGURE 9C-2 Comparison of CBCT and fan beam CT.

continuous exposure have enabled clinical systems affordable for use in the dental office, to be manufactured. Additional critical components of the CBCT system include detectors capable of rapidly receiving and clearing multiple images without afterglow, plus the development of algorithms to convert the successive 2-D basis images into a 3-D image volume.

CBCT is a valuable technology when used wisely. Considerations for minimizing patients' exposure to radiation to as low as reasonably achievable include: (1) image selection based upon professional judgment of diagnostic or treatment guidance need rather than using ionizing radiation exposure as a "routine" without prior individualized patient assessment by the dentist; (2) minimizing the FOV by collimation, to reduce radiation exposure to tissues that need not be imaged; and (3) using the fastest exposure/lowest dose parameters consistent with adequacy in diagnostic image quality.

The restriction of radiation dose is particularly important for younger individuals. The International Commission on Radiological Protection (ICRP) has indicated that the effect of dose to children can be as much as three times that for young adults aged 20 to 25 years and as much as an order of magnitude more than for mature adults aged around 60 years.[4,5] Children are more susceptible to untoward effects of ionizing radiation than adults, due to the longer life ahead of them during which changes might occur, and also due to their faster turnover of cells compared to adults. CBCT should only be performed after clinical evaluation of the patient by the dentist, and a after a judgment is made that the benefits of this procedure will sufficiently outweigh the risks to the patient even though these risks are small. Additional guidance on the use of CBCT in endodontics can be found in the 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. (Available online at http://aae.org or http://aaomr.org)

Many of the commercially available CBCT units (Figure 9C-3) have separate CBCT and panoramic detectors, allowing increased functionality. This multimodal design may allow a limited scan volume to be supplemented by a panoramic radiograph (Figure 9C-4) to assess the entire mandible and maxilla when necessary.

FIELD OF VIEW AND SPATIAL RESOLUTION

In endodontics, the region of interest (ROI) is generally small and predetermined. The extent of anatomy imaged in a CBCT scan is dependent on the scan volume or FOV selected, and is controlled by the detector size and shape, beam projection geometry and beam collimation. The FOV is generally divided into large, medium and limited sizes and is determined by the task at hand. The scan volume should only be slightly larger than the anatomy of interest, resulting in lower x-radiation exposure to the patient.

Additional benefits of limited field size are smaller volumes to be interpreted and increased resolution to allow for the detection of very small features, such as alterations of the periodontal ligament space that measures approximately

FIGURE 9C-3 Examples of hybrid limited FOV CBCT units. ***A.*** Carestream Dental CS 8100 3D; ***B.*** J. Morita Veraviewepocs 3D F40. (Courtesy of Carestream Dental, Atlanta, GA, U.S.A and J. Morita, Osaka, Japan.)

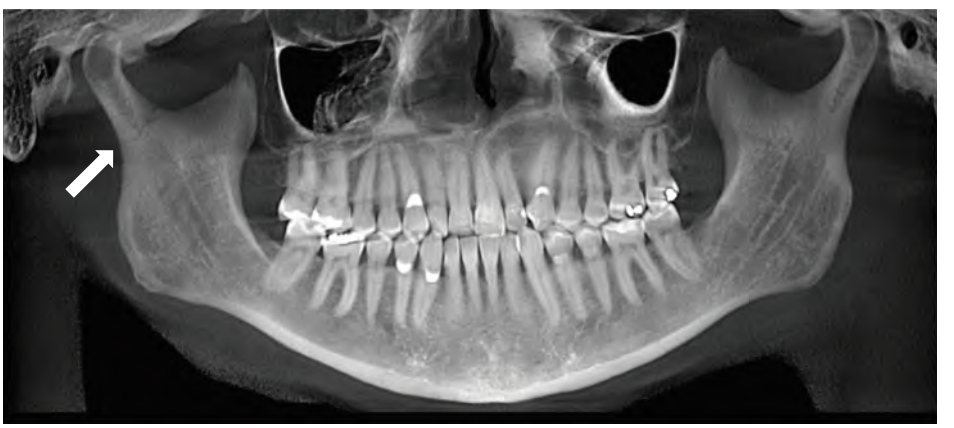


FIGURE 9C-4 Virtual panoramic radiograph shows a nondisplaced fracture through the buccal and lingual cortices of the right condyle. The angle, body, symphysis, and left condyle are intact. (Courtesy of Dr. Jarred Abel, Chevy Chase, MD.)

200 μm.[6] At this time, the nominal voxel resolution is reported to be 0.075 mm.

Bauman et al.[7] in examining the literature for detection of mesiobuccal canals in maxillary first molars found that as many as half of second canals could be missed using 2-D imaging, whether analog film or digital detectors were used. CBCT was successful in accurate detection of the number of mesiobuccal canals in 93.3% of cases when using 0.125 mm voxel resolution and the maximum number of basis images were employed. High resolution was essential for achieving this accuracy as with a setting of 0.4 mm voxel resolution (and half the number of basis images) the accuracy decreased to 60.1%. At 0.2 and 0.3 mm resolution settings the accuracies of detection were 77.7% and 88.8%, respectively. This study demonstrated the value of CBCT use in endodontics and also demonstrated that imaging parameters need to be selected carefully to assure resulting accuracy.

INTERPRETATION

All radiographs require careful and complete analysis by the ordering clinician because they may demonstrate findings that affect the health of the patient. A systematic

review and documentation of the findings is essential. Clinicians are responsible for evaluation of the entire image volume. Interpretation questions should be referred to an oral and maxillofacial radiologist.[8] For more details, see Chapter 10.

DETECTION OF APICAL PERIODONTITIS

Essential elements in endodontic assessment are patient chief complaint, history and clinical and radiographic examination. Intraoral radiographs should be used in the initial evaluation of the endodontic patient. Planar imaging modalities offer the possibility of accessible, relatively inexpensive and low radiation assessments of the teeth, supporting structures and the location of anatomic features. However, these *in vivo* imaging methods suffer from inherent limitations such as superimposition, magnification, geometric distortion, and misrepresentation of structures. Early studies showed that experimental bone lesions that did not affect the cortex eluded detection using periapical radiography.[9] The addition of cross-sectional imaging allows for the assessment of complex root canal morphology, the true extent of lesions and their spatial relationship to anatomic structures.[10] CBCT should be considered in the presence of a clinical condition that would likely benefit the patient if tomographic imaging is employed.

CBCT has been shown to be superior to conventional imaging in many endodontic applications. Lofhag-Hansen et al.[11] compared the accuracy of three calibrated oral radiologists using two paralleling intraoral radiographs with a 10° horizontal tube shift between exposures to assess 46 teeth. The same patients were scanned with a high-resolution, limited FOV CBCT that identified additional clinically relevant information in 69.5% of the teeth imaged. Although they showed that 10 teeth with small periapical radiolucencies were undetected using conventional imaging, 3 teeth with large lesions that perforated the cortex in the buccolingual direction were invisible to the observers. Low et al.[12] evaluated 156 roots of maxillary posterior teeth and found that CBCT identified 34% more lesions than periapical radiographs. Both studies showed that CBCT can detect apical radiolucencies in the cancellous and cortical bone that are normally hidden by the superimposition of unrelated structures onto the features of interest.[13]

Bone alterations associated with apical periodontitis may be undetectable on periapical projections because the lesions are small or are concealed by superimposition. Tsai et al.[14] determined that two CBCT units showed poor accuracy in detecting simulated lesions smaller than 0.8 mm, better accuracy if the lesion measured 0.8 to 1.4 mm and high accuracy when the lesion measured larger than 1.4 mm. Periapical radiography showed poor accuracy in all lesion sizes tested. Patel et al.[15] placed small and large synthetic lesions in the cancellous bone surrounding six human mandibular molar distal roots. CBCT images were 100% accurate in showing the presence (sensitivity 1.0) and absence (specificity 1.0) of periapical lesions. Periapical radiographs showed 24.8% of periapical lesions (sensitivity 0.248) but were accurate in showing the absence of lesions (specificity 1.0). They concluded that superimposition and poor geometric factors decreased lesion detection. CBCT resulted in improved lesion detection because the tooth could be viewed at any angle showing the true nature of the periapical bone destruction. Cortical bone lesions were detected with greater accuracy than in trabecular bone.

In a systematic review and meta-analysis regarding the diagnostic accuracy of conventional and digital intraoral radiography and CBCT imaging to detect apical periodontitis, Leonardi Dutra et al.[16] found only nine eligible studies, of which six qualified for meta-analysis. They found little or no data about inter- or intraobserver agreement. They also noted variation in sensitivity and that sensitivity while examining artificial apical periodontitis may have resulted from differences in bone lesion size, anatomic region studied, observer experience, and other factors. The study concluded that conventional and digital intraoral periapical radiography showed good accuracy, 0.73 and 0.72, respectively, in the discrimination of artificial apical periodontitis from no lesion; and CBCT showed excellent accuracy values (0.96). A reported limitation of this analysis was that all studies included were in vitro and involved skeletal tissues without diffuse borders associated with naturally occurring apical periodontitis.

Intraoral radiography compresses a three-dimensional object into a two-dimensional image. It requires an optimized geometric configuration of the X-ray generator, tooth, and detector to provide an accurate projection of the tooth.[17] Complex anatomy, where the superimposition of unrelated structures onto the features of interest may present challenges to lesion detection, are well documented in the literature. For example, imaging of the maxillary posterior teeth with a periapical radiograph in the presence of a low palatal vault may result in suboptimal geometry, causing superimposition of the zygomatic arch, sinus floor, palate, alveolar bone, and overlying teeth onto the roots. Radiolucencies in close proximity to the sinus floor are often missed with periapical radiography.[18] It can be difficult to interpret the status of each root, even if several angles are acquired. CBCT can accurately produce three-dimensional images of the teeth, supporting structures, and spatial relationship of anatomic features.[19] Using available tomographic slicing techniques, each root can be longitudinally aligned in parallel with the sagittal or coronal slicing tool, allowing undistorted examination of the axial slice.

ROOT CANAL TREATMENT OUTCOMES

Determination of the outcome of root canal treatment depends on the clinical signs and symptoms and radiographic assessment. Radiological assessment is important because many teeth with periapical lesions are asymptomatic (Figure 9C-5).

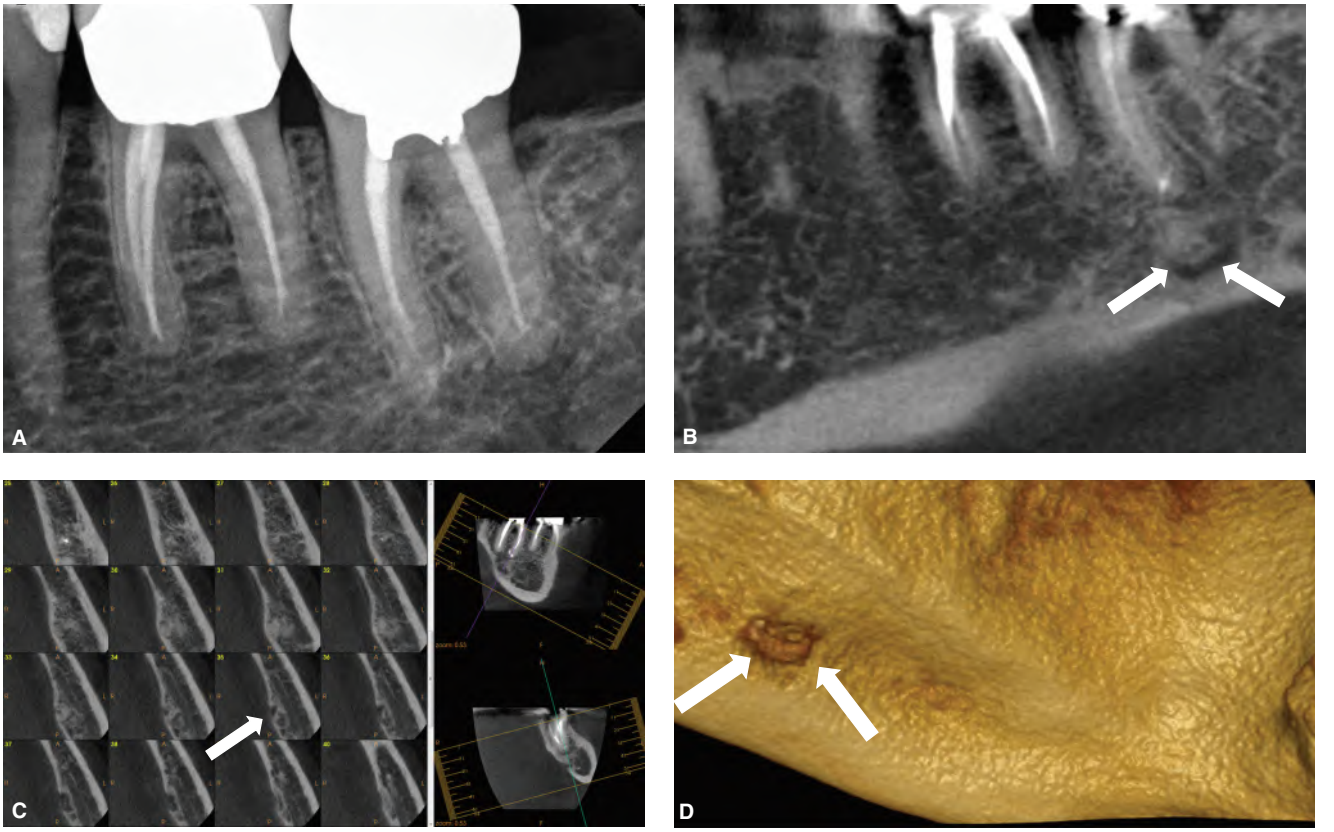


FIGURE 9C-5 Outcome predictors based on intraoral radiography and CBCT vary. ***A.*** Periapical radiograph shows previous incomplete endodontic treatment of an asymptomatic mandibular left second molar. There are short metal posts in the mesial and distal roots, and an area of low radiodensity at the furcation that extends to the distal aspect of the distal root. ***B.*** CBCT panoramic reformation shows a periapical discontinuity of the lamina dura and widening of the PDL space, with periapical osseous rarefaction that appears to track along the distal aspect of the mesial root in a narrow channel, to a large region of low radiodensity at the furcation, and also appears to track inferiorly to a 6-mm diameter somewhat well-defined serpiginous defect (solid arrows), causing erosion of the lingual cortex. ***C.*** CBCT-correlated multiple cross sections show two unilocular regions of low attenuation with well-defined, corticated borders constituting a multilocular lesion lying along the lingual cortical plate (solid arrow). ***D.*** 3-D reformation of the same area shows erosion of the cortex (solid arrows).

Outcome predictors using intraoral and tomographic imaging vary, and are further influenced by methodological rigor, such as inclusion and exclusion criteria. Wu et al.[20] reported that previously published systematic reviews of endodontic treatment outcomes showed a high percentage of cases confirmed as healthy by intraoral radiographs revealed apical periodontitis on CBCT and by histology. Furthermore, they stated that additional factors such as extractions, retreatments, low reevaluation rates, and the location of teeth evaluated limit the value of these studies. Patel et al.[21] compared the outcome of primary root canal treatment after 1 year in 123 teeth using periapical radiography and CBCT imaging. The absence of a radiolucency was 92.7% using periapical radiographs and 73.9% using CBCT scans. They concluded that periapical imaging showed a higher healing rate for primary endodontic treatment than CBCT.[21] The size of periapical rarefactions were underestimated when using two-dimensional projections because apical periodontitis confined within the cancellous bone or covered by a thick cortex was not visible using intraoral imaging.[22] Paula-Silva[23] compared endodontic treatment outcomes in dogs using periapical radiographs, CBCT, and histologic measurements after 180 days. CBCT closely corresponded with microscopic lesion measurements, whereas periapical radiographs consistently underestimated lesion size.[23] Temporal evaluations of healing using periapical radiography were adversely affected by the difficulty in achieving an exact geometric match.[24] In a retrospective study of 115 teeth, Liang et al.[25] reported that at the check-up visit, periapical radiographs and CBCT showed periapical areas of low density" on 12.6% and 25.9% of teeth evaluated, respectively. Outcome predictors using periapical radiographs were obturation length and density of root filling, whereas outcome predictors using CBCT were density of root filling and quality of coronal restoration. Periapical radiographs, which reliably show the mesiodistal direction only, underestimated the voids per root canal filling (16.1%) when compared with CBCT (46.2%).

CBCT can offer undistorted multiplanar imaging information that shows lesions hidden by superimposition that cannot be shown by periapical radiographs. (Figure 9C-6) The use of CBCT to assess the outcome of treatment should

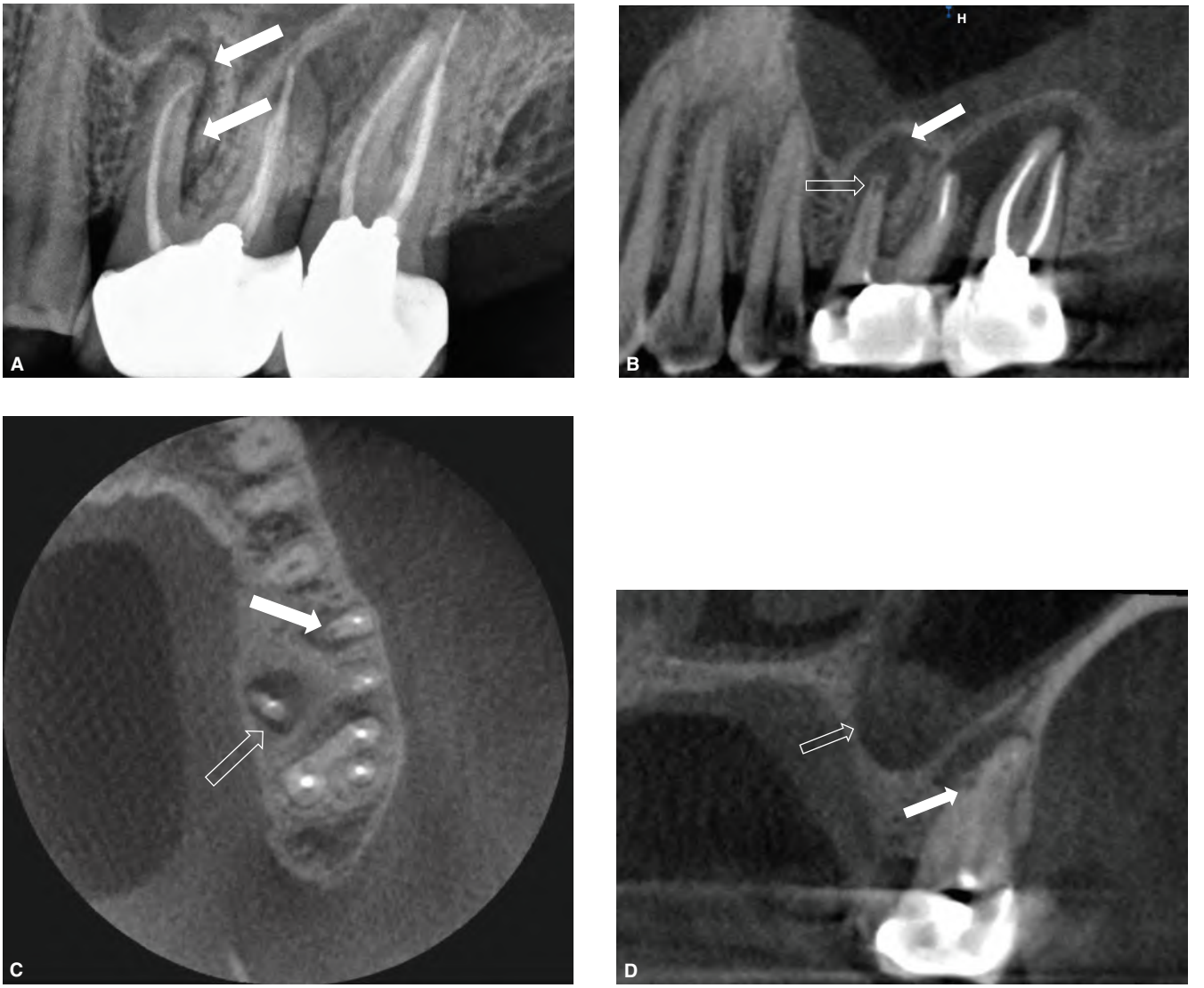


FIGURE 9C-6 Periapical radiographs can be suboptimal in representing complex anatomy compared with CBCT. ***A.*** Periapical radiograph of an endodontically treated maxillary molar is consistent with mesiobuccal root apical periodontitis continuous with a furcal defect (solid arrows). This first molar also evidences PDL widening distal to the distal root. The second molar has features consistent with apical periodontitis of the palatal root and widening of the PDL space anterior to the mesiobuccal root. ***B.*** CBCT sagittal reformation, left maxillary first molar shows features consistent with chronic periradicular abscess at the mesiobuccal root (solid arrow), continuous with a furcation defect. There is an untreated mesioaccessory canal and internal resorptive defect at the periapex not visible on the periapical radiograph (open arrow). The left maxillary second molar has features consistent with a large, periradicular abscess continuous with a furcation defect and periradicular periodontitis along the distal aspect of the distobuccal root extending to the distobuccal apex. The maxillary sinus within the CBCT image has features consistent with chronic sinusitis. ***C.*** CBCT axial reformation, left maxillary first molar has features consistent with a chronic periradicular abscess at the mesiobuccal root and also has an untreated mesioaccessory canal. Note internal resorptive defect at the periapex (solid arrow) and features consistent with chronic periradicular abscess at the palatal root (open arrow). There is a mucositis of the floor of the maxillary sinus overlying both the left maxillary first and second molars, and these teeth show radiographic features consistent with advanced periapical periodontitis along the mesial aspect of the mesiobuccal root and early periradicular periodontitis at the palatal root. ***D.*** CBCT corrected axial reformation shows radiographic features consistent with a chronic periradicular abscess at the palatal aspect of the mesiobuccal root. The subjacent floor of maxillary sinus shows radiographic features consistent with a mucositis (open arrow). There is an internal resorptive defect at the periapex of the mesioaccessory root (solid arrow).

be ordered on a case-by-case basis, predicated on the benefit and risk to the patient of increased radiation dose and cost. The use of tomographic technology in medically complex patients where the harm from apical periodontitis may pose a serious health risk should be considered. Medical conditions such as osteomalacia, common in elderly patients, may be apparent using CBCT assessments but not using conventional radiographics. Cases of osteometabolic diseases may be apparent using CBCT. The apparent rarefactions, thin cortex, hyperdense appearance of trabeculae, porosity in the jaws may be useful.

Conventional radiography has been shown to overestimate healing after endodontic surgery.[26] A recent study has proposed new CBCT-based healing criteria one year after apical surgery.[27] This novel approach presents three healing indices using buccolingual CBCT slicing: the cut root surface, apical area, and cortical plate. The apical area and cortical plate indices showed complete healing in only 34.3% of cases, suggesting that complete healing may require more than a one year follow-up.

PATIENTS WITH NONSPECIFIC OR CONTRADICTORY SIGNS AND SYMPTOMS

Nonlocalized pain or contradictory findings associated with an untreated or previously root-treated tooth or teeth is not uncommon. A major advantage of CBCT is that it can accurately show the true size and location of alterations involving the teeth and supporting structures[12] (Figure 9C-7).

Endodontic treatment may result in persistent pain lasting for more than 6 months, estimated at between 5.4% and 10.0%.[28] This pain may result from odontogenic or

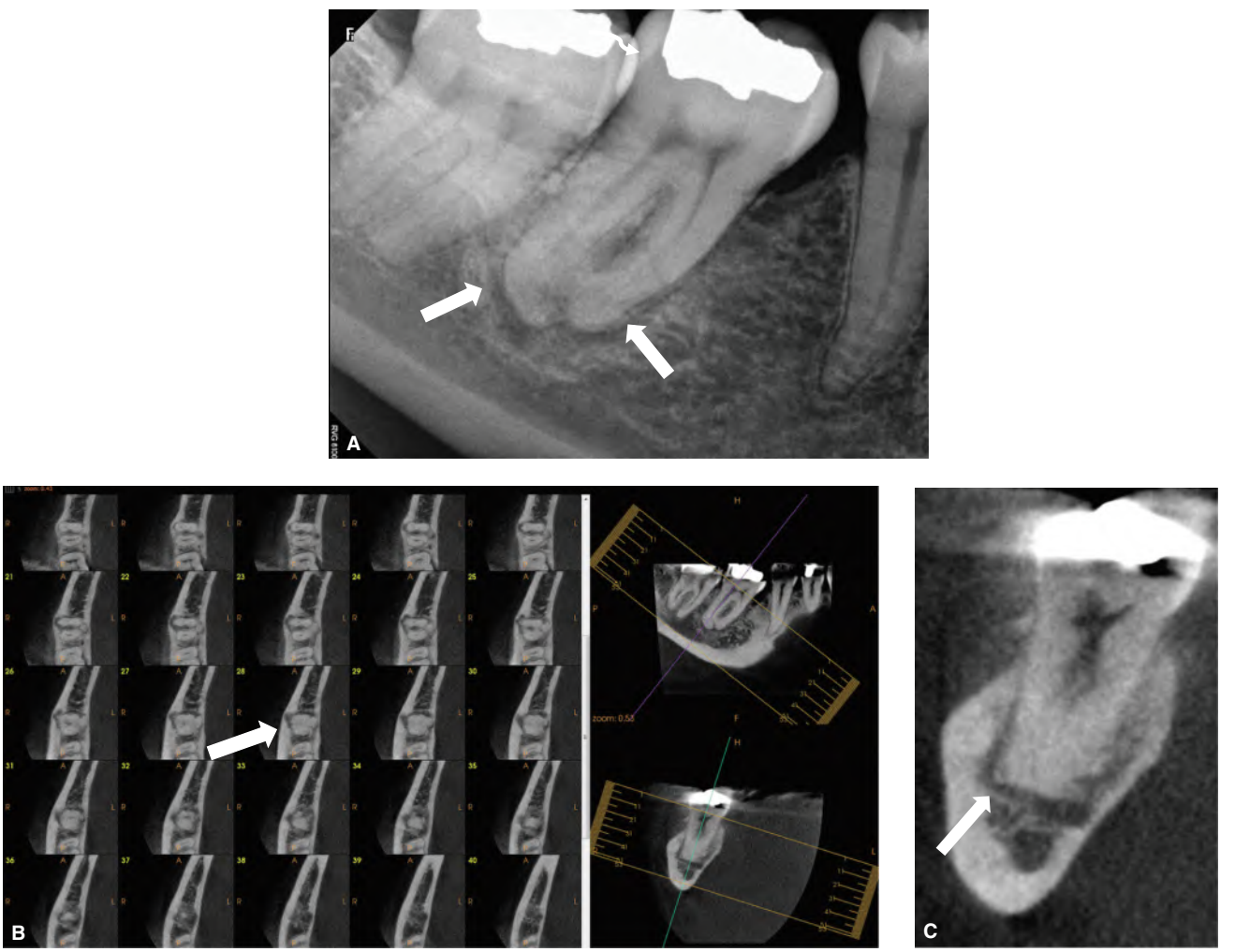


FIGURE 9C-7 ***A.*** Periapical radiograph of this 56-year-old African-American female shows the mandibular right second molar with small circular radiolucent bands surrounded by opaque bands approximately 2 mm wide, centered on a point around the apicies of the mesial and distal roots (solid arrows). The assessment of this lesion was confounded by a pulpal diagnosis of irreversible pulpitis. ***B.*** Correlated and corrected axial reformation showing erosion of the buccal cortex (solid arrow). ***C.*** Cross-sectional reformation of the same region showing multiple periapical areas of different densities and hypercementosis of the distal aspect of the distal root (solid arrow). These features were consistent with an irreversible pulpitis with cemento-osseous dysplasia.

nonodontogenic causes. The pathogenesis of the pain source is critical because treatment and outcomes are different for each individual. In patients who had odontogenic-related pain, such as from missed canals, over-instrumentation or overfilling, periapical radiographs and CBCT showed untoward findings in 57% and 100% of cases, respectively. Patients diagnosed with nonodontogenic pain had normal findings on their radiographs approximately 75% of the time, whereas approximately 25% had periapical radiolucencies. The ability of CBCT to show all of the odontogenic lesions was considered significant in the diagnosis of these patients. Some patients with a painful tooth 6 months after treatment may have nonodontogenic pain, with temporomandibular disorder the most frequent cause.[28]

The pathophysiology of this pain is uncertain, but hypothesized to involve deafferentiation of peripheral sensory neurons in predisposed patients. The diagnosis of atypical odontalgia (AO) or persistent dentoalveolar pain disorder (PDAP) is challenging, and depends on patient history, clinical examination, and the absence of radiographic findings. In some cases, the symptoms from apical periodontitis and AO are closely related. In a study of 20 patients with AO, with at least one tooth in the region of discomfort having had an invasive treatment including 21 of 30 teeth that underwent endodontic treatment, Pigg et al.[29] found that 60% had no periapical lesions and that CBCT showed 17% more periapical lesions than conventional radiography. This study demonstrated that CBCT may be a useful supplement to two-dimensional radiography.[29] In the majority of patients with PDAP, there were no signal changes in the painful region when examined by MRI. There was a high correlation between radiographic and MRI findings.[30] In a study that followed 46 patients previously diagnosed with PDAP over 7 years, approximately one-third showed significant temporal improvement, but for many patients, pain was persistent and treatment-resistant.[31]

COMPLEX ROOT CANAL MORPHOLOGY: ADDITONAL ROOTS, CANALS, ROOT CURVATUES, ANOMALIES, AND PROXIMAL ANATOMY

The success of endodontic treatment requires thorough cleaning and obturation of the root canal system. Endodontic treatment failures have been attributed to the inability to locate all of the root canals present. Anatomic variations are common and show certain morphologic characteristics among ethnic population groups.[32] CBCT has shown results that are comparable to *in vitro* histologic examination and provide images in different representations, such as multiplanar reformations and 3-D surface rendering.[33] Variations in root canal morphology may be categorized into additional roots, additional canals, root curvatures, and anomalies.

Additional Roots, Canals, and Root Curvatures

The operating microscope plays an important role in the intraoperative identification of root canal anatomy and can be supplemented by the use of CBCT when necessary. CBCT and the operating microscope result in an increased detection of canals and appreciation of roots curved in the buccolingual direction.[34] Blattner et al.[35] conducted an *in vitro* investigation comparing physically cut sections of extracted maxillary first and second molar teeth with CBCT assessments for the prevalence of mesio-accessory (MB2) canals. There was a positive correlation (79%) between the two techniques, showing that CBCT is a reliable method to detect MB2 canals. In a sample of 2575 Chinese subjects, CBCT showed the highest incidence of MB2 canals were in the second decade of life, and there was a high likelihood of MB2 canals in the contralateral tooth.

According to Matherne et al.[36] three endodontists evaluated 72 extracted human teeth with digital detectors and photostimulable phosphor plates. The same teeth were evaluated by an oral and maxillofacial radiologist using CBCT. When the two-dimensional images were compared with the CBCT scans, the evaluating endodontists failed to identify at least one root canal in 40% of the teeth examined.

The prevalence of additional roots and additional canals has been reported using various techniques, such as intraoral[37] and three-dimensional radiography, MRI, scanning electron microscopy, tooth sectioning, micro-CT, and magnification. The use of CBCT in combination with the operating microscope showed that the identification and localization of extra canals in maxillary first molars was significantly improved.[34] The ability to identify internal anatomy is affected by canal scarification and the spatial resolution of the CBCT employed. Bauman et al.[7] reported that the detection of the MB2 canal increased from 60.1% at 0.4-mm voxel size to 93.3% at 0.125-mm voxel size. Detection of the MB2 canal was also more accurate when viewed by more experienced observers.

Teeth with severe curves or dilacerations require an instrumentation strategy that will respect the natural form of the root canal.[38] Cyclic fatigue fracture of rotary instruments with the same radius decreases as the length of the arc increases.[39] In a study of 100 mandibular first and second molars, all teeth evaluated had curvatures in both the buccolingual and mesiodistal directions, with the highest incidence of secondary curves occurring in the mesiodistal view.[40] CBCT has been shown to aid in accurate measurement of the radius of root curvature, resulting in more precise instrumentation and a reduction in procedural errors.[41]

Anomalies

CBCT can aid in diagnosing anomalies such as developmental malformations. Taurodontism, for example, is a developmental malformation characterized by an apical extension of the pulp chamber and no constriction at the level of the cemento-enamel junction seen most frequently in multirooted teeth.[42] CBCT was successfully used in several cases

reported in the literature owing to the difficulty in understanding the location and size of the root canals.[43,44]

Dens invaginatus (DI), is a dental anomaly that may result from an infolding of the dental papilla during tooth development[45] in response to external forces on the tooth bud or genetic disorders.[46] DI may affect teeth in both arches, but maxillary lateral incisors, followed by maxillary central incisors are most often affected and bilateralism is not uncommon.[47] There are case reports demonstrating the value of CBCT in understanding the three-dimensional architecture of these teeth and allowing orthograde endodontic treatment with preservation of tooth structure and vital elements of the pulp.[48]

Dens evaginatus (DE), is characterized by the formation of an accessory cusp that contains pulp tissue that may extend up to and including the tubercle's dentin core.[49] Jaya et al.[50] reported that the use of CBCT was useful in understanding the complex anatomy in a rare case of DI coupled with DE in the same maxillary central incisor.

For more information about these anomalies, see Chapters 1 and 26.

Proximal Anatomy

Determining the proximity of neurovascular structures to root apicies, especially in the mandible prior to orthograde and surgical endodontic treatment, may be useful in establishing a margin of safety. Carruth et al.[51] found that the mental foramen is located approximately 2.8 mm inferior and 0.01 mm mesial to the apex of the mandibular second premolar. Kovisto et al.[52] determined the distance from the apicies of mandibular bicuspid and molar teeth to the superior border of the mandibular canal. They reported that the mesial roots of the mandibular second molar were the closest to the superior border of the mandibular canal in female patients compared to male patients. CBCT was an accurate method to localize the relative position of the mandibular canal and should be considered in cases where iatrogenic injury is a risk.[52]

INTRAOPERATIVE

Fractured Endodontic Instruments

Instrument fracture during endodontic treatment is a procedural mishap that may occur at any stage or any location in the root canal. In a graduate endodontic program over a 4 year period, fracture incidence of hand instruments were 0.25% and rotary instruments were 1.68% of cases treated.[53] The apical third of mandibular molar root canals are the most common location of instrument separation.[53] CBCT was successfully used in a case study reported by Ball et al.[44] to determine the space for further ultrasonic preparation, evaluate the proximity of the root apex from the mental foramen, and localize a resorptive defect in the root. CBCT was successfully used to localize a separated file in the middle third of a ribbon-shaped canal of a mandibular lateral incisor that was subsequently bypassed and removed.[54]

Elderly patients show many changes in their dentition, especially partial or nearly complete occlusion of root canal space by secondary and tertiary dentin formation. The United States will experience an expected growth of its aged 65 and over population from approximately 50 to 84 million between 2015 and 2050.[55] Root canals typically calcify coronally first, then show decreasing calcification apically. CBCT has been shown to reveal internal root canal anatomy not shown with conventional imaging projections. During endodontic treatment, using available multiplanar reformations, CBCT has been shown to improve the triangulation of calcified canals with the use of markers, such as instruments or other radiopaque materials, leading to improved access without removal of additional tooth structure.[6] CBCT scans may show radiographically complete calcification of the root canal system with no changes in the periodontal membrane, allowing for the preservation of dentin and elimination of the risks of further treatment.[44]

Assessment of Root Perforations

Root perforations are usually associated with iatrogenic events and account for approximately 10% of all nonhealed cases.[56] Root perforations can be caused by procedures such as a postperforation, interproximal instrumentation error resorption or separated instrument retrieval, and are difficult to assess with conventional imaging because no information is available in the buccolingual direction.[57] An *ex vivo* study of the diagnostic accuracy of CBCT and periapical radiography in the detection of furcal perforations was conducted by Kamburoglu et al.[58] They concluded that actual perforation width was accurately determined with CBCT imaging, and CBCT showed superior detection of perforative defects when compared with periapical radiography. In a case report, CBCT was successfully used intraoperatively to mark the exact location of a perforation and facilitate repair with the appropriate material.[44]

Materials Extending Beyond the Root Canal, Inferior Alveolar Neurovascular Bundle

Endodontic treatment complications, such as overextension of obturation materials, or over-instrumentation in close proximity to the inferior alveolar neurovascular (IAN) bundle, may result in significant morbidity, such as anesthesia, hypoesthesia, paresthesia, and dysesthesia.[59,60] The IAN bundle, consisting of the inferior alveolar vein(s), artery, and nerve,[61] resides within the cribiform bone-lined mandibular canal and courses obliquely through the ramus of the mandible, and horizontally through the mandible body, terminating in the mental and incisive foramina.[62] Numerous case reports have concluded that the mandibular second molar is most frequently involved, but cases involving first molars and bicuspids have been described.[63,64] CBCT allows the mapping of endodontic treatment complications by producing three-dimensional representations of the teeth, supporting structures and vital anatomic features.

Pogrel[64] presented a case series of injury to the IAN and recommended careful monitoring of patients when there is radiographic evidence of sealant expressed into the IAN. If neuropathic symptoms develop after the anesthetic period has elapsed, immediate referral to an oral and maxillofacial surgeon is indicated. Even patients with no immediate untoward symptoms should be monitored for the development of latent symptoms for a period of 72 hours. Surgical decompression and debridement of the nerve should be conducted within 48 hours to ensure the best outcome.[64] Damage to vital structures such as the IAN are well-known adverse events associated with endodontic treatment.[65] A case study of IAN injury, resulting from overextension of sealer into the IAN, treated with nonsurgical management, has also been reported.[66] Gambarini et al.[67] reported that limited FOV CBCT was an effective radiographic modality to assess endodontic-related IAN complications.

Materials Extending Beyond the Root Canal, Maxillary Sinus

Iatrogenic introduction of obturants, irrigants, and instruments into the maxillary sinus has been described in numerous case studies. Overextension of zinc oxide-eugenol containing sealers into the maxillary sinus may promote sinus aspergillosis.[68] Legent et al. evaluated 85 cases of aspergillomas and concluded that 85% of the cases resulted from endodontic treatment.[69] Hodez et al.[70] stated that CBCT is very effective for the assessment of sinusitis of dental origin, but lacks soft tissue discrimination necessary to explore tumoral processes. For more details, see Chapter 16. CBCT was also able to show fine calcifications, associated with aspergillus, around foreign bodies of dental origin.

VERTICAL ROOT FRACTURES

The detection of vertical root fractures is challenging. VRFs are complete or incomplete longitudinal fractures of the root, are usually directed buccolingually, may involve one or both proximal surfaces, are confined to the root, and may originate from the root at any level.[71] For more details, see Chapter 14.

The prevalence of VRF in extracted, root-treated teeth has been reported to range from 8.8 to 20%.[72,73] Teeth with VRFs have an unfavorable prognosis, may cause significant loss of supporting bone, and ultimately result in extraction.[74] VRFs are difficult to accurately diagnose because the clinical and radiographic signs are inconsistent, leading to the possibility of unwarranted extraction.[75] Root-filled mandibular posterior teeth show the highest incidence of fracture.[76] VRFs are difficult to visualize with intraoral radiographs unless the X-ray beam is parallel to the plane of the fracture.[77]

The accuracy of CBCT in the detection of VRF is controversial. In a review and meta-analysis of all *ex vivo* and *in vivo* studies published before November 2013, the use of CBCT in the detection of VRF showed a range of results.[73a] In the detection of VRF in unfilled teeth, *in vitro* studies showed significantly higher sensitivity and specificity using CBCT imaging with a voxel size of 0.2 mm or smaller when compared with periapical radiography. In root-filled teeth, CBCT imaging showed lower pooled sensitivity and specificity when compared with periapical radiographs. The low pooled sensitivity and specificity of CBCT in the assessment of VRF is thought to be related to beam-hardening and streak artifact. CBCT imaging may contribute other benefits to the diagnosis, such as the ability to see the true nature of the fracture, without tissue compression, magnification, and parallax error. Only two *in vivo* studies were considered in this meta-analysis, where CBCT showed high sensitivity and specificity in the detection of VRF, but more study was recommended.

The value of CBCT in the detection of VRF has been reported in *ex vivo* and clinical studies. VRF in root-filled teeth reduced the sensitivity and accuracy of periapical radiographs whereas it only reduced the specificity of CBCT.[78] In another *ex vivo* study, Brady et al.[74] concluded that the detection of simulated incomplete VRF in non-root-treated teeth was unreliable with either CBCT or periapical radiographs. Accuracy varied between the two CBCT scanners tested; however, both CBCT scanners showed better results than intraoral radiographs when the size of the VRF was ≥50 μm compared with VRFs of <50 μm. *Ex vivo* studies may show higher accuracy than clinical investigations, because patient-derived movement artifact is eliminated.[81,82] Additional study[83] showed that the results, determined for vertical root detection, were significantly different between different CBCT systems with the iCAT (ISI, Hatfield, PA), outperforming the other systems tested. The Galileos (Sirona, Bensheim, Germany) performed poorly, the NewTom 3G (VR Imaging, Verona, Italy) and Accuitomo xyz (J Morita Corporation, Osaka, Japan) provided fair results, the Scanora 3D (Soredex) moderate results, and the iCAT Next Generation (ISI, Hatfield, PA) provided good results. VRFs were better detected in molars than in premolars.

Successful detection of VRF requires high spatial resolution and benefits from the use of the axial view.[84] CBCT images suffer from artifacts generated by highly attenuating obturation materials and posts, resulting in reduced specificity.[85] Patel et al.[86] examined extracted teeth with synthetically generated incomplete fractures using a light microscope and optical coherence tomography. They reported that the maximum width of fractures ranged from 30 to 100 μm. The detection of VRF with all CBCT systems is influenced by the spatial resolution and contrast-to-noise ratio of the selected FOV,[82] size of the fracture, number of basis projections, technique factors, reconstruction algorithms, detector sensitivity, and imaging artifacts. Smaller voxel size and the application of sharpening high-pass filters improved sensitivity without affecting specificity for the detection of transverse root fractures.[87]

Numerous case series have been published to evaluate the usefulness of CBCT in the detection of VRF. While the resolution of 2-D digital detectors and CBCT detectors ranges from 16 to 25 lp/mm and 6 to 8 lp/mm, respectively, CBCT has been shown to have higher sensitivity and specificity where the detection of VRF was validated by surgery. Bias in these studies is difficult to control, however, because observations are

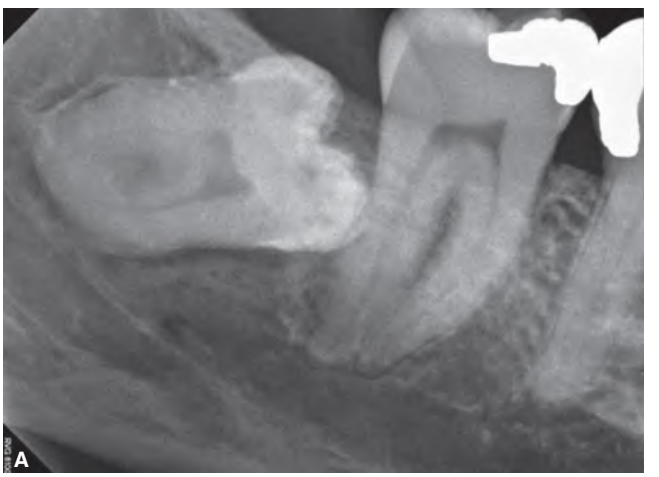
influenced by the presence of alterations in the tooth-supporting tissues and the selection of patients evaluated.[88]

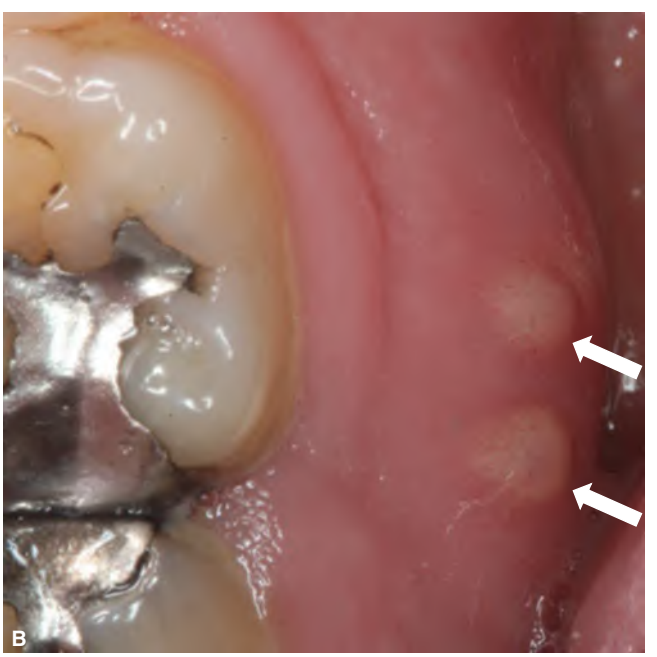
ROOT RESORPTION

Root resorption is a physiologic process that results in exfoliation of a primary tooth or a pathologic process that is usually associated with mechanical, thermal, or chemical injury to the supporting structures, cementum, or dentin.[89] For more details, see Chapter 15. This resorption may be inconsequential[90] or cause significant damage to the tooth and supporting structures if the resorptive process continues. Because of the inconsistent clinical presentation of resorptive lesions, detection of this condition is predicated on the appearance of radiographic alterations. The pathogenesis of external root resorption (ERR) and internal root resorption (IRR) are different, and the treatment protocols vary. However, several studies have reported that intraoral radiography may not show the full extent of resorptive lesions.[91] In an *ex vivo* study, Durack et al.[92] reported that the accuracy of CBCT was the same for 180° and 360° gantry rotation settings. The 180° scans resulted in a significant reduction in the x-radiation dose to the patient.

External Root Resorption

The use of serial cross-sectional CBCT to show the true size and location of ERR has been well documented[93] (Figure 9C-8). Additional *ex vivo* studies have shown that





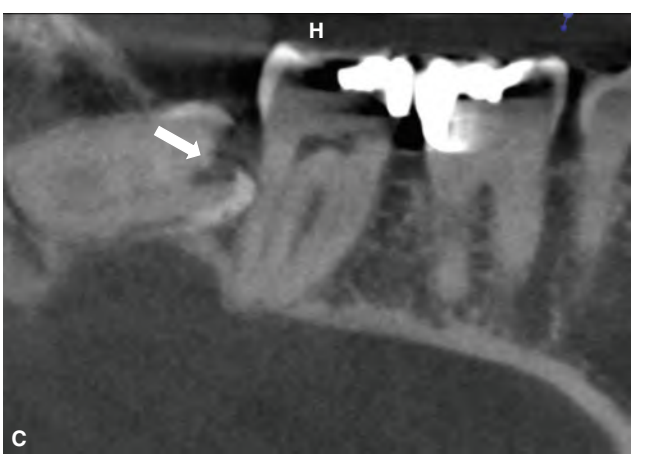


FIGURE 9C-8 Planar imaging is limited by superimposition of anatomic features. ***A.*** Initial periapical image of the mandibular right second molar and bony impacted-third molar shows no pathologic findings. ***B.*** A photograph of two nonproductive sinus tracts on the lingual of the mandibular right second molar that suggest a lesion in the supporting bone (solid arrows). ***C.*** CBCT of the same region shows a wedge-shaped resorptive lesion in the crown of the third molar (solid arrow) with erosion of the lingual aspect of the alveolar crest on the distal and mesial aspects of the mandibular second molar; features consistent with external resorption.

CBCT was more accurate than conventional imaging in the detection of simulated surface defects[94] and postorthodontic resorption in roots of lateral incisors as a result of impacted maxillary cuspids.[95] Patel et al.[96] compared the accuracy of intraoral radiography with CBCT in an *in vivo* study of 15 teeth. They found that CBCT showed a significantly higher prevalence for selection of the correct treatment option when measured against intraoral radiography. There was higher inter-examiner and intra-examiner agreement using CBCT, suggesting that less superimposition of structures and the ability to visualize the region in any plane assisted in the correct assessment. An *ex vivo* volumetric analysis has demonstrated improved quantification with high-resolution CBCT scans when compared with low resolutions scans and periapical radiography.[97] In an *ex vivo* comparison of CBCT and intraoral radiographs, Bernardes et al.[98] showed that CBCT provided significantly better detection values regardless of the simulated lesion size and tooth evaluated.

Invasive cervical resorption is a less common form of external root resorption.[99,100] CBCT can be relied upon to reveal the true extent of the lesion, allow for the identification of the portal of entry and to ensure that an appropriate treatment plan has considered[6] (Figure 9C-9).

Internal Root Resorption

Clinically detectible IRR is considered a rare occurrence. It is usually asymptomatic and appears as a round, radiolucent

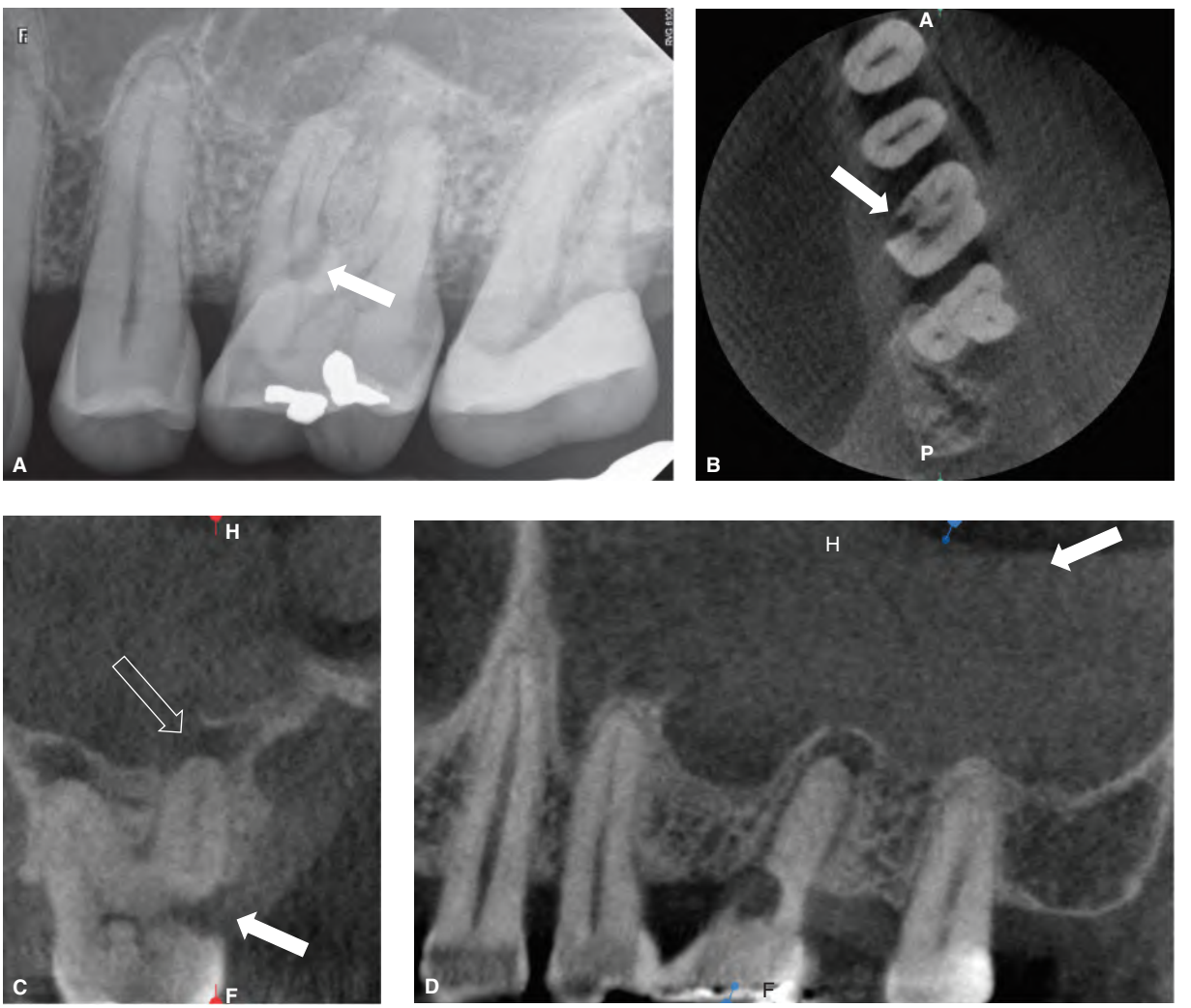


FIGURE 9C-9 CBCT has been shown to be more accurate than periapical radiography in the detection of invasive cervical resorption. ***A.*** Periapical radiograph shows the maxillary left first molar with an irregular region of medium attenuation at the junction of the mesiobuccal and palatal roots (solid arrow), rarefying osteitis at the periapicies of the of the mesiobuccal, distobuccal, and palatal roots and lifting the sinus floor. ***B.*** Axial reformation shows the true extent of an irregular region of low attenuation (solid arrow), a feature consistent with invasive cervical resorption. ***C.*** Cross-sectional reformation shows a wedge-shaped region of low attenuation of the palatal root (solid arrow), large pulp stone, rarefying osteitis of the mesiobuccal and palatal roots with erosion of the sinus floor (outline arrow) and mucositis of the portion of the sinus imaged. ***D.*** Panoramic reformation of the palatal root with osteoperiostitis and mucositis (solid arrow).

widening of the root canal space.[101] Several studies show that small resorptive lesions detected with scanning electron microscopy may be a common finding in teeth with pulpal inflammation or necrosis, but may not be radiographically visible.[102,103] CBCT has been shown to provide high sensitivity and specificity when serial cross-sectional views of external resorption were viewed.[104] A recent case study showing the assessment and subsequent repair of a massive internal resorptive lesion relied on CBCT to show the full extent of the lesion.[105] CBCT allows precise mapping of IRR lesions and allows for authoritative differentiation between IRR and ERR.[96]

DENTAL AND MAXILLOFACIAL TRAUMA

Approximately 5% of all injuries involve the oral region. Crown fractures and luxation injuries occur most frequently, affecting the maxillary central incisors in 80% of cases.[106] The prevalence of maxillofacial trauma and sequelae are generally underestimated. Systematic studies have suggested that facial injuries may frequently result in dental trauma.[107,108]

Root fractures occur in approximately 7% of traumatic dental injuries,[109] but are difficult to confidently diagnose and manage with conventional radiography alone[110] (Figure 9C-10). While intraoral radiography is essential to help identify the nature of the traumatic injury to the teeth, such as exposure of the pulp and stage of root development, these conventional projections may not show horizontal root fractures (HRFs), alveolar fractures and tooth displacement. Fractures in the apical, middle, and coronal thirds and the extent of dislocation of the coronal fragment are important in treatment planning. According to the International Association of Dental Traumatology's guidelines the exact nature of a HRF is difficult to assess with conventional imaging, even when several horizontal and lateral projections angles and an occlusal radiograph are acquired. Multiple intraoral projections and the discomfort to a child undergoing an intraoral radiographic examination in emergent conditions may favor a single periapical image and limited FOV CBCT examination. Limited FOV CBCT image volumes showed higher sensitivity, negative predictive value, and diagnostic accuracy in the detection of vertical fractures than multidetector helical CT at a slice thickness of 0.63 mm and intraoral radiographs.[111] Kamburoğlu et al.[112] compared the diagnostic accuracy of conventional radiographic images and CBCT in 36 teeth with simulated HRF. The sensitivity of CBCT was significantly higher than conventional imaging techniques. Bernardes et al.[113] compared conventional imaging with CBCT to detect HRFs in 20 patients. They found that CBCT was able to detect fractures in 90% of patients whereas periapical images could only detect fractures in 30% to 40% of cases.

PRESURGICAL TREATMENT PLANNING

Endodontic surgery is sometimes necessary if nonsurgical procedures are unsuccessful. For more details, see Chapter 24. CBCT provides undistorted multiplanar representations of the roots, supporting structures, and important anatomic features, allowing precise localization of pathologic alterations.[114] Individual roots can be assessed and the location and true size of endodontic pathoses can be determined. The value of CBCT in optimizing surgical procedures was first described by Rigolone et al.[115] in the planning for apical surgery on the palatal roots of maxillary first molars. They suggested that the distance between the palatal root and the vestibular cortex could be measured, possibly allowing trans-alveolar access instead of the more difficult palatal surgical approach. Using CBCT for successful localization of lesions and separated instruments prior to endodontic surgery have been reported by others.[116] Low et al.[12] compared intraoral radiographs with CBCT for the assessment of 37 maxillary bicuspids and 37 maxillary molars prior to apical surgery. They concluded that CBCT identified 34% more lesions than conventional radiography and that additional findings, such as sinus membrane thickening and missed canals, were only visible on CBCT image volumes. Missed canals, additional roots, dilacerations, and the true size of a periapical lesion may be visible with CBCT.[6]

Anterior teeth are not exempt from consideration of their proximity to important anatomic structures. Taschieri et al.[117] evaluated 57 maxillary central and lateral incisors with CBCT imaging. The maxillary central incisor measured 4.71 ± 1.26 mm from the anterior wall of the nasopalatine duct at 4 mm from the apex and 9.5 mm from the apex to the floor of the nasal cavity. CBCT has been shown to be useful in the pre-surgical assessment of the region of interest.

Studies support the use of CBCT in the treatment planning process. Ee et al.[118] examined preoperative intraoral radiographs and CBCT image volumes in 30 endodontic cases and concluded that 62% of the treatment plans were modified based on information provided by the volumetric images. The distance between the apex of mandibular molars and the roof of the mandibular neurovascular bundle could be determined in only 35.4% of cases studied.[119] CBCT can allow assessment of the full extent of lesions that are underrepresented by approximately 10%[120] and comorbidities in the maxillary sinus prior to surgery.[121]

MAXILLARY SINUSITIS OF ENDODONTIC ORIGIN

Maxillary sinusitis can result in dental pain because of shared innervation and the proximity of the antrum to the roots of the maxillary posterior teeth. Conversely, inflammation or infection of the maxillary posterior teeth can cause

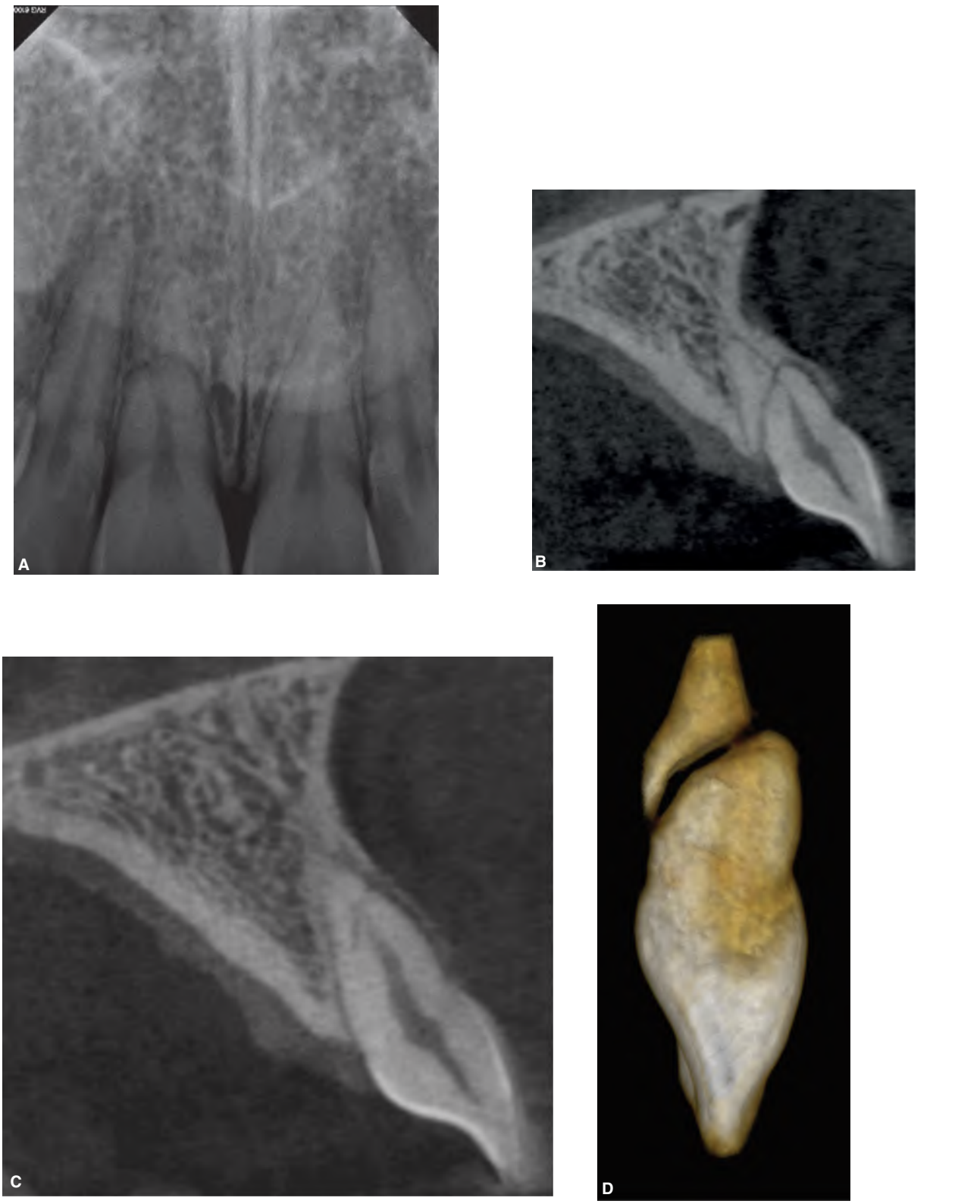


FIGURE 9C-10 The sensitivity of CBCT is significantly higher than conventional imaging techniques in the detection of horizontal root fractures. ***A.*** Periapical radiograph showing a horizontal fracture in the middle third of the maxillary right central incisor with dislocated apical fragment. ***B.*** Sagittal reformation of the same tooth showing the true nature of the dislocation of the apical third of the root. ***C.*** Sagittal reformation of the maxillary left central incisor showing nondislocated oblique fracture of the apical third not clearly visible with periapical radiograph. ***D.*** Segmented 3-D CBCT reformation of the maxillary right central incisor.

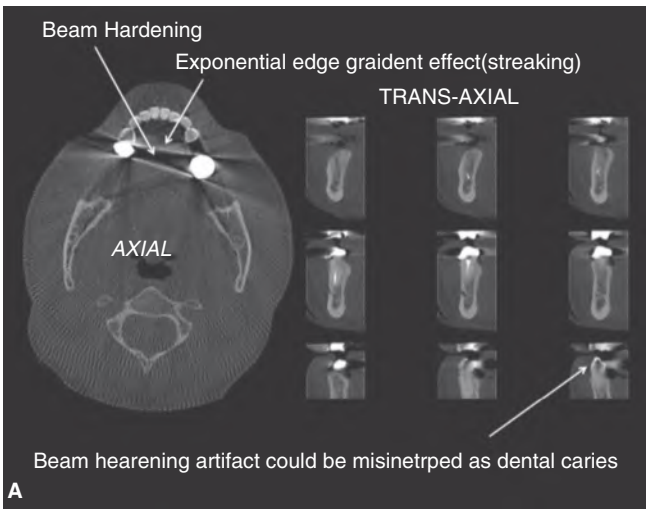
inflammatory changes in the sinus, possibly leading to sinusitis. For more details, see Chapter 16.

Maxillary sinusitis of dental origin may account for 10 to 12% of sinusopathy.[122] Teeth closest to the maxillary sinus have the highest incidence of maxillary sinusitis.[123] Numerous cadaver and clinical studies have suggested the importance of tomographic imaging, early intervention, and management in the event of maxillary sinusitis secondary to odontogenic causes. Nair et al.[124] reported a case series that showed lesions of endodontic origin with confounding clinical findings. CBCT showed involvement of the maxillary sinus, aiding with a presumptive diagnosis. They stated that an otolaryngology consultation may be indicated in these instances. In a case series, Cymerman et al.[125] reported that CBCT was useful in showing the etiology, extent of the lesions studied and aided in clinical management. In one of the cases, bronchitis of 1-year duration resulted from sinusopathy caused by a dental infection. Peri-implatitis is another possible cause of sinusopathy, and should be considered in the differential diagnosis, when indicated.[126] In a meta-analysis reported by Arias-Irimia et al.[127] maxillary first molars were the most frequently implicated. In a retrospective analysis of 143 patients with at least one maxillary posterior tooth showing a periapical area of low density, Nunes et al.[128] found that 64.3% of maxillary sinuses showed abnormalities in the presence of periapical areas of low density, without clinical correlation. Limited FOV CBCT should be considered as an adjunct to periapical imaging in cases where sinusitis of endodontic origin is suspected.

ARTIFACTS IN CBCT

CBCT is a valuable adjunct to endodontic practice; however, artifacts should be understood in order to reduce confusion that might otherwise lead to misdiagnosis. As with any imaging modality there is need to consider the possible artifacts that may either obscure real disease changes, or be mistaken for disease when no lesion is actually present. Understanding artifacts is important when selecting CBCT as an imaging method, in modifying exposure parameters or patient position, and in making correct assessments of the acquired image volume. An excellent review of CBCT artifacts in the maxillofacial region was made by Schulze et al.[129] Artifacts in CBCT include beam hardening, photon starvation, streaking through scatter, undersampling, ring artifact, aliasing, quantum noise, cone beam artifact, and motion.

Beam hardening artifact is produced by increased mean beam energy caused through absorption of low energy photons due to the presence of substances of high radio-density such as metallic dental coronal restorations, radiopaque endodontic filling materials, surgical plates, and dental implants. An extreme example of this is photon starvation with complete absorption of all photons along certain paths. Such features appear as dark areas or bands and can be accompanied by white streaks (streaking) from the exponential edge gradient effect due to scatter with X-ray deflection accompanied by faulty projection data. These phenomena can be readily interpreted in the axial view (Figure 9C-11A). However, in the last of the series of transaxial views, the premolar tooth has a dark area over the crown due to the beam that might easily be misinterpreted as dental caries. Narrow bands of beam hardening due to endodontic restorations could be misinterpreted as dental root fractures. A true vertical root fracture is compared to beam hardening artifact from endodontic fillings in Figure 9C-11B. Beam hardening and the exponential



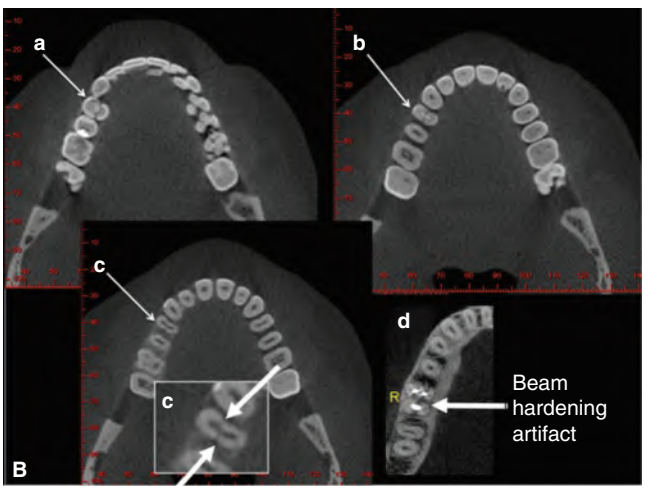


FIGURE 9C-11 ***A.*** Beam hardening and exponential gradient effect due to metallic restorations. Beam hardening artifact can be mistaken for dental caries when it projects over the crown of a tooth or teeth. When there are multiple metallic restorations, particularly in both jaws, the problem can be alleviated to some extent by having the head tilted at an angle to reduce superimposition of the restorations within the beam projection. ***B.*** Axial views of the same premolar tooth with a vertical fracture (a–c). Beam hardening artifact in an endodontically-treated molar tooth that does not have a root fracture (d).

gradient effect can create confusion (Figure 9C-12A). The initial axial view demonstrates that there are no teeth in the maxilla between the left central incisor and second premolar; however the transaxial view made at slice a-b appears to show an endodontically treated tooth in that region. Indeed, this apparent tooth was found throughout the gap between the left central. The apparent endodontic filling was streaking artifact and the apparent tooth dentin was a bite block positioned to stabilize the jaws during imaging (Figure 9C-12B).

Ring artifact (Figure 9C-13) is caused by the circular trajectory in CBCT and bad pixels at the periphery of the scan. The ROI should be kept central within the FOV rather than the extreme edge of the detector. Moire pattern (aliasing) is caused by undersampling often in a misguided attempt to excessively reduce dose and thereby diminish image quality to below diagnostic utility (Figure 9C-14). Overly minimizing dose by reduced milliamperage or kilovoltage can also lead to quantum noise and grainy and undiagnostic image

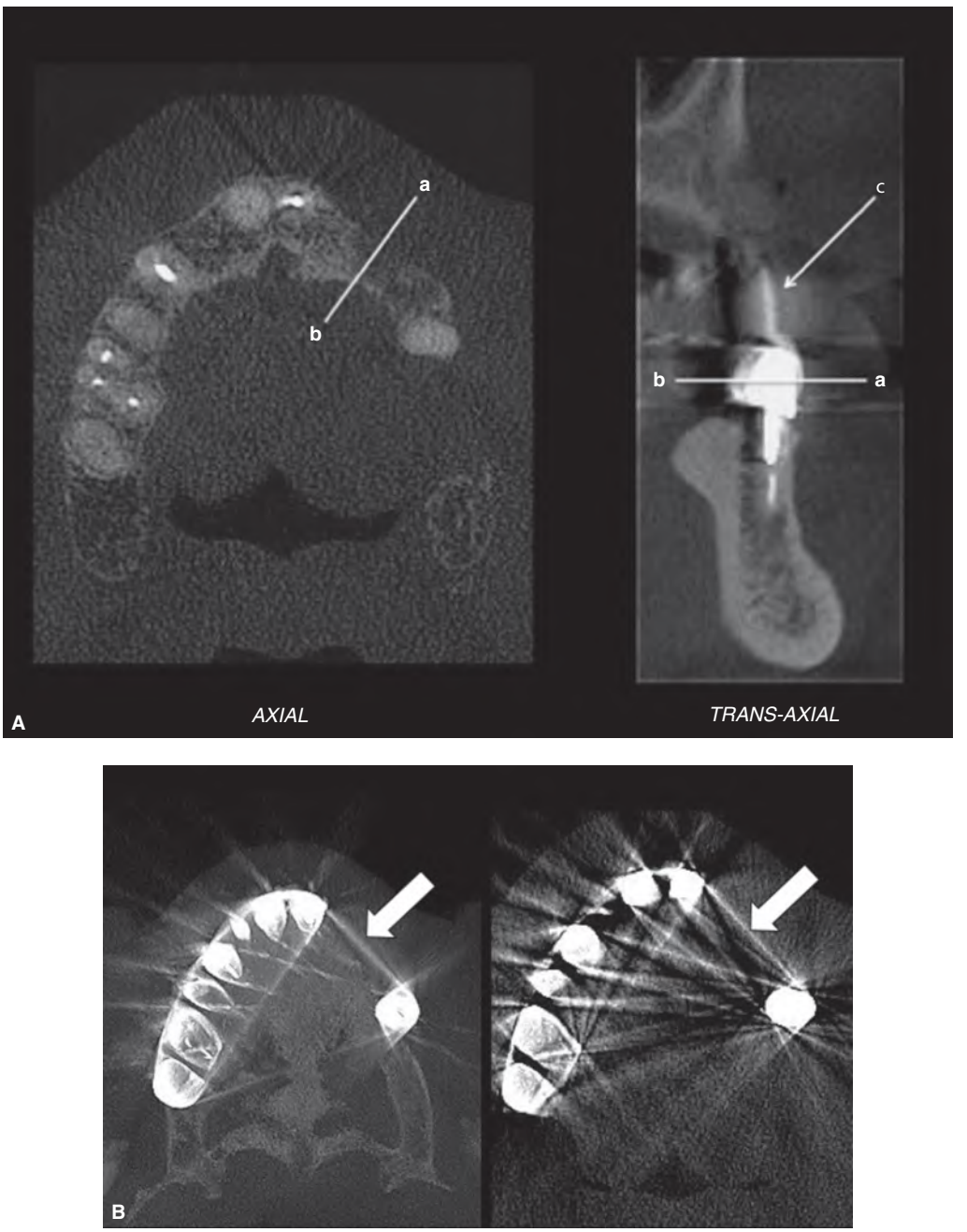


FIGURE 9C-12 ***A.*** The axial view demonstrated that the maxilla is edentulous between the endodontically treated left central incisor and the left second premolar. However, the transaxial view made from (a) to (b) shows what appears to be an endodontically treated tooth in this site in the maxilla. Transaxial and axial views made at different levels reveal the apparent endodontic filling in the figure to be due to streak artifact from exponential edge gradient effect. ***B.*** The apparent tooth dentin is a prop placed to stabilize the jaws during imaging.

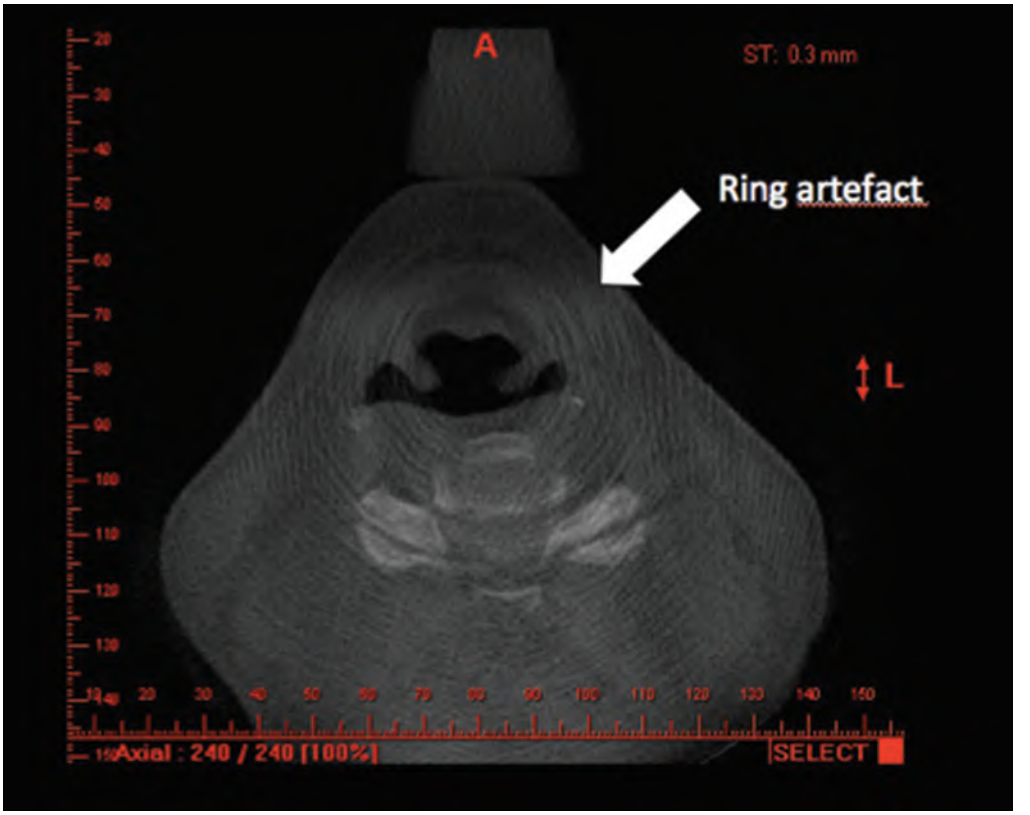


FIGURE 9C-13 Axial view showing ring artifact due to circular trajectory and bad pixels at the periphery of the detector.

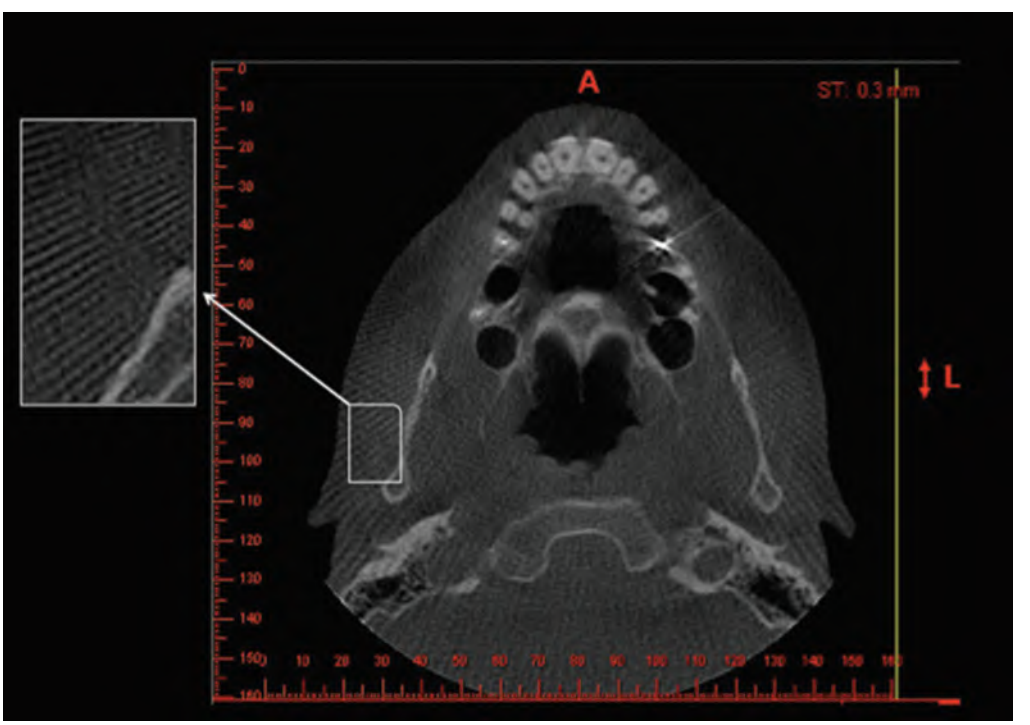


FIGURE 9C-14 Axial view: Moire ("cartwheel") pattern due to undersampling causing aliasing.

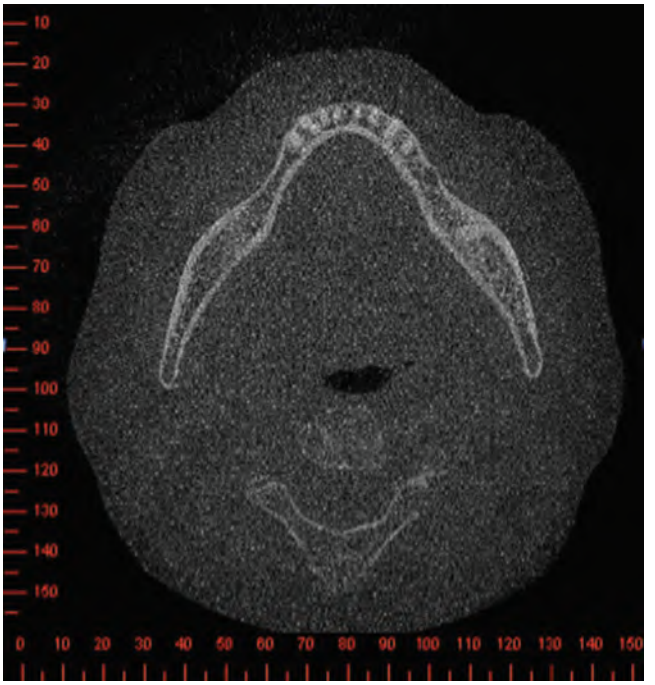


FIGURE 9C-15 Axial view: Quantum noise due to low exposure producing a grainy image suboptimal in diagnostic quality.

(Figure 9C-15). The diverging beam in CBCT and scattering of radiation mean that CBCT is not entirely consistent in pixel gray scale value across the image volume, hence unlike multislice CT, voxel Hounsfield Number determination should not be counted upon to be precise.

As with other imaging techniques, it is imperative that the patient remains motionless during CBCT exposure. The selected voxel resolution will not be realized in the final image volume should the patient move. For this reason, patient stabilization within the cephalostat is a must. It is recommended that patients close their eyes during the exposure to prevent movement while following the trajectory of the generator and detector.

SUMMARY

Intraoral and panoramic radiography delivers a cost-effective, high-resolution imaging modality that may be augmented by CBCT when necessary. CBCT has been shown to improve endodontic assessments by providing undistorted representations of the dentition and maxillofacial structures without compression of anatomical features. CBCT is an important, task specific imaging modality for the comprehensive treatment of endodontic disease.

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Chapter 9D

Magnetic Resonance Imaging

Hatice Dogan-Buzoglu

Magnetic resonance imaging (MRI) is a well-established, noninvasive technique to create high-resolution images of internal structures of the body using strong magnets. It is accepted as one of the best imaging technique for soft tissues and vessels rather than hard tissues.[1–3]

OPERATING PRINCIPLE OF MRI

MRI utilizes nonionizing electromagnetic radiation to produce high quality cross-sectional images of the body, depending on the behavior of hydrogen protons inside the magnetic field. Normally, the patient's hydrogen protons spin on their axis. When the patient is placed into a strong magnetic field, the nuclei of many atoms in the body, including hydrogen, align with the effect of the magnetic field. A radiofrequency (RF) signal that is sent toward the body structures knocks the hydrogen protons and systematically alters the alignment of this magnetization moving from a longitudinal to a transverse plane. This produces a rotating magnetic field from the body that is detectable by a scanner ultimately creating the magnetic resonance (MR) image by a computer.

MRI is based on the natural magnetism of the human body. The strength of this magnetism depends on the proton density that is dependent on the number and type of hydrogen atoms in the tissue fluids and fats. Loosely bound hydrogen atoms, such as those present in soft tissues and liquids, can align themselves with the external magnetic field and produce a detectable signal. On the other hand, tightly bound hydrogen atoms of hard dental tissues fail to produce a usable signal as they cannot align themselves.[3–6]

As soon as the RF pulse is turned off, the return of the hydrogen protons to their original direction (longitudinal) occur at a lower energy. This process is called *relaxation*. There are two relaxation types: *spin-lattice* and *spin-spin*. Spin-lattice controls the re-growth of the magnetization along the *z*-axis and is known as the *longitudinal proton relaxation time* or *T1-weighted sequence*. A tissue with long T_1 produces a low intensity signal and appears dark in MR image. T_1 weighted images are also called *fat image* because fat has the shortest T_1 relaxation time and appears bright in the image. It shows the high anatomic detail due to excellent image contrast.

Spin-spin controls the loss of transverse magnetization in the *xy*- plane, and is known as *transverse proton relaxation time* or the *T2-weighted sequence*. It is also termed *pathology scans* or *water image* since collection of abnormal fluid in pathological tissues gives similar signal sequences like in water that has the longest T_2 relaxation time with bright image, as opposed to darker normal tissues with short T_2 sequences. These images are useful for detecting infection, hemorrhage, and neoplasm. In practice, images often must be acquired with both T1 and T2 weighting to separate the several tissues by contrast resolution.[5–7]

Most MRI machines are graded based on the strength of the magnet, measured in Tesla units (T); equivalent to 10,000 times the magnetic field strength of the Earth. MRI with scanners with the power of 0.5 to 4 T have been used in clinical practice. Higher T produces higher MR signal rates and offers advanced neuroimaging with suitable software. Although magnets as high as 11, 4 T were used in vitro for high quality image resolution, a safe magnet value is limited to 4 T in vivo.[4,8]

MRI IN DENTISTRY

In dentistry, alternative imaging techniques have been suggested as diagnostic aids and to overcome the limitations of intraoral radiographs such as transfer of three-dimensional anatomy into a two-dimensional image, geometric distortion, and anatomical noise. MR imaging attracted attention for potential use in dentistry during the 1980s, after first being used as a tool for medical diagnosis in humans in 1977 (Figure 9D-1).[8–10]

MRI is generally used in dentistry to diagnose disorders of the temporomandibular joint, inflammatory conditions of the facial skeleton, salivary glands, maxillary sinuses, and early bone changes such as neoplasms, osteomyelitis, fractures, and changes of mouth floor and tongue. MRI has an essential role in determining the extent of soft-tissue invasion of maxillofacial tumors. Some cases of squamous cell carcinoma of the tongue can only be visualized by MRI. It provides greater specificity than CT in distinguishing between odontogenic cysts and tumors by examination of signal characteristics.[11,12]

The potential use of MRI in dentistry has increased with the development of methods that allow imaging of hard dental tissues, such as enamel, dentin, cementum, and bone. The thickness of cortical bone, extent of periapical lesions, number of roots in multirooted teeth as well as smaller branches of the neurovascular bundle entering apical foramina can be successfully detected. It can also be useful for periapical surgery, dental implant planning, and periodontal structures. Furthermore, some researchers have successfully studied caries lesions and pulp vitality as well as pulp anatomy.[13–18]

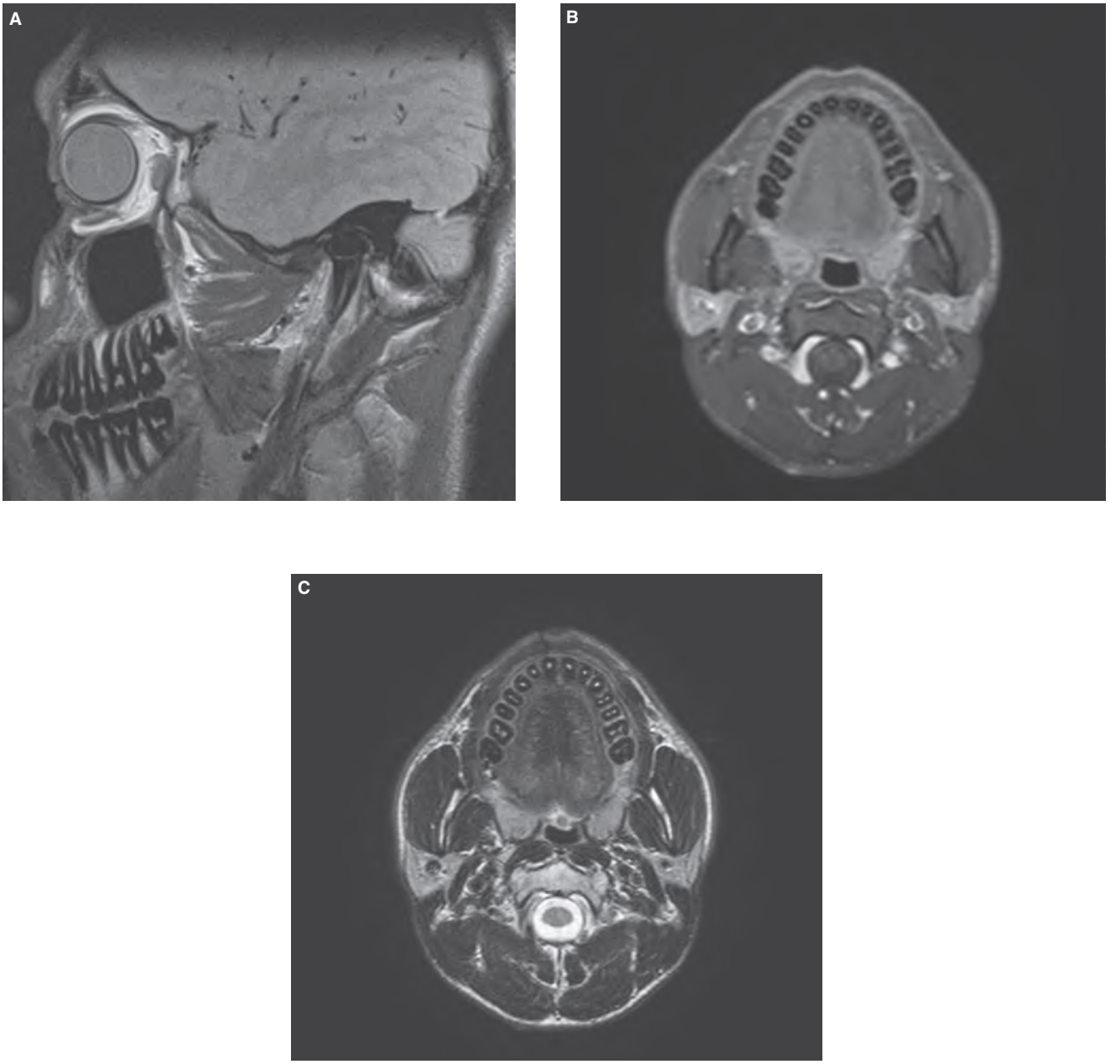

FIGURE 9D-1 MRI scan of teeth and related tissues using head and neck coil with 3-T magnet. ***A***. Sagittal MR imaging of orofacial area. Pulp tissues produce high MRI signal with brilliant image. ***B***. T_1-weighted axial MRI scan. ***C***. T_2-weighted axial MRI scan. (Courtesy of Dr. Burce Özgen Mocan, Ankara, Turkey.)

USE OF MRI IN ENDODONTICS

Soft dental tissues have higher water content and much longer T_2 relaxation times. Therefore, MRI imaging of these tissues by standard MRI techniques, such as 2D or 3D gradient-echo is possible. Spin-echo offers echo times in the range of a few milliseconds and gradient-echo offers echo times as short as 1 ms. Both pulse sequence methods are considered as the "liquid" imaging. In the literature, detailed-MRI images of dental pulp anatomy were accurately demonstrated in vitro and in vivo by additional volumetric image program or high magnetic field.[1,11,19] Moreover, comparison of signal intensities before and after contrast agent administration showed a significant difference, with high sensitivity, between vital and nonvital teeth, depending on age.[16,20]

There are different clinically relevant MRI methods for obtaining images of densely calcified dental tissues. Dentin comprises up to 70% mineral hydroxyapatite, about 20%

organic material and only 10% water. In enamel, the mineral content can even get as high as 96%. Due to the high mineral content, the lack of free hydrogen protons causes weak magnetization. This results in extremely low T2 relaxation time and a scarcely detectable signal. In order to obtain images, it is important to reduce the echo, or detection time, to microseconds.[13,21,22]

Projection reconstruction, single point imaging or its improved version, SPRITE, with moderate spatial 3D resolution, turbo-spin echo (TSE), ultra-short echo time (UTE), zero-echo time (ZTE), FID-projection imagines and stray-field imaging (STRAFI) are useful for obtaining MR images of hard dental tissues. STRAFI produces suitable images by high resolution when the T2 time is very short, but is less appropriate when T2 time is long, as is typical for soft tissues.[22–26] Recently UTE and ZTE MR imaging were successfully used to detect lesions in enamel, dentin, and pulp tissue (Figure 9D-2).[26]

A new technology named SWIFT (sweep imaging with Fourier transform) has also been used in hard dental tissues. This system can visualize tissues in vitro using a 9.4 T, ranging from 100 seconds to 25 minutes scanning time with 100 µm resolution. The same technique can be used in vivo for imaging tissues with 4T, 10 min scanning time and 400 µm resolutions. It can simultaneously image both hard and soft dental tissues with high resolution in relatively short scanning times and thereby be useful to determine the extent of carious lesions and the status of the pulp tissue (Figure 9D-3).[8] Similar results were observed when pulp tissue response to caries was assessed by diffusion-weighted imaging technique that determines the apparent diffusion coefficient (ADC) of water in the tissue (Figure 9D-4). However, long scanning time necessary for this method prevents its use under in vivo conditions.[27] Present clinical diagnostic modalities do not provide accurate information on pulp inflammation as related to caries, except for visualizing the formation of reparative dentin against the insult. Therefore, additional techniques, using MRI, should be assessed as potential markers to accurately diagnose the pulp response.

In caries lesions, properties of hard dental tissues are significantly altered. Increase of porosity and water concentration due to a demineralization process in the carious lesions prolongs T_2 relaxation time of dentin. This makes caries detection possible using even standard MRI techniques, such as T_1-weighted MRI. Caries lesion is observed as brightly radiopaque-like pulp and periodontal ligament in MR imagines.[8,11,27]

Bone tissue fails to produce adequate MRI signal. However, bone marrow emits fat tissue signals. Therefore, MRI is the diagnostic method of choice in cases with an infection such as periapical abcess and osteomyelitis that spreads to the soft and bone tissues. It can detect cancellous bone marrow abnormality in acute osteomyelitis as well as the disruption of cortical bone in periapical lesions. The principal MR examination technique used for fat suppression is the short inversion time inversion recovery (STIR).

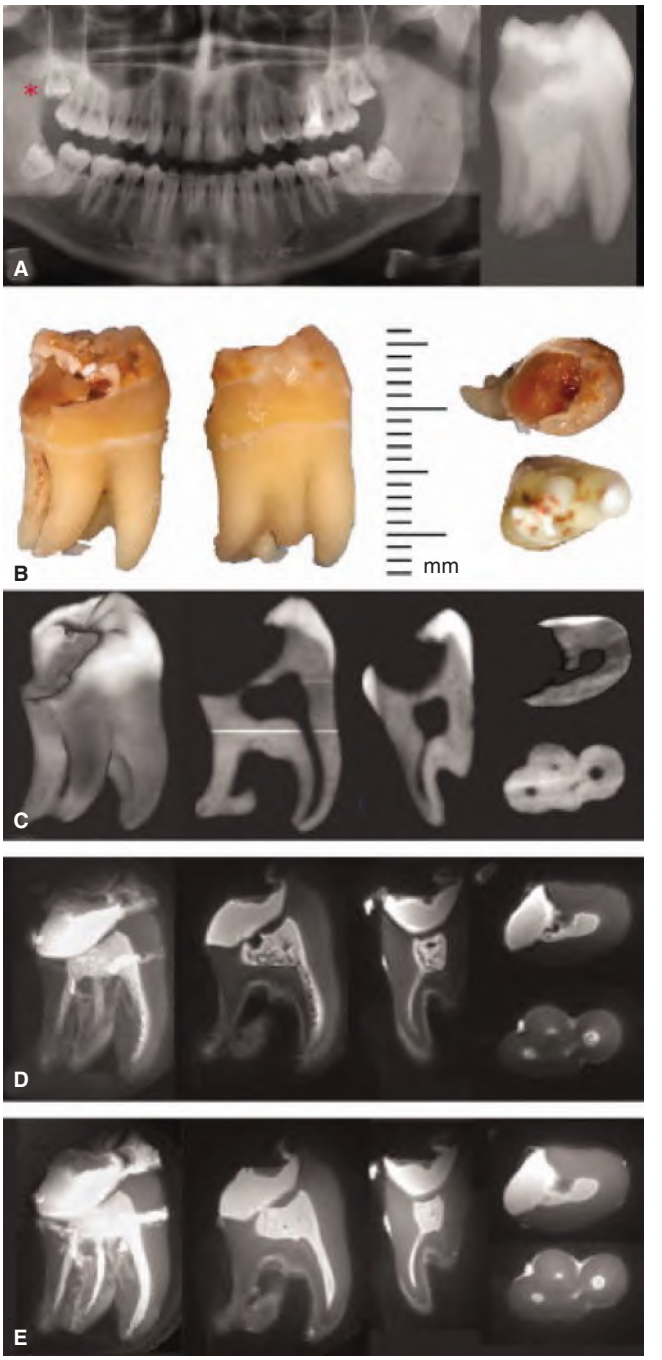

FIGURE 9D-2 Comparison of imaging modalities. MR images were obtained by using 7T and 9,4 T devices. ***A***. In vivo and ex vivo radiographs; ***B***. photography; ***C***. 3D cone-beam computed tomography (CBCT); ***D***. 3D UTE MRI; ***E***. 3D ZTE MRI of carious molar. (Adapted from Hövener JB, Zwick S, Leupold J, et al. Dental MRI: imaging of soft and solid components without ionizing radiation. *J Magn Reson Imaging* 2012;36: 841-846. Used with permission.)

High signal intensities on STIR images with reduced T1 and increased T2 signal, indicate a decrease in fat component and an increase in the water component, reflecting the edema in bone marrow.[14,15]

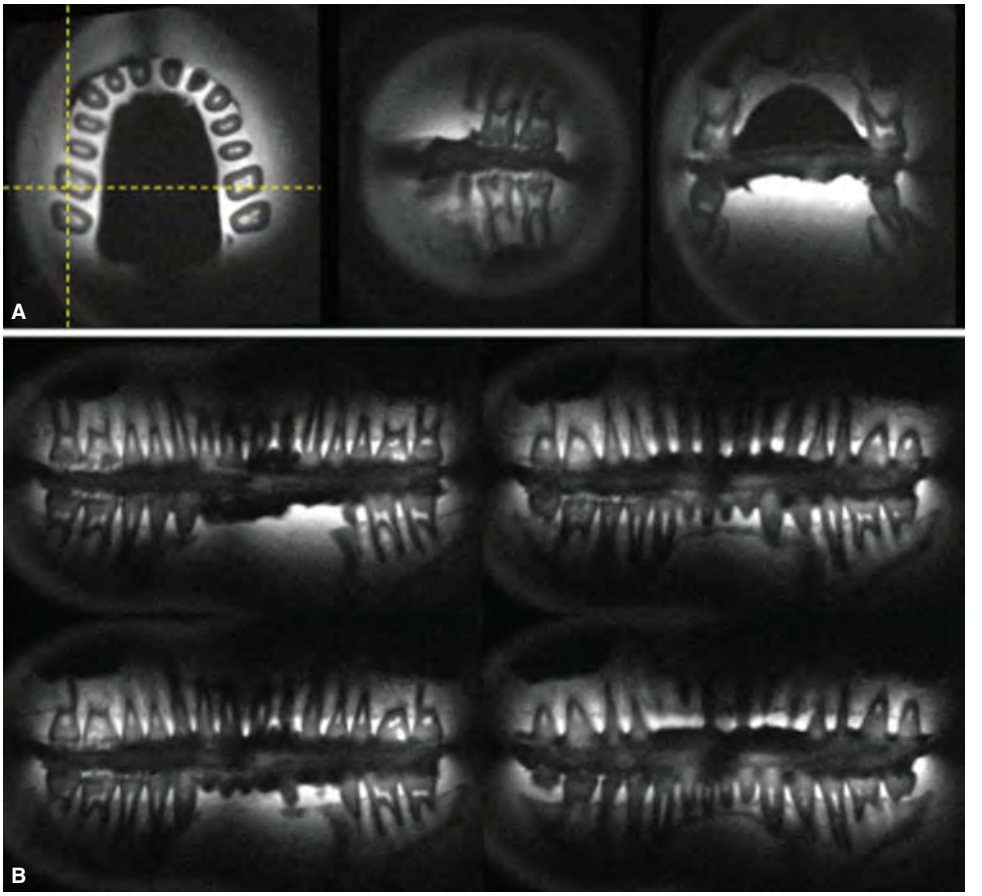

FIGURE 9D-3 ***A***, Selected orthogonal slices. ***B***, Selected panoramic slices obtained by 3D SWİFT image; maximum coil sensitivity reflects the interested structures such as teeth and supporting tissues. (Adapted from Idiyatullin D, Corum CA, Nixdorf DR, Garwood M. Intraoral approach for imaging teeth using the transverse B1 field components of an occlusally oriented loop coil. *Magn Reson Med* 2014;72:160-5. Used with permission.)

ADVANTAGES AND DISADVANTAGES OF MRI USE

MRI can produce very high spatial resolution images in any plane and provides images without exposure to ionizing radiation used in X-rays and CT scans. Conventional tooth radiographs cause superposition of pulp structures since it shows only 2-D projection of a tooth in the bucco-lingual direction. Therefore, the fourth canal of maxillary molars can be scarcely visualized. Using MR imaging, the anatomical variations in teeth including the size and shape of the pulp chamber and root canals are well demonstrated by MRI 3-D images that can be converted into a projection and give significantly more information than radiographic 2-D projections. MRI signals of soft tissues may also provide valuable information about the vitality of the pulp tissue. Therefore, it is considered as a convenient method for dental pulp imaging that cannot be directly assessed by radiographic imaging.[11,19,28]

Every object exposed to a magnetic field displays a certain amount of magnetism depending on composition of the material. Although fixed dental prostheses, amalgam restorations, and orthodontic appliances can produce image distortions on MRI scan of orofacial area, they exhibit minimal deflection in a static magnetic field that can be a major problem with CT technology and are, therefore, regarded safe for MRI.[3,6,29]

MRI has several disadvantages when compared to simple radiographs such as, poor resolution and long scanning times, reducing patient comfort and risking safety. Another aspect of the MRI technology is that it requires high financial resources for setup and operation of MRI units. Different types of hard tissues such as enamel and dentine cannot be differentiated from each other or from metallic objects; they all appear radiolucent. Specific absorption rate that leads to tissue heating may also limit the use of MRI. Although it was suggested that MRI can be used in children and even pregnant patients, claustrophobia (i.e., morbid fear of closed places) may affect the patient's motion. Additionally, contrast materials may cause allergic tissue reactions. Due to these reasons, MRI still has limited use for management of endodontic diseases.[6,11]

A strong magnetic field has the capability to pull heavy objects towards the scanner at a very high velocity.

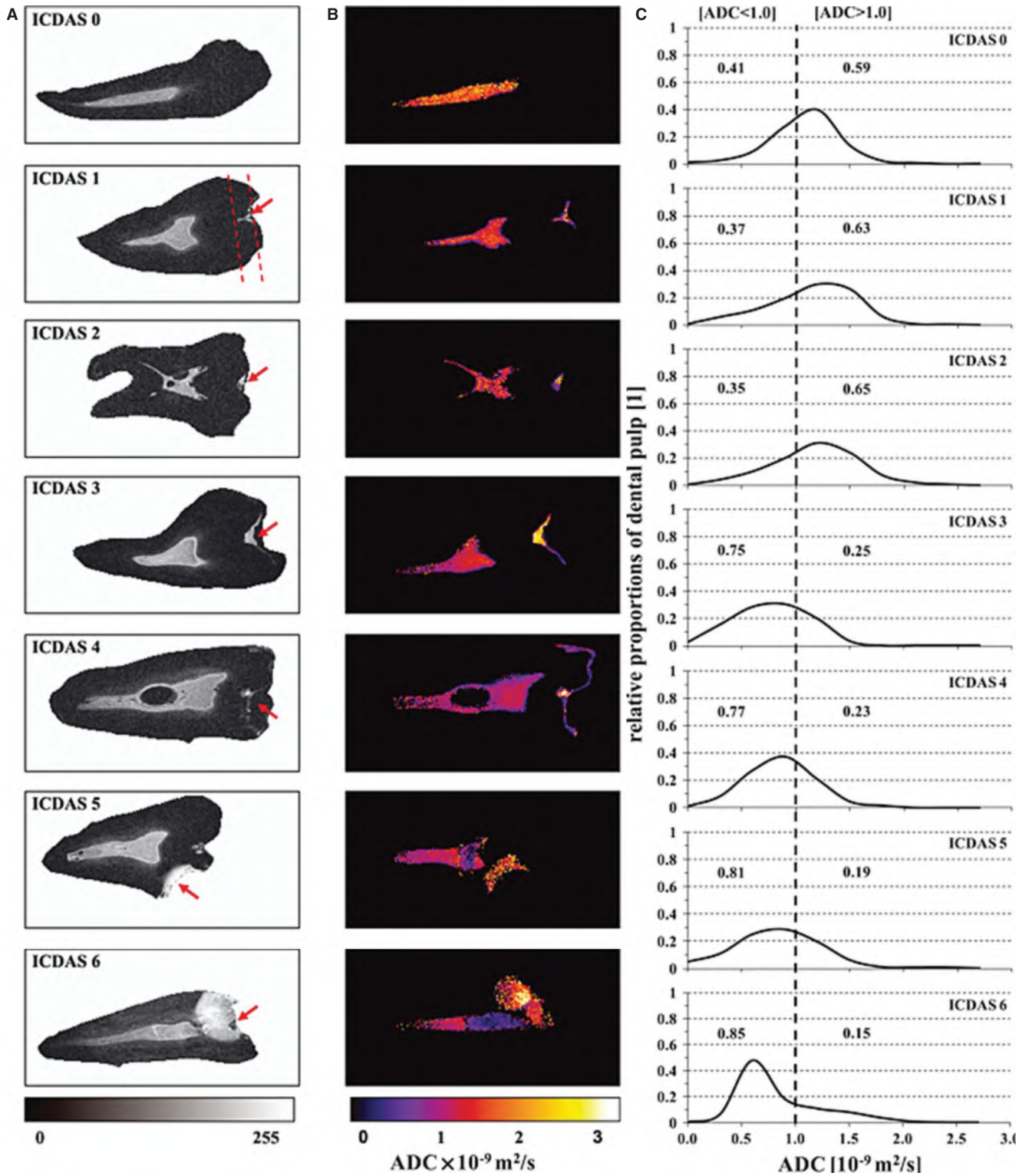

FIGURE 9D-4 T1-weighted MRI with ADC mapping for assessment of dentin–pulp complex response to caries at 2.35 T. ***A***, T1-weighted MR images of intact and decayed teeth. Detection of caries lesions were classified according to ICDAS (Caries detection and assessment system) scores. Caries lesion and pulp tissue present white brilliant image. ***B***, ADC maps clearly indicates the morphology of demineralized hard dental and pulp tissue in T1-weighted MR images. ***C***, Normalized ADC distributions of dental pulps having different ICDAS scores. The threshold ADC values of 10^{-9} can be considered as a boundary between the intact and decayed dental pulp. With increasing ICDAS scores, depending on caries lesion, ADC peak is below the threshold. (Adapted from Vidmar J, Cankar K, Nemeth L, Serša I. Assessments of the dentin-pulp complex response to caries by ADC mapping. *NMR Biomed* 2012;25:1056-62. Used with permission.)

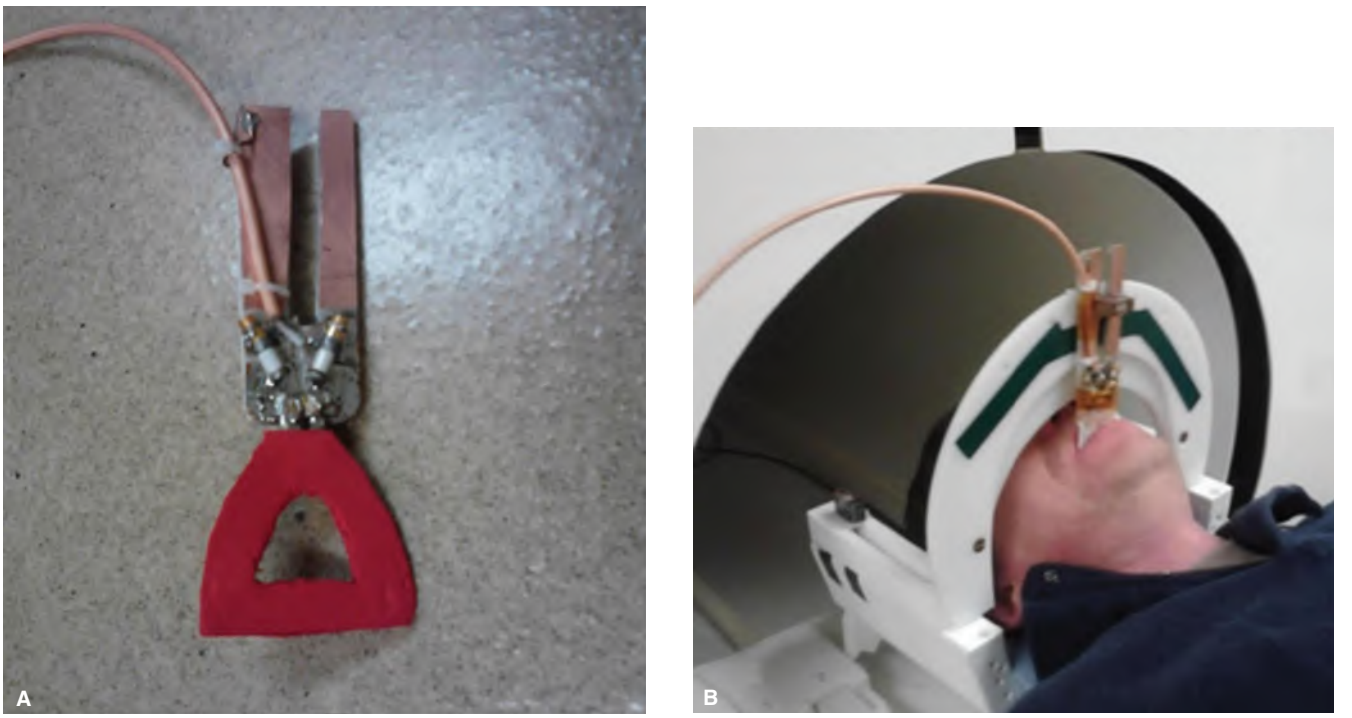

FIGURE 9D-5 ***A***, Intraoral RF loop coil containing copper foil of 10 mm in width. Its design was fit to the average adult maxillary arch. ***B***, In-vivo experimental coil set. Loop coil was placed in occlusal position. (Adapated from Idiyatullin D, Corum CA, Nixdorf DR, Garwood M. Intraoral approach for imaging teeth using the transverse B1 field components of an occlusally oriented loop coil. *Magn Reson Med* 2014;72:160-5. Used with permission.)

This is called a *projectile effect*. Patients with biomedical devices such as pacemakers, cochlear implants, neurostimulators, and infusion pumps are considered to be at high-risk. MRI's magnetic field can cause these devices to become nonfunctional, potentially generating life-threatening situations and dislocation.[3,6]

The signal-to-noise ratio and resolution are both significant parameters for high- quality dental MRI resolution. It is highly dependent on the configuration and performance of the RF coil that provides a limited magnetic field. In clinical practice, MR imaging for dental use is obtained mostly by a head and neck coil. Recently, Idiyatullin et al.[24] developed a new intraoral loop coil in the occlusal position. This device produces high nominal resolution images (0.3 mm^3) with stronger signal detection of teeth and associated structures, thereby increasing accuracy (Figures 9D-5). Additionally, contrast-enhanced dental MRI (covering the teeth with water-based contrast materials) may offer a better visualization and signaling of the different anatomical dental structures.[19,28]

Human teeth are relatively small. Therefore, the resolution needs to be high. It requires the use of a strong magnet with proper RF coil, specific for dental applications. Its magnetic field produces very stable and extremely homogeneous magnetic field. Therefore, these magnets are practically the only efficient solution for high-resolution MRI of teeth. Currently strong magnets (9, 4 or 11, and 4 T) are only available for in vitro use due to safety.[8,23,26] With advancements in clinical MRI hardware, those magnets may be applied to clinical imaging for new treatment modalities in regenerative endodontics and in other fields of dentistry.

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Chapter 9E

Ultrasound

Elisabetta Cotti

Given the importance of imaging in the diagnosis, description, follow-up, and study of endodontic lesions in the mandible and maxilla, as well as the development of advanced diagnostic systems in traditional radiology, alternative imaging techniques are constantly being investigated.[1–4]

Osteolytic endodontic lesions in the jaws are a consequence of pulpal and periapical inflammation.[5–7] The clinical diagnosis of a tooth with a root-end radiolucency of endodontic origin is apical periodontitis. However, following biopsy and depending on the microscopic features of an apical lesion, it may receive a histopathologic diagnosis of either granuloma or cyst. The possibility of making a clinical differential diagnosis between a granuloma and a cyst may be important in the management of such lesions, especially in predicting the outcome of endodontic treatment or explaining a lack of healing.[6, 8–13] Traditional radiographs are not good guides for differentiating these types of lesions, because they allow the visualization of hard tissues only and not soft tissues.[1–4] Attempts to correlate the appearance of endodontic lesions on computed tomography (CT) scans, and later on cone beam computed tomography (CBCT), to their histopathologic features have not been fully successful. Trope et al.[10] suggested that a cyst could be differentiated form a granuloma because it exhibits a CT density similar to the background (darker than a granuloma), while a granuloma shows more density, similar to that of the surrounding soft tissues. Controversial results have been served when investigators identified the cut off point between different shades of gray as a way to differentially diagnose endodontic bone lesions on CBCT.[11–13]

In recent years, ultrasound real-time echotomography, a non-invasive and safer imaging technology not using ionizing radiation, has been successfully used to supplement traditional radiology, in the study of apical periodontitis.[2–4]

BASIC PRINCIPLES OF ULTRASOUND REAL-TIME IMAGING

Ultrasound real-time imaging, also called echography or real-time echotomography, has been a widely used diagnostic technique in many fields of medicine since its introduction in 1942.[14,15] The production of ultrasonic images is based on the generation and reflection of ultrasound waves.

Ultrasound (US) waves are generated as a result of the so called *piezoelectric effect*: when a quartz or a synthetic ceramic crystal is exposed to an alternating electric current, the electrons cause a change in the structure of its grid. This change emits mechanical energy in the form of US waves. The crystals distribute waves at a frequency that depends on the alternating current (AC) they are exposed to (i.e., crystals exposed to a 7 MHz AC create ultrasound waves oscillating to a frequency of 7 MHz). Ultrasound waves, generated for a diagnostic purpose, employ frequencies more often ranging from 1.6 to 20 MHz; US waves oscillating at the same frequency are bundled by means of an acoustic lens and directed toward the area of interest in the body via an ultrasonic probe (transducer) that contains the crystals (Figure 9E-1A).

Biologic tissues of the body possess different mechanical and acoustic properties. When the US waves encounter the interface between two biologic tissues, with different acoustic impedance, they undergo phenomena of reflection and refraction. The echo is the part of the US wave that is reflected back from the tissue interface toward the crystal (Figure 9E-1B).

The crystal is capable of transforming an incoming ultrasound wave (echo) of the same frequency back to an electromagnetic wave. The echo is then transformed into electric energy that in turn is transformed into a light spot, using a gray scale, into a computer monitor built into the ultrasound apparatus[15] (Figure 9E-2). The intensity of the echoes depends on the difference in acoustic properties between two adjacent tissue compartments; the greater the difference, the

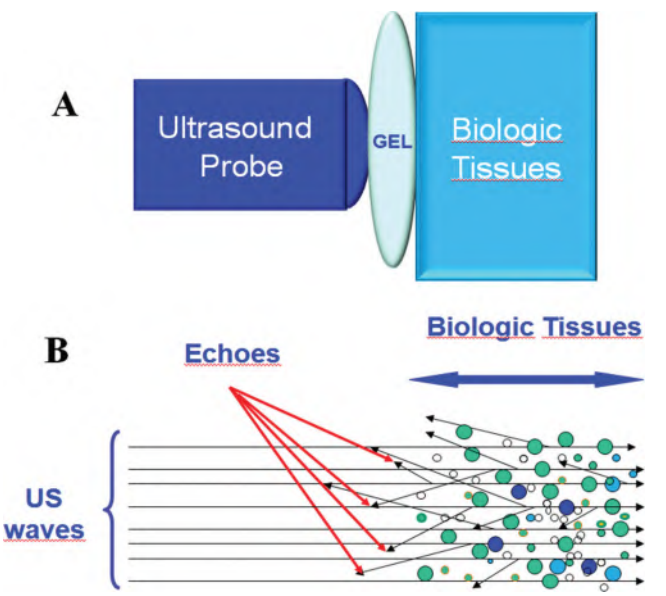


FIGURE 9E-1 ***A***. Schematic representation of sending ultrasound (US) waves into biologic tissue with a probe. ***B***. Production of echoes from ultrasound waves sent into biologic tissues and reflected back to the source.

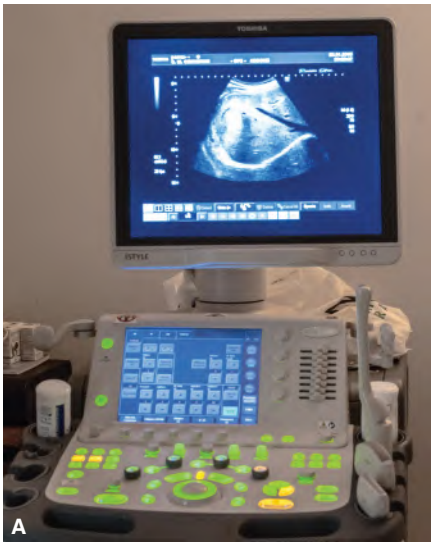


FIGURE 9E-2 Ultrasound technical equipment. ***A.*** Ultrasound imaging apparatus with the transducers. ***B.*** Detail of three different transducers/probes (arrows).

greater the amount of reflected ultrasound energy, the higher the echo intensity. Low echo signals appear as dark spots whereas high echo signals appear as bright/white spots.

The point in the tissue where the echo originates is calculated by measuring the time delay from the beginning of the wave signal until the return of the echo. The ultrasound images seen on the monitor are produced by the automatic movement of the crystal over the tissue of interest. Because each movement of the crystal gives one image of the tissue (depending on its plane), and an average of 30 images per second are produced, the tissue being examined will appear on the monitor as a sequence of moving images. The movement of the probe by the operator, on the area of examination, creates a change on the sector plane, thus allowing the creation of a "real-time three-dimensional image" of that space.[14,15]

The latest US imaging systems use multi-frequency, wideband transducers with multiple, low-impedance linear crystals (up to 256) organized in 3 to 5 layers and electronically connected in a variable sequence. The resolution of the US image depends on the distance between the focus and the section of the tissue studied. The dynamic focalization gives a precise resolution in structures smaller than 2 mm by varying the frequencies within 5 to 10 MHz.[16] The interpretation of the gray values on the images is based on a comparison with those of normal tissues. When an area in a given tissue has high echo intensity, it is called "hyperechoic." When it displays low echo intensity, it is termed "hypoechoic"; if characterized by absence of echo intensity, it is called "anechoic" or "transonic."[15] In areas filled with fluids (i.e., cysts) reflection of US waves does not occur (anechoic); whereas in bone, total reflection occurs (hyperechoic). Areas that contain different types of tissues show a "dishomogeneous echo." The presence of air in the field of observation exhibits total reflection. It is, therefore, necessary to use a gel as an interface between the probe and the tissues while performing the exam (Figure 9E-1A).

The echographic features of the tissues may be described as follows:

- ***Solid tissue structures***: echogenic. They reflect the echoes in many different ways and can result in hypoechoic or hyperechoic effects. Within this group are: a) solid homogeneous structures; b) solid dishomogeneous structures; c) solid structures with "posterior shadows" (i.e., solid structures with calcific formations); and d) solid structures with colliquations (dissolved tissues)
- ***Liquid structures***: transonic. They may present "reinforced contours" and "lateral acoustic shadows." Liquid structures may also present themselves as: a) liquid with aggregates; b) liquid with septa; and c) liquid with parietal echogenic vegetations.

The ultrasound examination can be supplemented by use of Color Power Doppler (CPD) flowmetry to determine the perfusion of the tissue of interest. When applied to the ultrasound, examination with the CPD offers the opportunity to observe the presence and direction of the blood flow and the intensity of the Doppler signal with its changes in real time

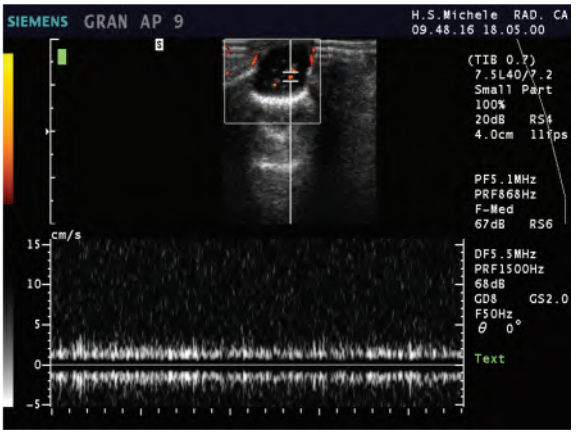


FIGURE 9E-3 Image representing the echo color power Doppler applied to the echographic examination of a periapical lesion. On the upper right, the ultrasound image of the lesion is showed (framed). The colored spots represent the presence and direction of the blood flow (color Doppler). The intensity of the Doppler signal with its modification in time (cm/s) is represented in the lower part of the figure.

(Doppler), in the format of color spots superimposed on the images of blood vessels (color), and with very high sensitivity (power).[15,17–20]

The Doppler ultrasound test uses sound waves transmitted to a vessel from a transducer in the probe. The part of the waves reflected back to the probe is processed by a computer that creates graphs or pictures representing the blood flow within that vessel. The movement of blood cells causes a change in pitch in the reflected sound waves (Doppler effect). The Doppler shift of the moving blood is monitored continuously to form the Doppler signal. Because the transmitted frequency is about 2 to 4 MHz, the Doppler shift of moving blood is in the audible range, and is represented on a graph with its changes in real time; the spectra obtained are plotted as a function of time (horizontal axis) and frequency shifts (vertical axis).

The CPD technique combines ultrasound real-time imaging with the color encoding of the Doppler frequency shift and is used to visualize the presence and velocity of blood flow within an image plane. The frequency shift is color-encoded depending on the velocity and direction of the blood flow relative to the transducer and is superimposed on the grayscale image. The way in which the frequency shifts is encoded and defined by the color bar located to the left of the image (Figure 9E-3).

Blood flow is assigned different hues that reflect the flow velocity and the insonation angle. Fast flow is assigned lighter hues, whereas slow flows appear with deeper hues[17–19] (Figure 9E-3). Positive Doppler shifts are caused by the blood moving toward the transducer and are encoded as red; negative Doppler shifts are caused by blood moving opposite to the transducer and are encoded as blue. Because color Doppler presents some shortcomings such as dependency on the insonation angle and the "aliasing," *power Doppler* is best associated with it to improve its sensitivity to low flow rates. The power Doppler is based on the integrated power spectrum, is angle independent, and is up to five times more sensitive in detecting blood flow than color Doppler.[17–19] CPD discloses the presence of the network of minor vessels and slower flows within the tissue of interest.[17–19] Intravenous injections of contrast media increase the echogenicity of the blood by enhancing the difference in acoustic impedance in the area of interest, thus rendering the CPD examination more sensitive.[15,20,21] The use of second-generation contrast media that reach the lumen of the smaller vessels allows the evaluation of the blood flow in its wash-in and wash-out phases and the study of the micro-vascular system.[19]

Three-dimensional (3-D) ultrasonic imaging can also be performed to obtain 3-D scanning and volumetric reconstructions of the US scatters. It can be obtained manually (optically tracked) or mechanically, either by moving a 1-D ultrasonic array, or by 2-D array technology.[22]

Ultrasound imaging is considered to be a relatively safe technique. Ultrasound waves targeted into biologic tissues are absorbed in the form of heat energy. This could be hazardous because it causes cavitation and vibration.[15] On the other hand, it is important to note that the crystal emits energy only during 0.1% of its activity, whereas the remainder of the time it is receiving echoes - it is "listening." Any potential adverse effects of the system depend on the length of application time of the sound energy. Safety, therefore, is achieved by limiting the number and the repetitions of the examinations. No adverse effects of ultrasound waves have been reported in experimental and clinical studies.[23–25] In any case, the risk entailed is much lower than that associated with radiographic investigations.[23–27]

To perform an echographic examination in the maxillary bones, the patient is in a clinostatic position or sitting or in an examination bed. The probe is protected with a latex/plastic cover and topped with the echographic gel and is moved by the radiologist inside the buccal area of the mandible or the maxilla that corresponds to the periapical area of the tooth that has been identified has having apical periodontitis. The same examination can also be performed extra-orally, by placing the probe on the external surface of the skin. The radiologist controls the progression of the exam in the computer monitor, while moving the probe around the area of interest to obtain an adequate number of scans to picture the lesion in real time. Each moving sequence or single image can be selected and stored in the computer.

The transducers (Figure 9E-2B) best suited for dentistry are linear probes, and allow the formation of a high frequency (7,5 MHz–15 MHz) rectangular or trapezoidal image that exhibit both optimal lateral resolution and optimal contrast of the superficial structures.[16] The transducers can also be in the format of intra-operatory probes (smaller probes useful for intraoral examination). To better evaluate bone lesions in the jaws, and especially to reduce the artifacts in fluid structures (cysts), these are simultaneously insonated with converging boundless of US waves sent from different directions (*compound technique*).[20]

To date, ultrasound real-time imaging has been applied to dentistry for the evaluation of maxillo-facial soft-tissue inflammatory lesions,[22,28] bone lesions,[29] fractures,[22,30] and

for the diagnosis of temporo-mandibular joint disorders[16,22,31] as a complement to clinical and radiographic examinations.

ULTRASOUND IMAGING IN ENDODONTICS

Ultrasound real-time imaging has been successfully applied to the endodontic field for the detection and description of apical periodontitis,[32] for differential diagnosis between cystic and solid lesions of endodontic origin,[4,33–37] for differential diagnosis between endodontic lesions and non-endodontic lesions,[29,36] and for evaluating the short-[38] and long-term[39–41] outcome of orthograde and surgical endodontic treatment.

Detection of Endodontic Lesions of Apical Periodontitis

In 2001, a pilot project was conducted at the University of Cagliari, Italy, to try ultrasound real-time examination in the study of periapical lesions of endodonticorigin. Several patients diagnosed with apical periodontitis, based on clinical signs and symptoms and intraoral and panoramic radiographs, were asked to participate. They signed an informed consent and had echographic examinations of the areas affected by the apical lesions. An endodontist, an oral surgeon, and a radiologist expert in echography were involved in the project.

After a few trials, it was possible to consistently trace the lesions within the mandible and maxilla and describe them by means of echography. Twelve patients diagnosed with endodontic periapical lesions were chosen to participate.[32] Intraoral and/or panoramic radiographs were used as a guide. An Elegra Siemens apparatus (Siemens, Erlangen, Germany) with a standard, high-definition multi-frequency ultrasound probe of 7 to 9 MHz was used for the examinations. The echographic probe was moved into the buccal area of the mandible or maxilla, corresponding to the area of tooth of interest. When the bony defect was identified, the probe was moved around the area to obtain an adequate number of transversal scans to define the lesion.

The data were accurately reported in a descriptive chart. It was noted that examinations were relatively easy to perform and that the patients reported no discomfort. In all the cases examined, it was possible to obtain an ultrasound real-time three-dimensional image of a given lesion, to measure its dimensions, and to evaluate its content (fluids, solid, or a combination of both). Following the application of CPD to echography, it was possible to see the presence of blood vessels within a lesion.

Based on these first data, application of US real-time echotomography,[2,4,32–41] the healthy and pathologic tissues within the maxillary bones appeared as follows in the echographic images:

1. Alveolar bone, if healthy, is hyperechoic because it exhibits a total reflecting surface that appears white in the image (Figure 9E-4A).
2. The roots of the teeth are also hyperechoic and appear as an even whiter shade than bone and can be seen in three dimensions (Figure 9E-4B).
3. Solid lesions in the bone are echogenic or hypoechoic presenting various intensities of echoes and appear as different shades of gray (Figure 9E-5).
4. A bone cavity filled with clear fluids (i.e., serous) is anechoic (or transonic) because it has no reflection and it appears dark (Figure 9E-6A).
5. A bone cavity filled with fluids containing inclusions is hypoechoic and exhibits various degrees of darkness, depending on the contents of the fluid (Figure 9E-6B)
6. The irregular bone (resorbed) around a lesion shows a dishomogeneous echo.
7. The reinforced bony contour of a lesion is usually very bright (hyperechoic) (Figure 9E-6).
8. By applying the CPD to the examination procedure, the presence and direction of blood vessels around and within the lesion can be detected (Figures 9E-3 and 9E-5B).

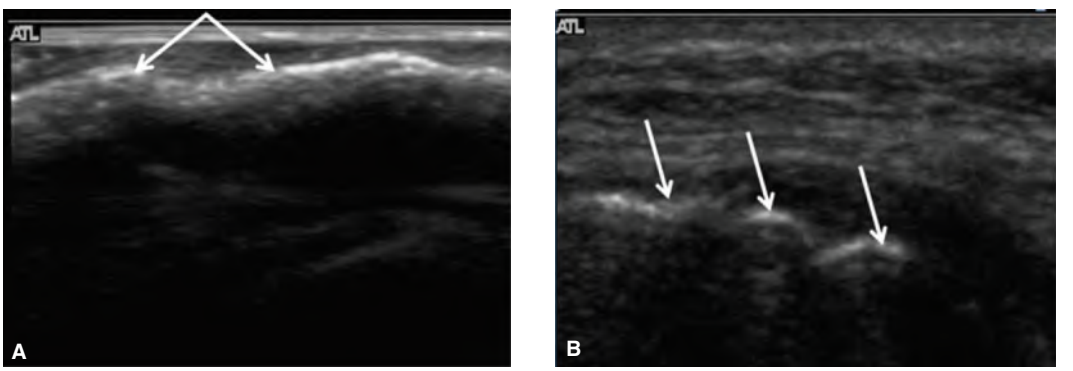


FIGURE 9E-4 ***A.*** Ultrasound image of the profile of the maxilla (arrows); the image shows a "hyperechoic" area (white) where total reflection occurs. ***B.*** Ultrasound image of the maxilla that shows the contours of three roots of teeth (arrows); the contours are hyperechoic (white).

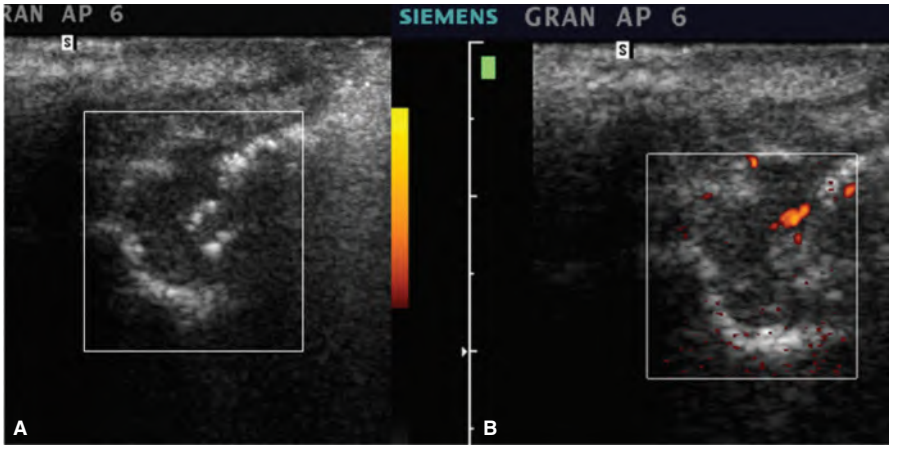


FIGURE 9E-5 Ultrasound real-time imaging of 2 different lesions in the jaws. ***A.*** Periapical lesion (granuloma) as seen in the ultrasound image (framed); it is an "echogenic," hypoechoic area where echoes are reflected at different intensities. ***B.*** The same lesion after the application of color power Doppler. The colored spots represent the vascularization within the lesion.

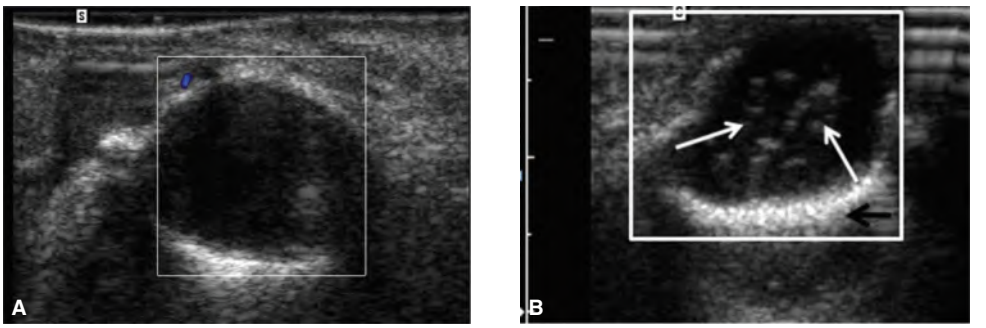


FIGURE 9E-6 Ultrasound image of two cystic lesions. ***A.*** This cyst presents an "anechoic"/ "transonic" cavity (framed) with reinforced, hyperechoic, bone contours. ***B.*** This cyst presents a transonic cavity filled with fluids and containing inclusions (arrows), with reinforced bone contour (arrow head).

9. The mandibular canal, the mental foramen, and the maxillary sinus can often be distinguished and appear mostly transonic.

Differential Diagnosis of Cysts and Granulomas

A study was done to differentiate between cystic lesions and granulomas based on the ultrasound examination complemented with the CPD procedure.[33] It included 11 patients with clinical diagnoses of apical periodontitis whose treatment plan included endodontic surgery with apicoectomy, root-end filling, and biopsy. The ultrasound examination with CPD was performed in each area of diagnostic interest in the maxilla. A representative echographic image was selected for each lesion, analyzed, and recorded. An attempt was made to differentiate a diagnosis between cystic lesions and granulomas based on the following principles:

1. Cystic lesion: a transonic, well-contoured cavity filled with fluid, surrounded by reinforced bony walls with no evidence of internal vascularization based on CPD (Figure 9E-7).
2. Granuloma: a distinct lesion that could be echogenic or mixed in content, with non-precisely defined contours and exhibiting the presence of blood vessels based on CPD (Figure 9E-8).

The results from the histopathologic examination confirmed the tentative echographic diagnosis. Six of 11 lesions that were described as transonic cavities were well contoured, and corresponding to the features of a cyst: a cavity lined with stratified squamous epithelium with necrotic debris and impressions from cholesterol crystals within its lumen. One lesion, whose transonic content showed also scattered echogenic particles, revealed a cystic lumen with areas of secretory epithelium. This case supports the specific sensitivity of this technique with respect to the content of bone pathosis. Four of the remaining lesions were echographically diagnosed as granulomas because of their echogenic appearance and the presence of internal blood vessels. Their reports described the typical appearance of granulomas: connective tissue with peripheral mononuclear (PMN) cells, lymphocytes, monocytes, and newly formed blood vessels. The only case that presented with both an echogenic appearance in the lower portion and a hypoechoic aspect toward the upper

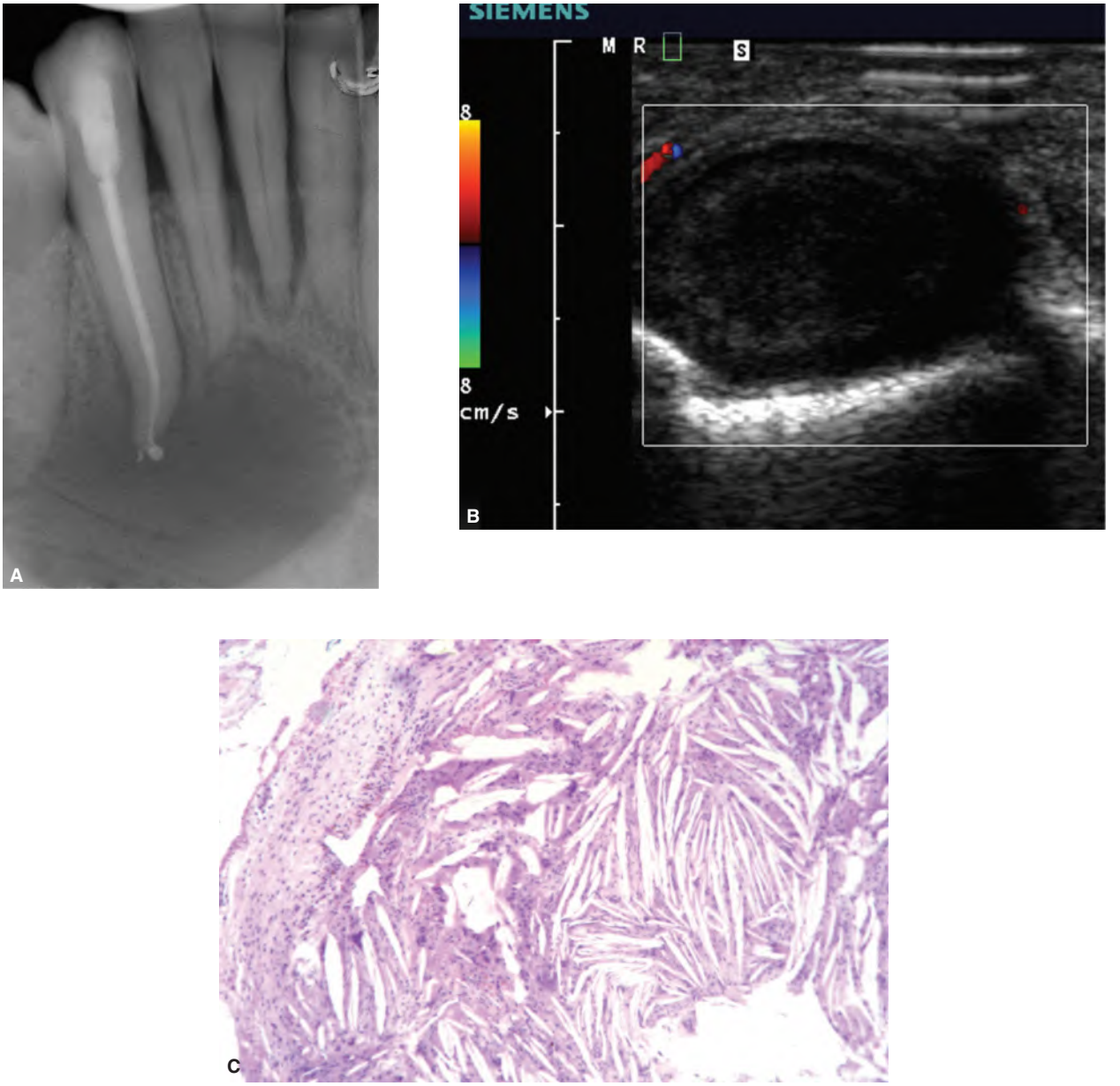


FIGURE 9E-7 Ultrasound real-time imaging of a lesion of endodontic origin in the mandible. ***A.*** Periapical lesion as seen in the intraoral radiograph; the lesion involves the periapical area of the mandibular right lateral incisor, canine, and first premolar. ***B.*** The same lesion as seen in the ultrasound image (framed): it is a "transonic" cavity with reinforced, hyperechoic bony contour. The vascular supply appearing at the CPD (red spots) is external to the lesion. ***C.*** Histopathologic features of the lesion confirmed the echographic diagnosis of cystic lesion.

end showed the histopathologic feature of a large granuloma containing a cyst. Based on these findings, ultrasound imaging showed enough potential sensitivity to allow the distinction between a cyst and a granuloma, to define a mixed type of lesion, and to evaluate the various degrees of turbidity of the fluid content in a bony lesion.[33–34]

Studies with similar designs have reported results consistent with our findings on lesions from endodontic origin.[35–37] According to Raghav et al.,[37] US examination exhibits a high sensitivity (0.95) and specificity (1.00) and is significantly more accurate than conventional and digital radiography for differential diagnosis of apical periodontitis.

Differential Diagnosis of Other Lesions in the Jaws

When lesions of a various nature in the jaws (keratocysts, dentigerous cysts, residual cysts, radicular cysts, periapical granulomas) are examined with US real-time imaging and CPD, the results are slightly less predictable.[29,36] While the diagnosis of periapical granuloma is always consistent with

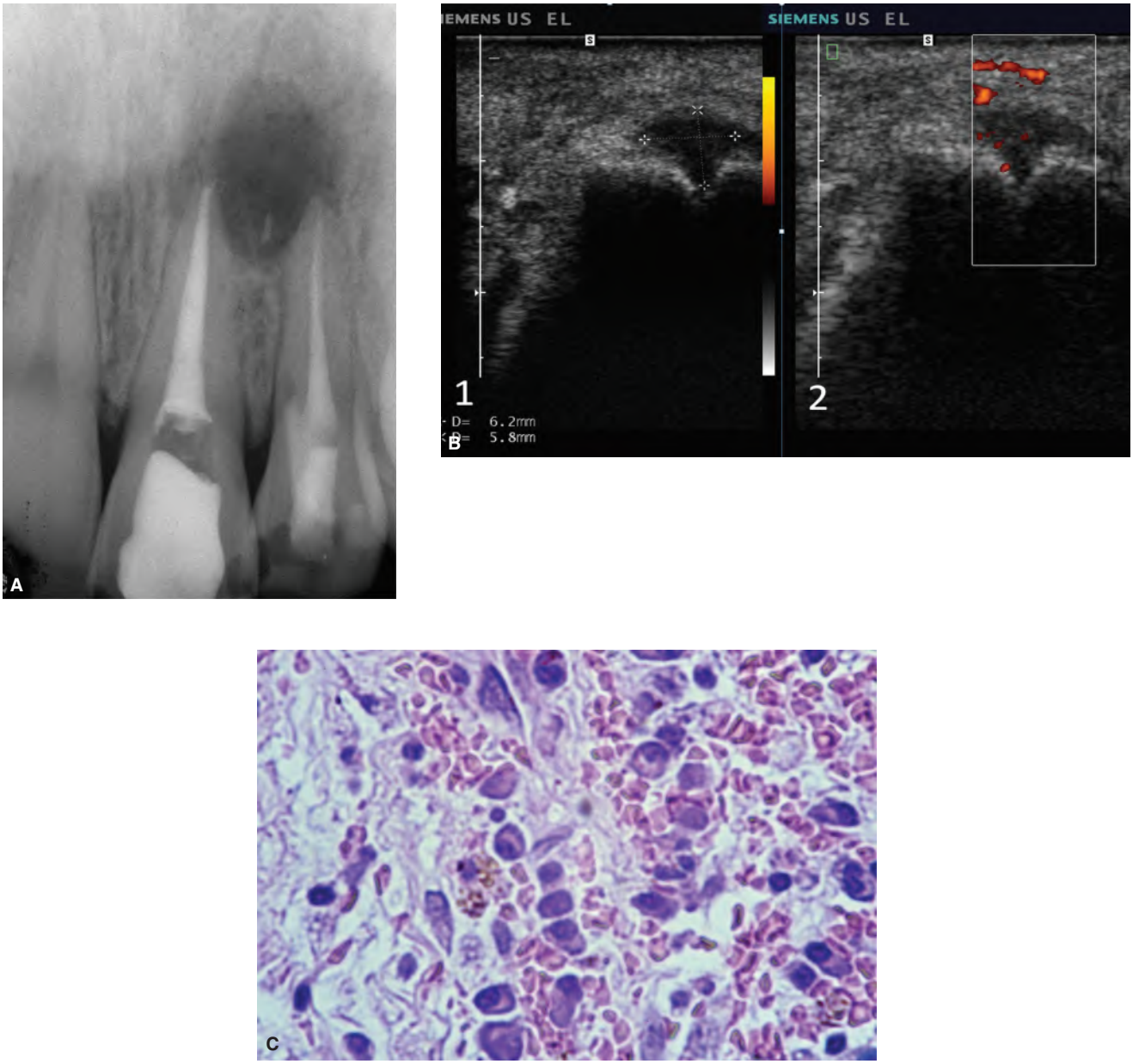


FIGURE 9E-8 Ultrasound real-time imaging of a lesion of endodontic origin in the maxilla. ***A.*** Intraoral radiograph showing a periapical lesion involving the maxillary left central and lateral incisors. ***B(1).*** Periapical lesion as seen in the ultrasound images. It is possible to distinguish an "echogenic" area with irregular contours; the dotted areas correspond to the diameters of the lesion. ***B(2).*** The same lesion with the CPD showing the presence of blood vessels within the lesion. The lesion was diagnosed as periapical granuloma. ***C.*** Histopathologic features of the previous lesion (granuloma) confirming the echographic diagnosis.

the biopsy reports, lesions presenting histopathologic features of different kinds of cysts sometimes exhibit a more composite ultrasound appearance. Infected cysts may disclose a mixed transonic and echogenic appearance because of the presence of dense secretion. In addition, when the perilesional capsule of a cystic lesion is very thick it may simulate a solid component and the presence of an internal vascular supply.

In conclusion, ultrasound diagnosis of periapical granuloma, specifically, and of solid lesions, generally, is reliable, whereas the diagnosis of cysts depends on the type of cyst, the thickness of its capsule, and its status of infection.[29,32,36,42]

Follow-up of Endodontic Treatment

By providing data on changes in echogenicity, size, and vascularity within the periapical lesion or at the surgical site, US examination associated with CPD allows for the assessment of postoperative short- and long-term healing response to endodontic treatment that is not feasible using conventional radiography alone.[4,38–41]

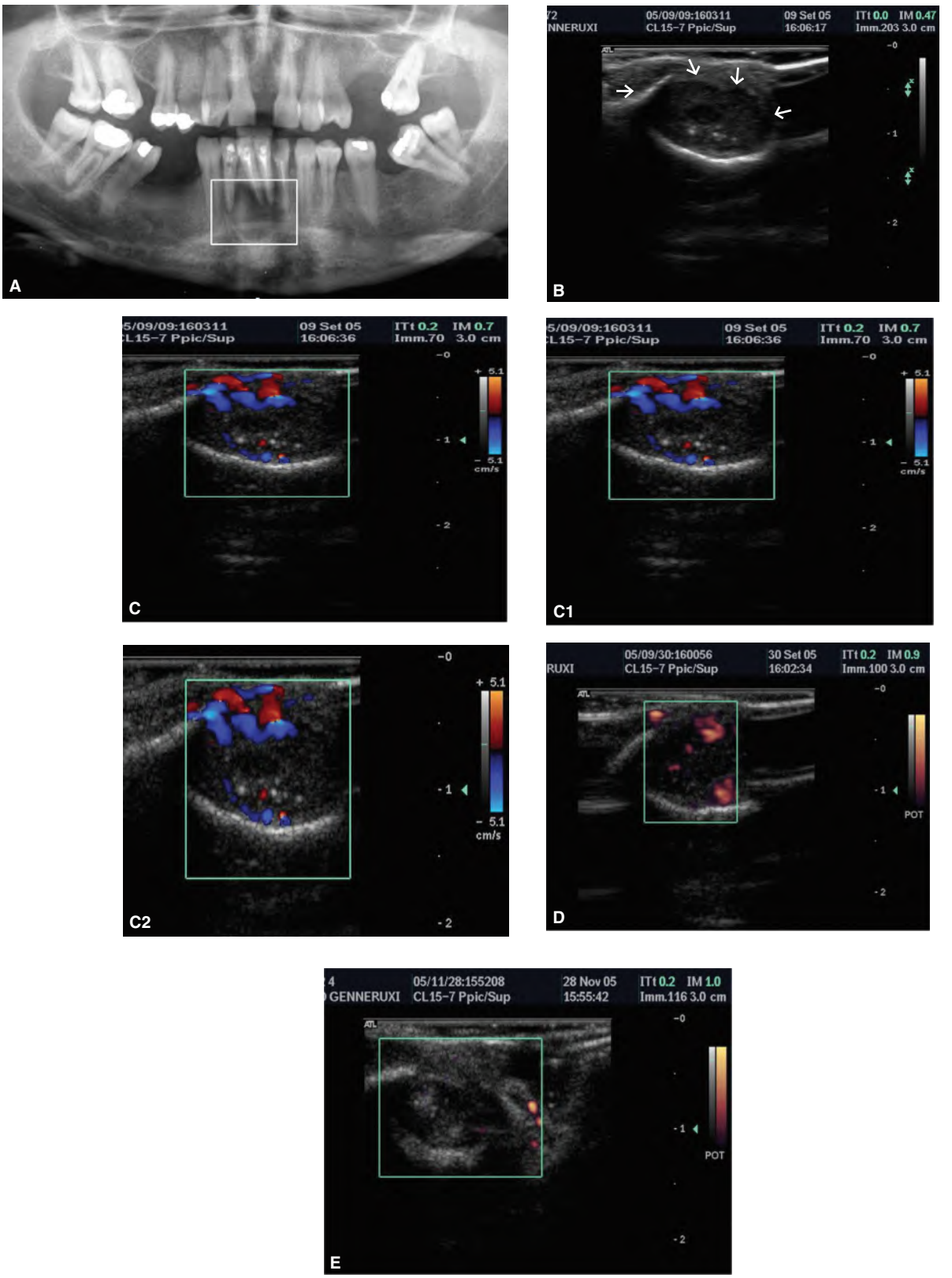


FIGURE 9E-9 Ultrasound real-time imaging follow-up of an endodontic treatment with application of CPD. ***A***. Panoramic radiograph showing an extensive periapical lesion involving all four mandibular incisors, before endodontic re-treatment (framed). ***B***. The same lesion seen in the ultrasound image (arrows): it is an "echogenic" image with solid content and well-defined contours. ***C***. The two echographic images of the same lesion after the application of the power Doppler (C1) and Color Doppler (C2) showing the presence of vascular supply. The diagnosis was periapical granuloma. ***D***. Echography of the lesion (framed) one week after endodontic re-treatment was initiated and an intracanal medication was placed. The CPD shows reduced vascularization. ***E***. Ultrasound image of the lesion (framed) one month after completion of retreatment on all four teeth. The CPD indicated that the vascular supply was greatly reduced.

Immediate Response to Treatment

The possibility of monitoring inflammatory changes in diseased bone, as an early response to endodontic treatment using the US examination associated with CPD, is the subject of an ongoing research. The rationale is that the various degrees of inflammation of a given lesion can be assumed by evaluating the diffusion of blood vessels within or/and around the lesion, and that the application of CPD has the sensitivity to detect the presence of newly formed vessels.

In a preliminary study,[38] 12 patients, presenting with apical periodontitis, whose treatment plan included endodontic orthograde treatment, have participated. A clinical protocol was established and it was decided to complete the treatment in two appointments using calcium hydroxide as an interappointment canal medication. Routine conventional periapical radiographs, and echographic examinations with CPD of each case, were carried out as follows: 1) before treatment; 2) 1 week after root canal cleaning, disinfection, and application of intermediate medication; and 3) 1 month after completion of root canal treatment.

Two independent observers evaluated the exams after obtaining a K value of 8.5. According to the data obtained, all lesions presented a vascular supply before treatment. In 9 cases, a progressive reduction of the vascularity within and around the lesion was documented during the one week and four weeks controls with the echography and the CPD, indicating a reduction in the inflammatory burden of the lesion as a consequence of cleaning, shaping and disinfection (Figure 9E-9). In 2 cases, there was an increase of vascularization at the one-week post-treatment control, and a decrease at four weeks, indicating a temporary increase in the inflammatory reaction following the initial treatment. In one case, the vascularization of the lesion remained stable during both controls indicating a failure of treatment in controlling the periapical inflammation. These data are still preliminary, but they open new horizons to the possibility of assessing the response of apical periodontitis to the different stages (and types) of treatment[38] (Figure 9E-8).

Long Term Follow-up

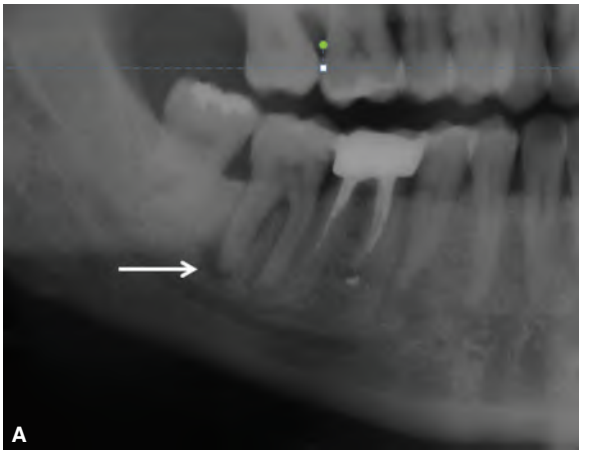
US examination with CPD has also been used to monitor the long-term response to endodontic treatment.[39–41] Rajendran[39] performed echography with CPD on 5 lesions treated with orthograde endodontic therapy by assessing the following parameters: a) changes in dimension and volume; b) variation in the echogenicity of the lesions; and c) presence, characteristics, and modifications of blood flow within the lesions. The patients were followed-up immediately after treatment and at 1, 3, and 6 months, with clinical and radiological examinations to monitor the healing process. A postoperative echography was then performed 6 months after treatment was completed. They reported that: a) the exam showed a gradual increase in echogenicity of the lesion throughout the healing phase; b) the Doppler signal was able to show the progressive formation of new vessels in bone during the initial healing process; c) as the bone was remodeled, the flow signal would progressively decrease to the point of disappearing when the lesion has completely healed; d) the presence of arterial flow and the increase in flow velocity were also a testimony of the healing process; and e) a lack of flow signals during the earlier stages, following treatment, was indicative of delayed or impaired bone healing.[39]

Later studies, based on US CPD, proved that initial signs of healing of apical periodontitis are evident as early as six weeks post operatively,[41] and that at six months follow-up, US CPD imaging is significantly more sensitive than conventional radiographs in detecting changes in healing of hard tissues at the site of endodontic surgery.[40] When monitoring the vascularity of apical periodontitis, it is advisable to use the ultrasonic probe intraorally, to avoid interferences of labial and skin blood flow.

CONCLUSIONS

Ultrasound real-time represents an advanced imaging technique that can be used in endodontics as a complement to radiographic examinations for the detection and dimensional measurement of apical periodontitis.[2,4,32–41] The most important advantage of ultrasound real-time imaging over other diagnostic imaging systems is that it is the only exam that possesses the sensitivity to assess the features of endodontic lesions (fluid versus mixed or solid content) and their internal and external blood supply, that can be used to address the differential diagnosis between solid lesions (granulomas) and cystic lesions of the jaws.[33–37] It also permits to evaluate the outcome of the treatment of apical periodontitis, based on the changes in echogencity and vascularity of the lesions.[38–41] Furthermore, the changes in the vascular supply, within a lesion of endodontic origin, observed with US and CPD, may be used to assess the immediate response of the periapical tissues to any given treatment.[38]

Ultrasound real-time examination is a non-invasive, painless method that entails lower biologic adverse effects than techniques using ionizing radiations.[23–27] A major drawback of US examination is that images interpretation is very critical and needs an expert eye, and that there are not landmarks to help distinguish the position of a lesion within a given area of the mouth, once the exam has been performed. High frequency transducers dedicated to dental use are currently being produced[43] and the 3-D volumetric reconstructions of the US scatters promise to provide better information for the study of apical periodontitis (Figure 9E-10). In the near future, the use of ultrasound looks promising for the diagnosis of caries, cracks, and fractures in teeth, periodontitis, and bone characterization for implant placement.[16,22]



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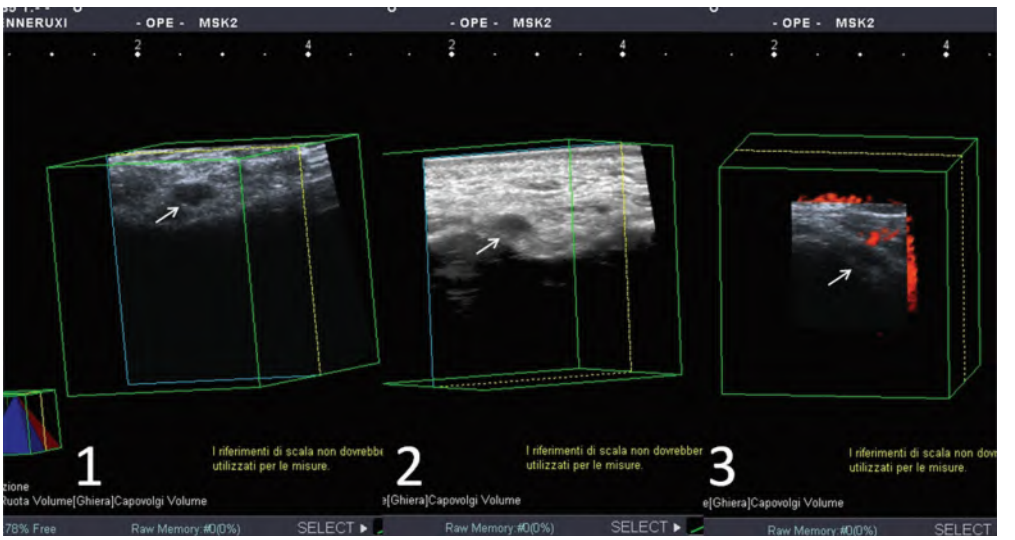

B

FIGURE 9E-10 Three-dimensional (3-D) ultrasonic imaging of a periapical lesion in the mandible. ***A***. Detail from the panoramic radiograph showing the presence of apical periodontitis on the distal root of the mandibular second molar (arrow). ***B***. 3-D echographic examination of the lesion disclosing an hypoechoic appearance and the presence of vascularity (periapical granuloma): (1) regular 3-D image (arrow); (2) 3-D subtraction image of the same lesion (arrow); (3) 3-D image with echo-CPD of the same lesion showing the vascular supply (arrow).

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Chapter 10

Imaging Interpretation

Parish P. Sedghizadeh, Rafael A. Roges, Jose-Maria Malfaz

Endodontics has long utilized imaging devices and techniques as indispensable tools for clinical, educational, and research applications. Imaging and imaging interpretation are essential to the contemporary practice of endodontics for diagnosis, therapeutics, and outcome assessment. In order to accurately interpret imaging studies and manage patients at an acceptable standard of care, the clinician must have knowledge of the normal imaging appearance of anatomy and morphology of maxillofacial structures such as teeth, jaws, articulation, and sinuses, as well as of common pathologic conditions affecting these structures.

Commonly used imaging modalities used in endodontic practice have included intraoral dental radiographs, panoramic radiography, and more recently cone-beam computed tomography (CBCT). Therefore, this chapter will focus on these modalities for clinical relevance.

Choice of imaging modality by the clinician should be predicated by multiple parameters related to individual clinicopathologic factors, and is, therefore, ideally a case-by-case decision-making process rather than a "one size fits all" approach. Certain principles of minimizing radiation exposure to patients, such as: "as low as reasonably achievable" (known by the acronym ALARA), also guide radiographic decision-making clinically.

It is important to recognize that there are advantages, disadvantages, and limitations to any imaging study; those can affect interpretation. One of the most important advantages of any of the aforementioned imaging techniques is that it provides the clinician with visualization of hard tissue anatomy, or pathology, not normally visible by clinical examination alone. For conventional dental intraoral radiographs, advantages include low cost, being readily available in the dental practice, high spatial resolution, and low radiation dose that is even further minimized with digital radiographs. As far as the disadvantages, intraoral X-rays have inherent limitations in that 3-dimensional (3-D) anatomy is represented in a 2-dimensional (2-D) image. This may result in geometric distortion and anatomic noise that can mask a region of interest.[1] Superimposition of anatomic structures when viewed in two dimensions can create a false representation of reality, suggesting normality when pathology exists, or suggesting pathology when normality exists (Figure 10-1). This case also highlights the importance of familiarity with normal anatomy and anatomic variations for accurate diagnosis. Some of the limitations of intraoral radiographs were already described decades ago by Seltzer and Bender in their classic experiments.[2,3] Panoramic radiography also represents a 2-D image of 3-D anatomy that again has some limitations. It also contains artifacts and unique distortions that can further complicate panoramic interpretation.

The overall purpose of this chapter is to provide an objective approach to radiographic diagnosis and interpretation for clinical applications in the endodontic practice. Radiographic interpretation inherently has subjectivity and observer bias in addition to having objectivity. For example, disagreement was found in the interpretation of radiographs by different evaluators regarding the presence of periapical radiolucencies,[4] as well as, the same evaluator interpreting the same image over time.[5] Limitations of radiographic interpretation have been minimized by exposing multiple radiographs, from different angulations, to improve visualization of changes in an area in question.[6]

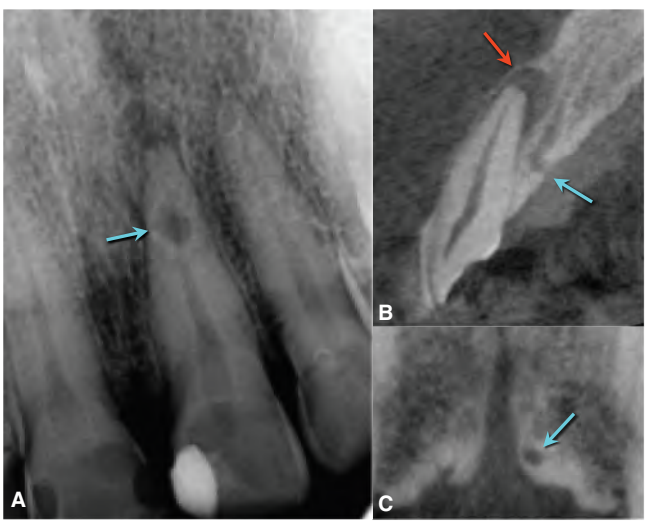

FIGURE 10-1 *Canalis Sinuosus* mimicking the appearance of root resorption. ***A.*** Periapical radiograph of a maxillary left central incisor with recurrent caries and pulpitis showing apical radiolucency, and what appears to be root resorption in the apical third of the root (blue arrow). ***B.*** CBCT sagittal reconstruction showing the same tooth with an apical lesion (red arrow), however, without evidence of the root resorption. Instead, *canalis sinuosus* was detected. *Canalis Sinuosus* is a normal anatomic variation of the anterior superior alveolar neurovascular bundle with rare manifestations in the anterior maxilla. ***C.*** CBCT coronal reconstruction showing the terminal region of the *canalis sinuosus* that is distinct from the incisive canal (blue arrow). Because of the nonmidline location, *canalis sinuosus* can mimic various pathologic lesions on conventional radiographs when superimposed on other anatomic structures, such as a tooth root in this case. (Courtesy of Dr. Daniel S. Schechter, Los Angeles, CA.)

Over the years, advancements in devices, hardware, software and modes of operation significantly improved the information obtained. Digital radiography has allowed for image enhancement with control of parameters such as brightness, contrast, magnification, inversion, color, and resolution. This is a clear advantage over traditional radiography. The introduction of digital radiography has improved the speed of image acquisition and potentially reduced patient exposure to radiation. Nevertheless, the overall limitations of conventional radiography and 2-D imaging remain inherently present.[7] For more details, see Chapters 9A and 9B.

THE ADVENT OF CBCT AND THE THIRD PLANE OF VISUALIZATON

CBCT has been optimized for dental applications. Smaller fields of view and higher resolution scans appear to be ideal for endodontic use and may impact the nature of endodontic practice and radiographic decision-making in the near future.[8–10] For more details, see Chapter 9C.

One of the disadvantages of CBCT use is cost and access to the technology. However, with time, costs should continue to decrease and access to increase. Other areas of controversy and concern are radiation exposure to patients, the training needed to operate and interpret the scans optimally, as well as the amount of time necessary to evaluate them. Like all radiographic studies, CBCT imaging and diagnostic accuracy rely heavily on parameters such as image quality, viewing conditions, and observer performance characteristics. Interpretation of the collected volumes, potentially affecting treatment planning, can be a challenge with respect to defining normal versus pathological conditions.[11] Hardware and software parameters also heavily influence image quality and resolution.[12]

CBCT, like other radiographic modalities, can also contain artifacts and noise that make evaluation and interpretation difficult in some cases. Common artifacts to be aware of include noise, aliasing, scatter and beam hardening, motion artifact, and finally streak artifact that usually results from metal or high density restorations in the oral cavity such as metal crowns, orthodontic appliances, amalgam fillings, separated instruments and tooth, and root canal filling materials.[13,14] (Figure 10-2).

A major advantage of CBCT is the visualization of anatomic structures in three planes, generally referred to as coronal, axial (transverse), and sagittal reconstructions. CBCT technology also allows for panoramic and oblique reconstructions in addition to anatomic-specific reformatting such as temporomandibular joint (TMJ) analysis. This 3-D evaluation as compared to 2-D evaluation allows for unprecedented applications in dentistry and endodontics, and in some instances has improved the sensitivity and specificity or positive predictive value of accurately detecting lesions or conditions affecting the jaws.

For endodontic applications, the future is the third plane of visualization (3-D), however, there is still some controversy regarding CBCT use over conventional dental radiography for routine cases. Some of this controversy may be due to lack of higher level evidence-based clinical studies comparing CBCT with conventional radiography in specific contexts. Nonetheless, endodontics has successfully utilized CBCT mainly in the following contexts:

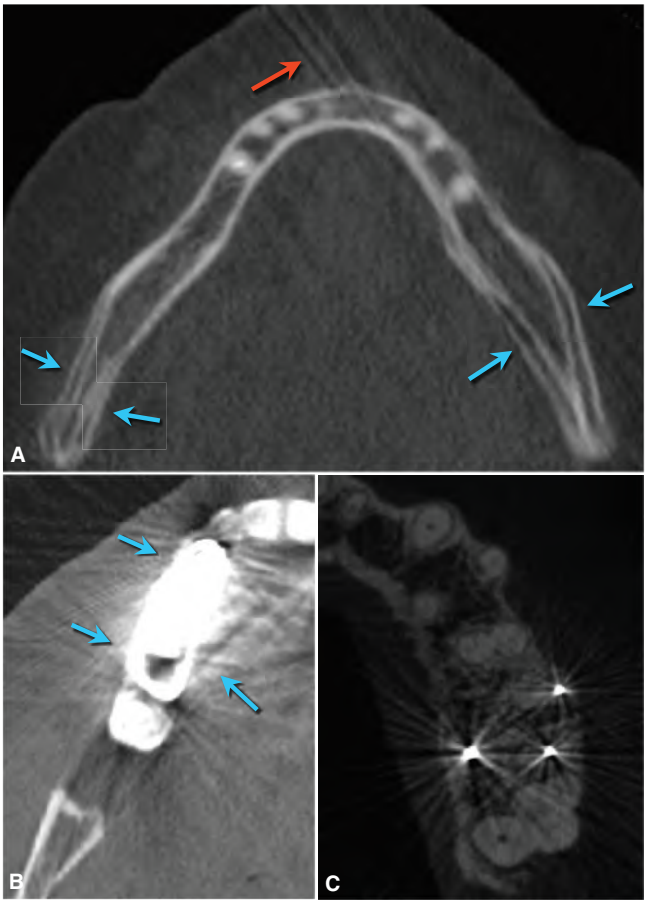

FIGURE 10-2 Common artifacts associated with CBCT. ***A.*** Motion artifact from patient head movement during image acquisition that produced a blurry or double-image of the mandible (blue arrows). Note the aliasing artifact (red arrow) presenting as linear radiolucent lines. ***B.*** Streak artifact (blue arrows) from metal restorations in the oral cavity. ***C.*** Streak artifact from silver points used for root canal obturation. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain (Figure 10-2A & B) and Dr. Parish P. Sedghizadeh, Los Angeles, CA. (Figure 10-2C).)

1. Evaluation of endodontic complications such as traumatic dental injuries, root fracture, resorption and perforation[15–21] (Figures 10-3 to 10-11)
2. Presurgical endodontic assessment and posttreatment evaluation[1, 22–25] (Figure 10-12)
3. Evaluation of root and canal morphology[26–29] (Figure 10-13)
4. Differential diagnosis of odontogenic, pulpal, and periapical pathology[30–35] (Figures 10-14 to 10-16)

Currently, CBCT imaging is generally recommended for endodontic cases in which conventional dental radiographs may not yield adequate information to allow

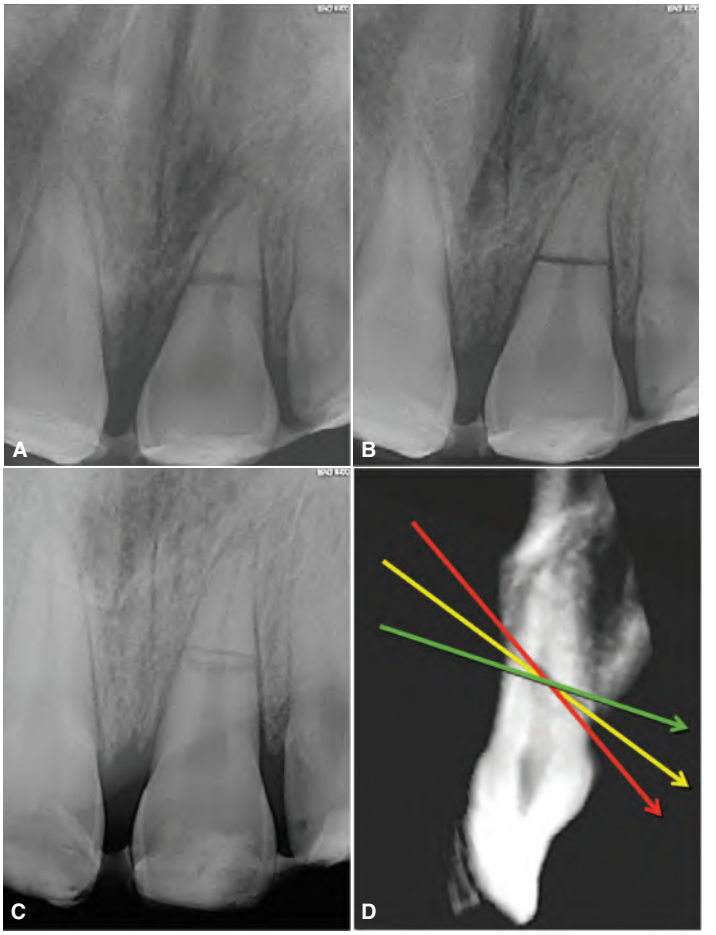

FIGURE 10-3 Importance of beam angulation in diagnosis of horizontal root fracture. ***A.*** Periapical radiograph with incorrect beam angulation of a maxillary central incisor that suffered dental trauma (red arrow in D). ***B.*** Correct beam angulation matching the line of fracture (yellow arrow in D). ***C.*** Incorrect beam angulation displaying the double line of fracture (green arrow in D). ***D.*** High resolution CBCT displaying the oblique fracture and the previously described radiographic angulations in A, B, and C (arrows). (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

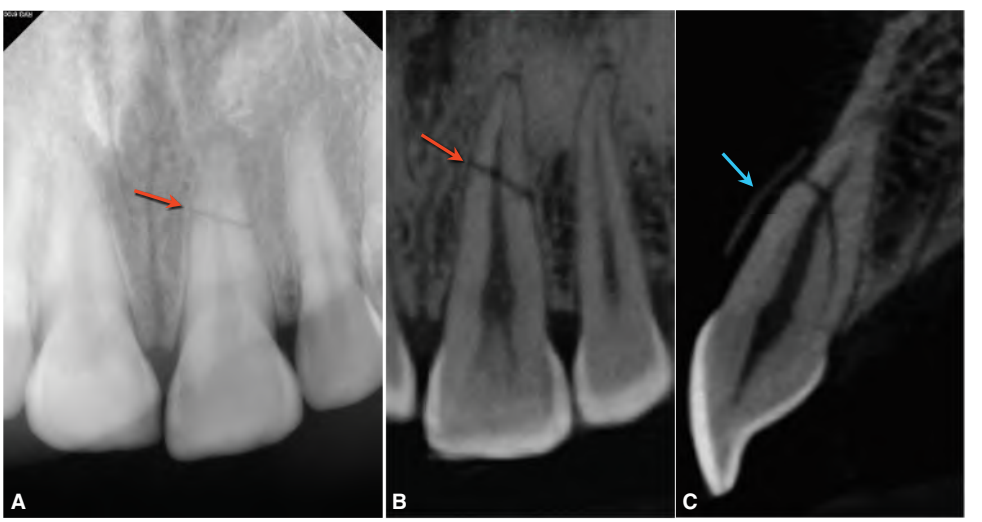

FIGURE 10-4 Horizontal fracture in the middle third of a maxillary central incisor after dental trauma. ***A.*** Periapical radiograph. Note fracture line (red arrow). ***B* and *C.*** Sections of a high resolution CBCT displaying a horizontal fracture extending from the palatal middle third to the buccal apical third; this fracture has a poor prognosis. Note, bony fracture with fragment separation in the buccal aspect (blue arrow). (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

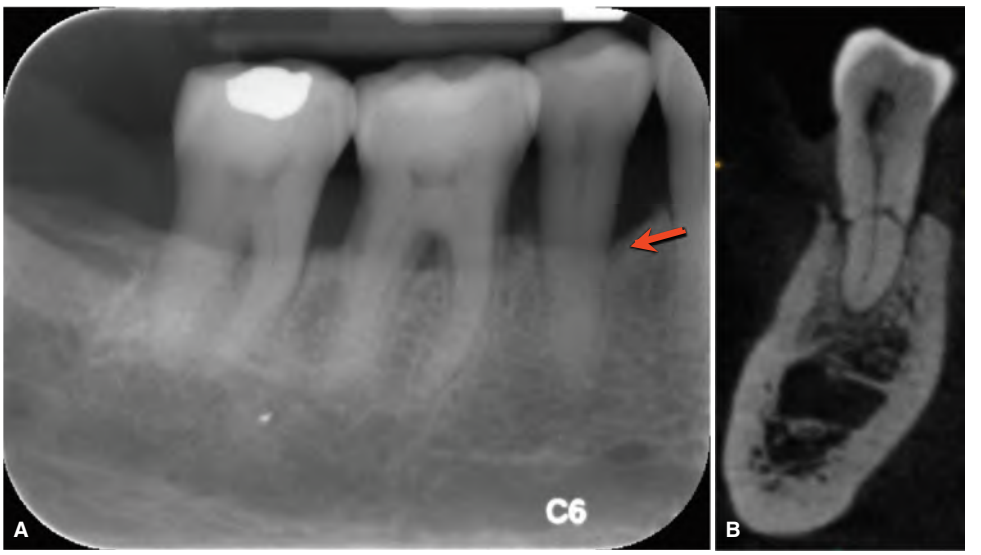

FIGURE 10-5 Mandibular right second premolar with clinical mobility and periodontal disease. ***A.*** Periapical radiograph. Note bone level around the involved tooth (red arrow). ***B.*** High resolution CBCT reveals a horizontal fracture near the middle third of the root. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

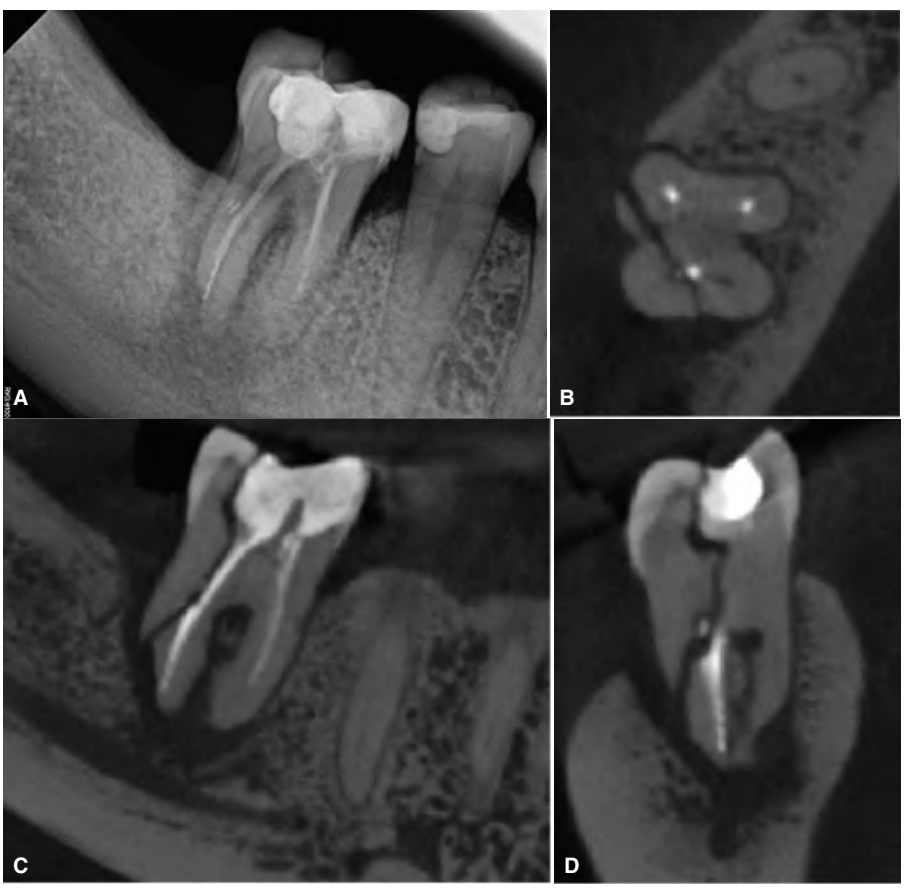

FIGURE 10-6 Vertical root fracture in a mandibular right first molar. ***A.*** Periapical radiograph. ***B.*** High resolution, 90 μm voxel size, CBCT axial view showing the fracture line. ***C.*** CBCT sagittal view. ***D.*** CBCT coronal view. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

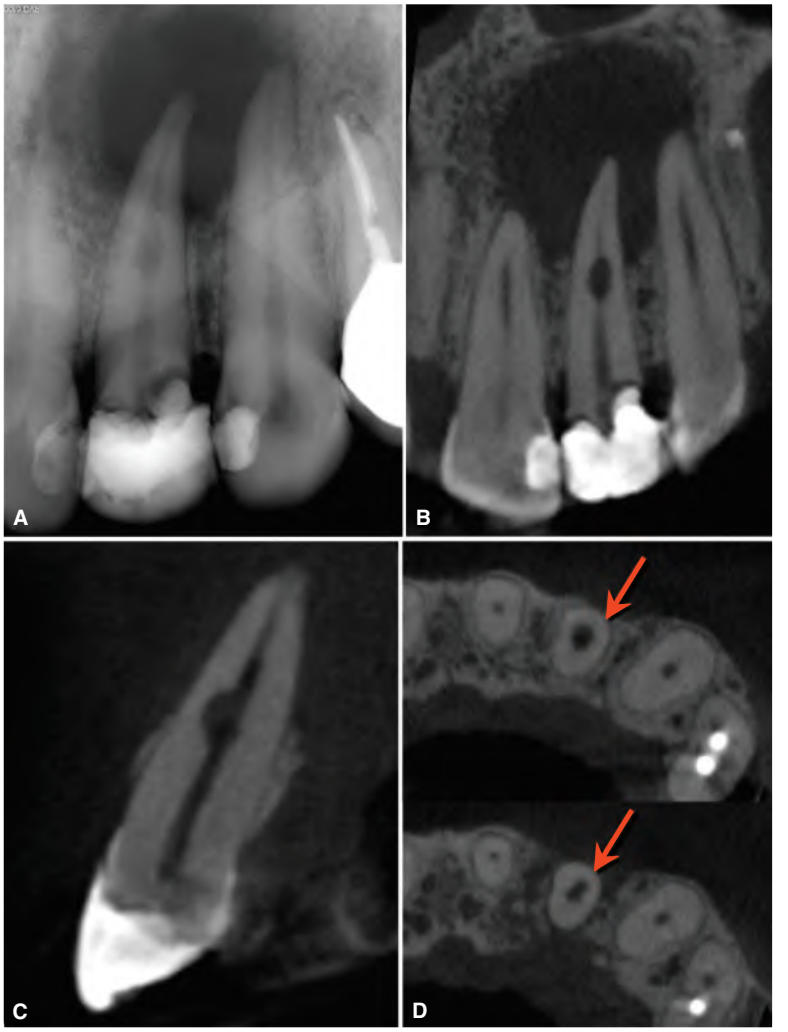

FIGURE 10-7 Internal resorption in a maxillary left lateral incisor. ***A.*** Periapical radiograph. ***B.*** High resolution, 90 µm voxel size, CBCT coronal view. ***C.*** CBCT sagittal view. ***D.*** CBCT axial views. Note the area and morphology of resorption near midroot level (red arrows). (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

appropriatevmanagement of the endodontic problem.[36] It should also be noted that the dental specialty of oral and maxillofacial radiology is most ideally trained, in general, to comprehensively evaluate and interpret CBCT images for dental applications, particularly when it comes to the diagnosis of potential pathology. When a CBCT scan is obtained, the entire volume and all anatomic structures in the field of view or acquisition should be reviewed for potential pathology.

RADIOGRAPHIC IMAGES OF LESIONS

In general, the most common conditions or pathology an endodontist may encounter via radiographs can be classified into *odontogenic* or *nonodontogenic* lesions. This pathology can also be further categorized by etiology as *developmental, reactive,* or *neoplastic*. Examples of odontogenic and nonodontogenic conditions within these etiologic categories are provided in Table 10-1.

RADIOGRAPHIC INTERPRETATION

It is recommended to apply a systematic approach to radiographic diagnosis and interpretation for all endodontic cases. In this chapter, clinical cases are used to highlight the process of imaging interpretation, particularly with reference to conditions that have endodontic relevance, many of which are listed in Table 10-1. Importantly, radiographic interpretation requires at least a basic knowledge of radiation physics and anatomy. Diagnosis and management of endodontic conditions require clinical evaluation and correlation of imaging findings with clinical findings. In some cases, this may include biopsy for more definitive histopathologic diagnosis, whereas in other cases, radiographs and clinical findings alone are sufficient.

Once a clinical assessment is made and appropriate images are acquired (of satisfactory diagnostic quality and with minimal artifacts), image analysis can begin. The key to accurate imaging diagnosis is to have a systematic and thorough approach to interpreting any image as well as also

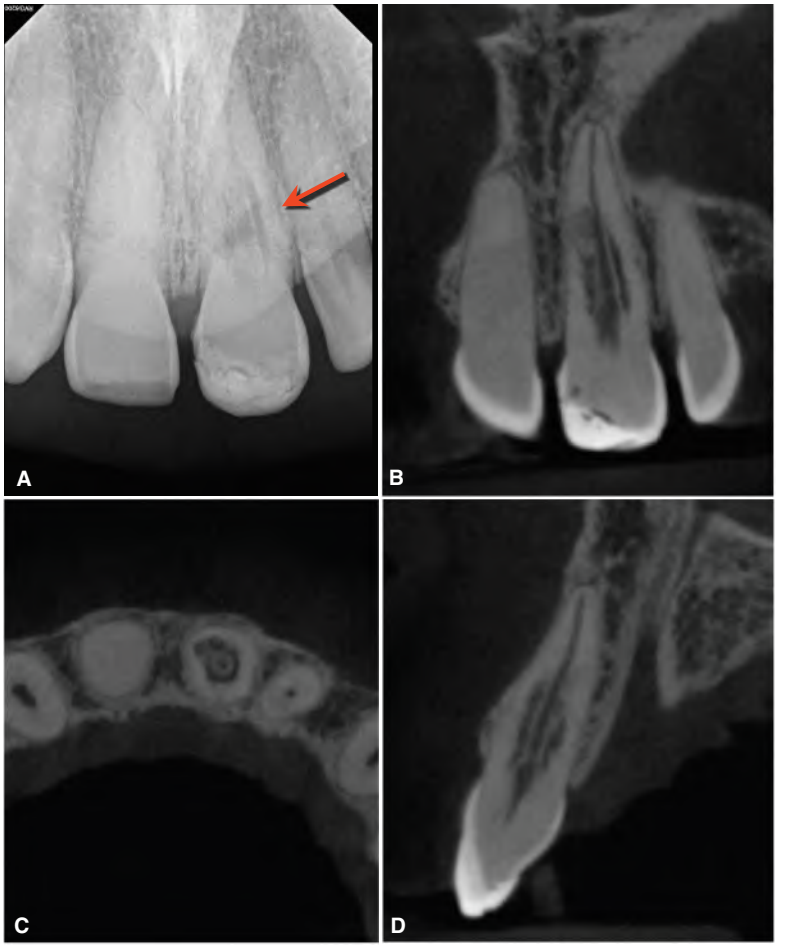

FIGURE 10-8 External root resorption in a maxillary left central incisor. ***A.*** Periapical radiograph. Red arrow points to the affected tooth. ***B.*** High resolution CBCT coronal view. ***C.*** CBCT axial view. ***D.*** CBCT sagittal view. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

appropriate viewing conditions. All visible anatomic structures should be evaluated to detect any potential abnormality. Since more than one abnormality may be present in a patient, all aspects of each image should be evaluated carefully in order to avoid missing important pathosis or failing to diagnose a significant condition. Improved imaging analysis and interpretation comes with practice and experience. The more normal radiographic anatomy one views, the more likely one may identify abnormalities (Figure 10-17). The exact approach recommended for accurate interpretation has been delineated in detail in textbooks on oral radiology.[37,38] In general, the steps involved include the following:

1. Systematic evaluation of normal anatomic structures on the radiograph or the entire CBCT volume, to look for lesions or abnormalities. When viewing conventional dental X-rays such as periapical or bitewing films, one should systematically observe each tooth in the image along with adjacent bone or other visible anatomic structures to look for changes suggestive of pathology. With a dental full mouth X-ray series there is even duplication of teeth in more than one image so one can observe from slightly different angles or representations the same tooth. This enables one to identify pathoses more accurately and avoid misinterpretation resulting from artifacts or superimposition of anatomic structures. When viewing new images, always start at the same place and then carefully trace each tooth's lamina dura for breaks or discontinuity in these structures.

   Conventional and digital dental radiography remain the imaging tools of choice for caries detection (Figure 10-18). In caries diagnosis, the endodontic challenge is to determine the spatial relationship between the carious lesion and the pulp.[39] On one hand, the notion is that proximal caries goes deeper than what can be seen in a conventional dental radiograph. On the other hand, caries located on the buccal or lingual aspect of the tooth may project over the pulp and give a false impression of pulpal involvement.[39] Additionally, interpretation of radiographic findings in restored teeth can present unique challenges.
2. When an abnormality, or pathosis, is suspected, there are specific parameters that should be evaluated in each case to achieve an accurate differential diagnosis (Table 10-2). These are the anatomic location and epicenter of the lesion, relative radiodensity (Figure 10-19), size

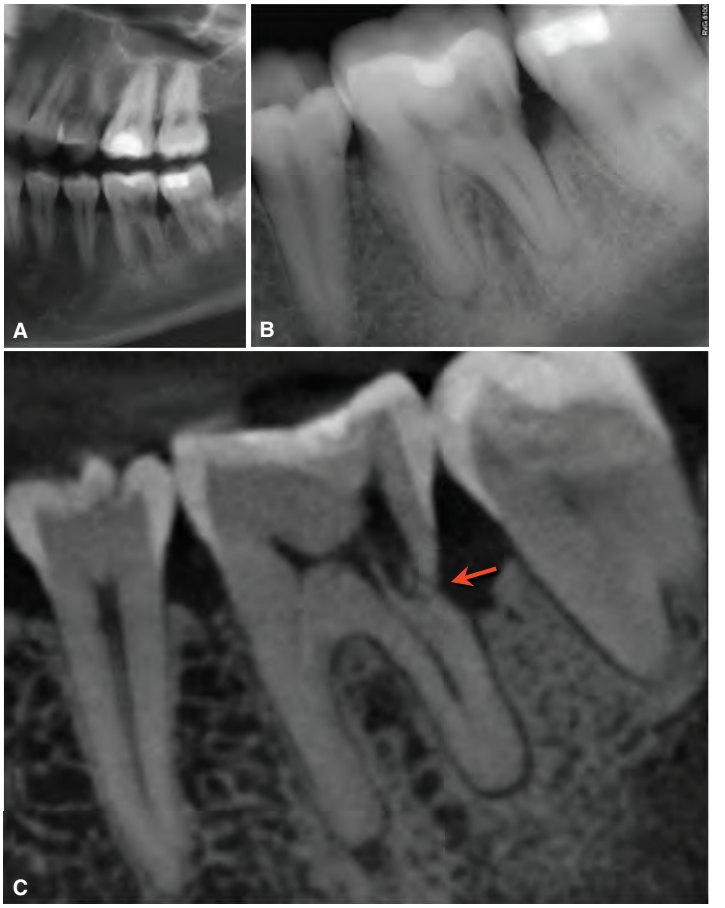

FIGURE 10-9 External resorption in a mandibular left first molar. ***A.*** Panoramic radiograph section displaying a lesion in the crown with the appearance of dental caries. ***B.*** Periapical radiograph. ***C.*** High resolution CBCT sagittal view of the entry point of the external resorption (red arrow). (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

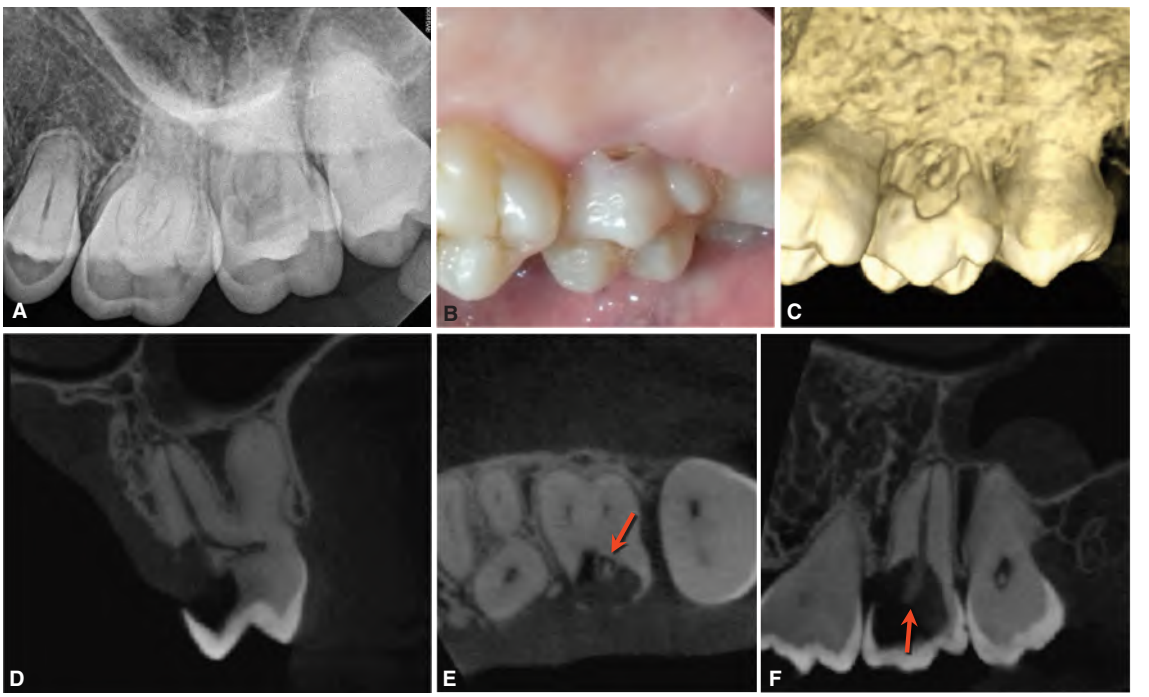

FIGURE 10-10 Invasive cervical resorption in a maxillary left second molar ***A.*** Periapical radiograph. ***B.*** Clinical view showing a defect in the palatal cervical area. ***C.*** Volumetric reconstruction of the area. ***D.*** High resolution CBCT coronal view. ***E* and *F.*** CBCT axial and sagittal views showing intact palatal canal (red arrows). (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

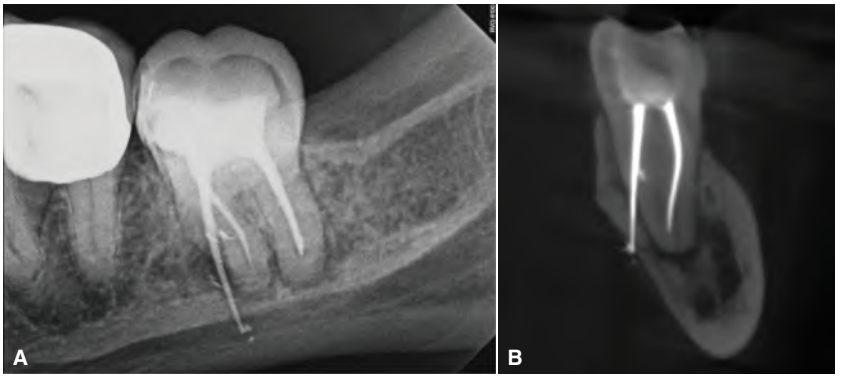

FIGURE 10-11 ***A.*** Periapical radiograph of a mandibular left second molar with gross canal overfill in the mesial root. ***B.*** High resolution CBCT coronal view showing perforation in the mesiolingual root and a perforation of the lingual cortical plate. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

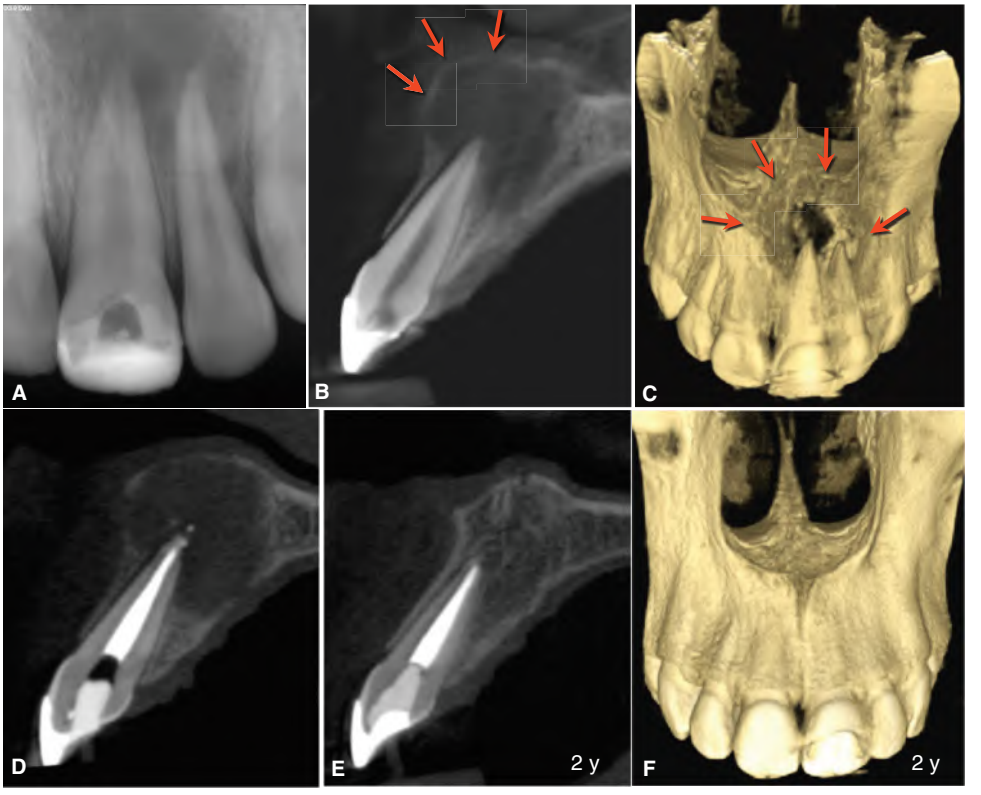

FIGURE 10-12 Asymptomatic *chronic apical periodontitis (*with a granulomatous lesion), in a maxillary left central incisor. ***A.*** Periapical radiograph. ***B.*** High resolution CBCT showing a large lesion with cortical plate expansion (red arrows). ***C.*** Volumetric reconstruction of high resolution CBCT showing extensive bone destruction (red arrows). ***D.*** CBCT sagittal view after nonsurgical endodontic therapy. ***E & F.*** CBCT images and volumetric reconstruction 2 years after treatment. Note evidence of bony healing and remodeling. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

and shape or growth pattern, internal structure, border characteristics, and the effects of the lesion on adjacent structures. These parameters are important to evaluate because they provide insight into pathophysiologic processes occurring at the tissue level that may also be observed at the radiographic level. This process is similar to that used in clinical diagnosis or histopathologic diagnosis to some extent.

For example, Figure 10-20 shows two hyperdense or radiopaque-appearing lesions that are very different in nature and pathogenesis. Clinically, in the case of *idiopathic osteosclerosis* the patient was asymptomatic and didn't present any clinical signs such as expansion of bone or nerve paresthesia. Pulp sensibility tests in the associated teeth were normal. Conversely, in the case of *fibrous dysplasia*, the patient had significant maxillary and mandibular bone expansion and paresthesia of the left inferior alveolar nerve. Multiple associated teeth did not respond to pulp sensibility tests. Radiographically, if we evaluate the parameters for each lesion as shown

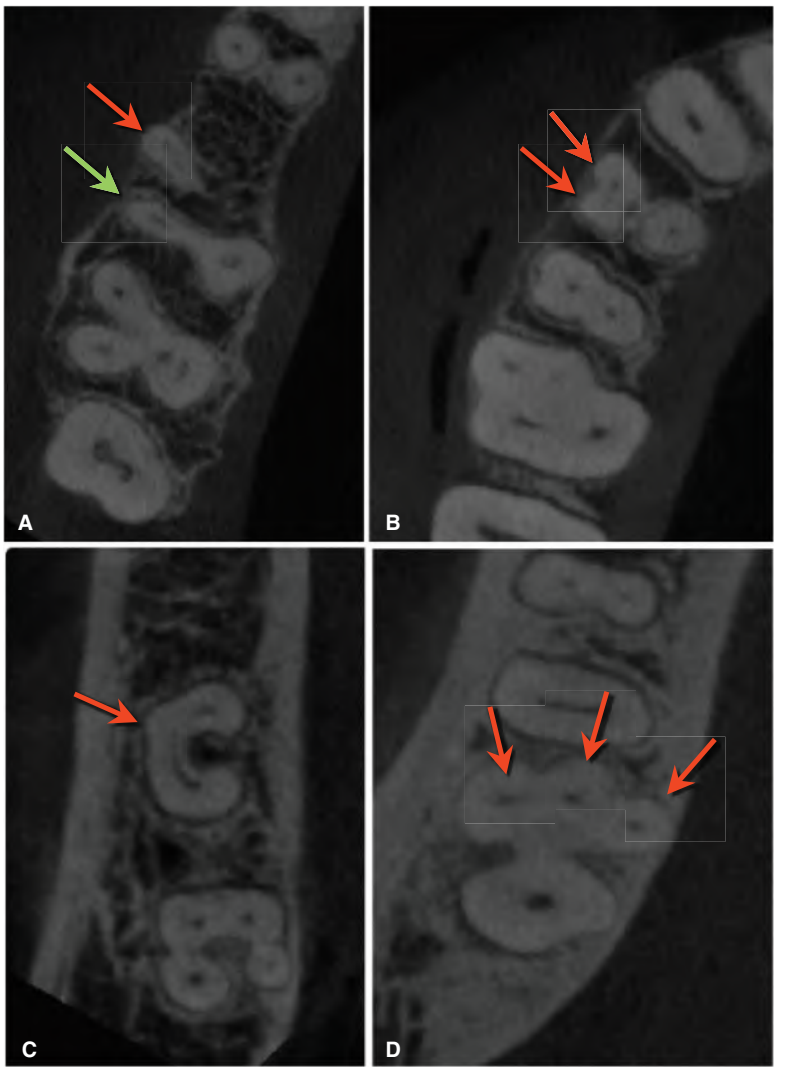

FIGURE 10-13 Axial (transverse) CBCT reconstructions are useful for characterizing root and canal morphology. ***A.*** A maxillary first molar with fused distobuccal and palatal roots; red arrow denotes mesial root, and green arrow denotes fused root. ***B.*** A maxillary first premolar with three separate canals; red arrows denote mesiobuccal and distobuccal roots. ***C.*** A mandibular second molar with "C" shape anatomy (red arrow). ***D.*** A mandibular third molar; red arrows denote fused mesial root with three separate canals. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

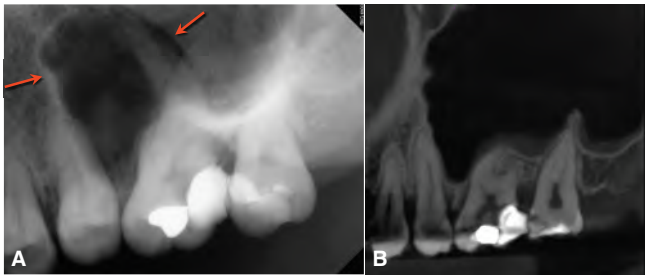

FIGURE 10-14 ***A.*** Periapical radiograph showing a radiolucency between the maxillary left second premolar and first molar (red arrows) mimicking a cyst-like process. ***B.*** High resolution CBCT sagittal view displaying the maxillary sinus between the maxillary left second premolar and first molar with no evidence of a lesion. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

in Table 10-2, in the case of *idiopathic* osteosclerosis the *anatomic location*, or epicenter, of the lesion is the mandibular bone near teeth apices, the *relative radiodensity* is that of dense cortical bone (see the inferior border of the same mandible for comparison), the *size* is smaller as compared to the other condition, the *shape, or growth pattern,* is relatively regular and uniform, the *internal structure* is homogenous and solid, the *borders* are relatively distinct with blending into adjacent tissue or bone, and there are *no effects on adjacent structures* such as teeth. In the case of *fibrous dysplasia*, radiographically, the *anatomic location*, or epicenter, of the lesion is bone in both jaws, the *relative radiodensity* is that of enamel compared to dense cortical bone, the *size* is much larger and multifocal as compared to *idiopathic osteosclerosis*, the *shape or growth pattern* is irregular, or nonuniform, the *internal*

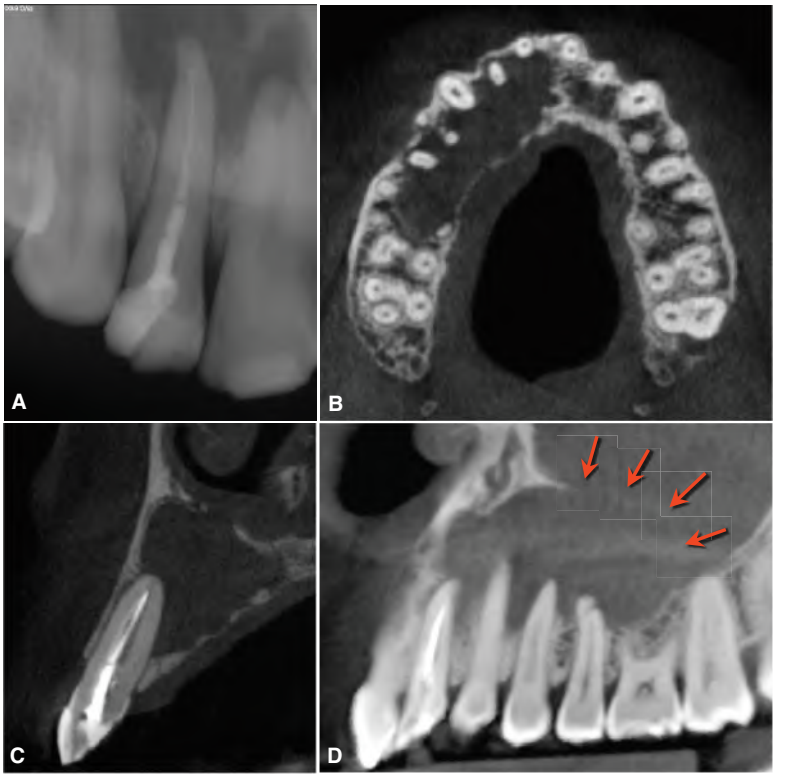

FIGURE 10-15 ***A.*** Periapical radiograph showing a periapical radiolucency associated with root-filled maxillary right lateral incisor. ***B.*** 180 µm voxel size CBCT axial view displaying the extensive bone damage extending from the lateral incisor to the second molar. ***C.*** 90 µm voxel size CBCT coronal view displaying obturation defect. ***D.*** 180 µm voxel size CBCT sagittal view displaying extension into the maxillary sinus (red arrows). (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

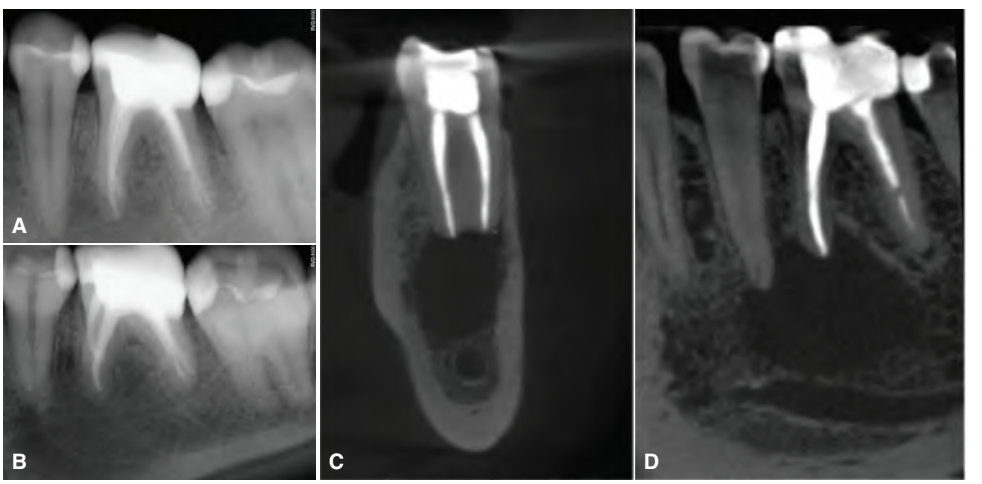

FIGURE 10-16 ***A & B.*** Periapical radiographs from two different angles do not overtly reveal the pathosis seen in the next image. ***C & D.*** Coronal and sagittal CBCT images of same tooth showing a well-defined periapical radiolucency, the anatomic extent of the lesion, relationship to the inferior alveolar canal and involvement of the adjacent premolar. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

structure is heterogeneous and solid with "cotton wool" appearance, the *borders* are also blending but indistinct, and there are major *effects on adjacent structures*.

Although these conditions are both in the spectrum of bone pathology, the differences in the evaluated parameters indicate that they are very different with respect to diagnosis and behavior (which is what we see clinically and radiographically with both conditions) and these findings, therefore, impact differential diagnosis and management in each case. In these specific cases, the *fibrous dysplasia* patient required more definitive diagnosis, including biopsy and systemic workup, to rule out other osseous involvement or a syndromic-related form of *fibrous dysplasia*. It also required more complex

TABLE 10-1 Odontogenic and Nonodontogenic Conditions That Can Be Encountered During Endodontic Imaging Interpretation

Pathology	Developmental	Reactive	Neoplastic
Odontogenic lesions	Amelogenesis imperfecta Dentin dysplasia Dentinogenesis imperfecta Dens in dente/invaginatus Dilaceration Enamel pearl Fusion Gemination Hypercementosis Mesiodens Supernumerary roots Systemic syndromes Tauradontism Turner's hypoplasia	Apical root resorption Apical scar Calcified root canal Caries Chronic apical periodontitis External resorption Internal resorption Pulpal calcification Pulpitis Periapical granuloma Periapical abscess Radicular cyst Root perforations Tooth/root fractures	Ameloblastoma Ameloblastic fibroma Calcifying odontogenic cyst Calcifying odontogenic tumors Cementoblastoma Cementoma Glandular odontogenic cyst Keratocystic odontogenic tumor Odontogenic fibroma Odontogenic myxoma Odontoma
Nonodontogenic lesions	Antral pseudocyst Canalis Sinuosus Dentigerous cyst Idiopathic osteosclerosis Incisive canal cyst Lateral periodontal cyst Pneumatized sinus Systemic syndromes Tori/exostoses	Ankylosis Antral Pseudocyst Metabolic bone pathology Osteomyelitis Osteonecrosis Periodontitis Periostitis Sinusitis Traumatic bone cavity	Fibro-osseous lesions Cemento-osseous dysplasia Florid osseous dysplasia Fibrous dysplasia Ossifying fibroma Central giant cell granuloma Hemangioma Primary malignancy Metastatic cancer

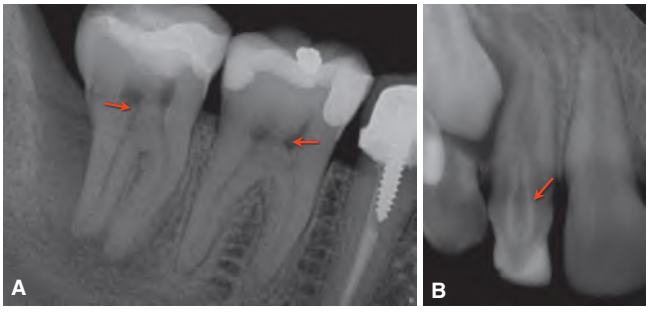

FIGURE 10-17 ***A.*** Identification of *pulpal calcification* (red arrows), or stones, in mandibular molars. ***B.*** Presence of a *dens invaginatus* (red arrow) can only be made with knowledge of normal tooth and pulpal anatomy as viewed on radiographs. Teeth affected by this developmental anomaly can present endodontic treatment challenges. (Courtesy of Dr. Reham Sharroufna, Los Angeles, CA. (Figure 10-17A) and Dr. Carl M. Allen, Columbus, OH. (Figure 10-17B).)

surgical management and long-term follow-up. The *idiopathic osteosclerosis* patient didn't require any treatment; only periodic reevaluation. This highlights the importance of clinico-radiographic findings for diagnosis, management, and outcome. Even with more subtle or challenging cases, the rules of imaging interpretation still apply and should be utilized.

3. Once lesion parameters (Table 10-2) have been characterized radiographically and correlated with clinical findings, it is important to identify the potential etiology of the lesion as *developmental*, *reactive*, or *neoplastic*. Most organic pathoses can be categorized into one (or more) of these three etiologic classifications.

Developmental conditions, in general, tend to be hereditary or acquired, usually manifest early in life, and have a symmetric and bilateral/diffuse distribution or a midline distribution based on head and neck embryologic patterns. Examples may include head and neck syndromes (Figure 10-21), tori (Figure 10-22), incisive canal cyst (Figure 10-23), dentinogenesis imperfecta (Figure 10-24), and dentinal dysplasia (Figure 10-25).

Reactive conditions are generally inflammatory in nature, where the body is reacting to either exogenous or endogenous irritants. This group may include infections, autoimmune conditions, trauma, and foreign body reactions. Clinically, one or more of the classic signs of inflammation such as redness, heat, swelling, pain, or loss of function may exist; although not in all cases. Examples may include acute or chronic apical periodontitis (Figure 10-26), inflamed radicular or periapical cyst (Figure 10-27), antral pseudocyst (Figure 10-28), traumatic bone cavity (Figure 10-29), and osteomyelitis (Figure 10-30).

Neoplastic conditions are generally benign or malignant in nature. Benign tumors are usually well-defined and slowly growing as compared to cancers that can grow more rapidly and present as ill-defined radiographic lesions. In the jaws and around teeth, examples of benign neoplasms encountered include odontogenic tumors such as odontoma (Figure 10-31), ameloblastoma (Figure 10-32) and odontogenic myxoma

TABLE 10-2 Parameters and Potential Pathologic Features That Should Be Evaluated When Making Endodontic Imaging Interpretation

Imaging Parameters	Potential Pathologic Features
Anatomic location and epicenter of lesion	Unifocal or multifocal; localized or generalized; unilateral or bilateral; association with anatomic structures such as teeth, nerve canals, and sinuses
Relative radiodensity of lesion	Lucent (lytic), opaque, or mixed; soft tissue or hard tissue density
Size, shape, and growth pattern	Regular or irregular shape; uniform or nonuniform; hydraulic/cystic appearance or solid; expansile or nonexpansile; permeating; infiltrative
Internal structure	Homogenous or heterogeneous; solid or cystic; scalloped or circular border; punched-out; septated; sequestrum formation; dystrophic calcifications; ground glass or cotton wool appearance
Border characteristics	Well-defined or poorly defined margins; distinct or indistinct margins; blending margins; sclerotic or corticated periphery; lobular; perforation
Effects on adjacent structures	Resorption of teeth or bone; lamina dura widening or destruction; reactive bone formation; periosteal reaction; space-occupying; displacing

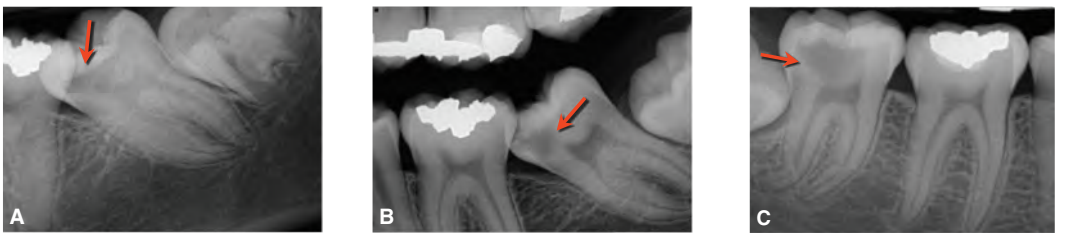

FIGURE 10-18 ***A & B.*** Conventional dental radiographs show *caries* lesion (red arrows) and its relationship with the pulp. ***C.*** *Caries* lesion (red arrow) appears to extend toward the distal pulp horn of this mandibular second molar. (Courtesy of Dr. Jaydeep Talim, Los Angeles, CA.)

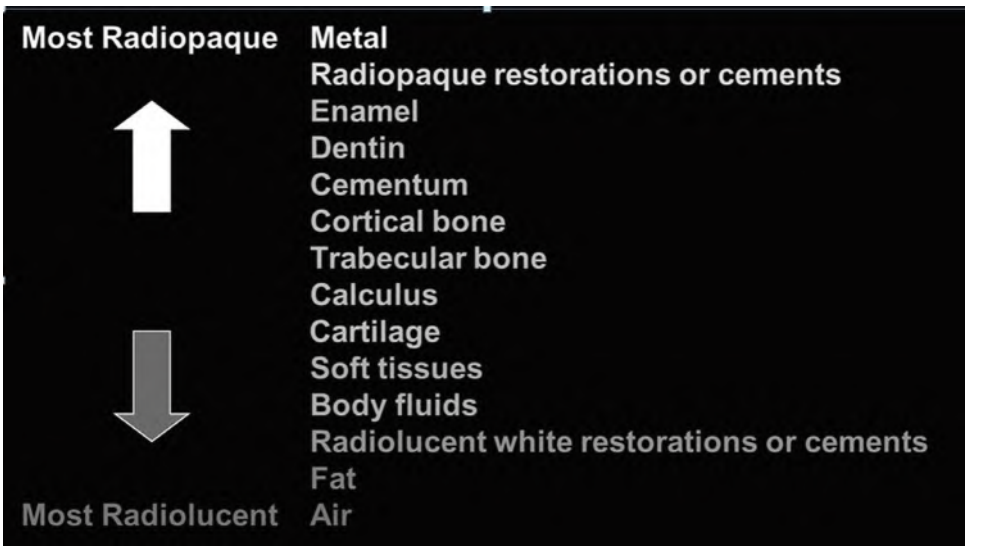

FIGURE 10-19 Relative radiodensity scale showing the most radiopaque appearing or high density objects (top) to the most lucent-appearing objects (bottom). (Courtesy of Dr. Ernest W. Lam, Toronto, Canada.)

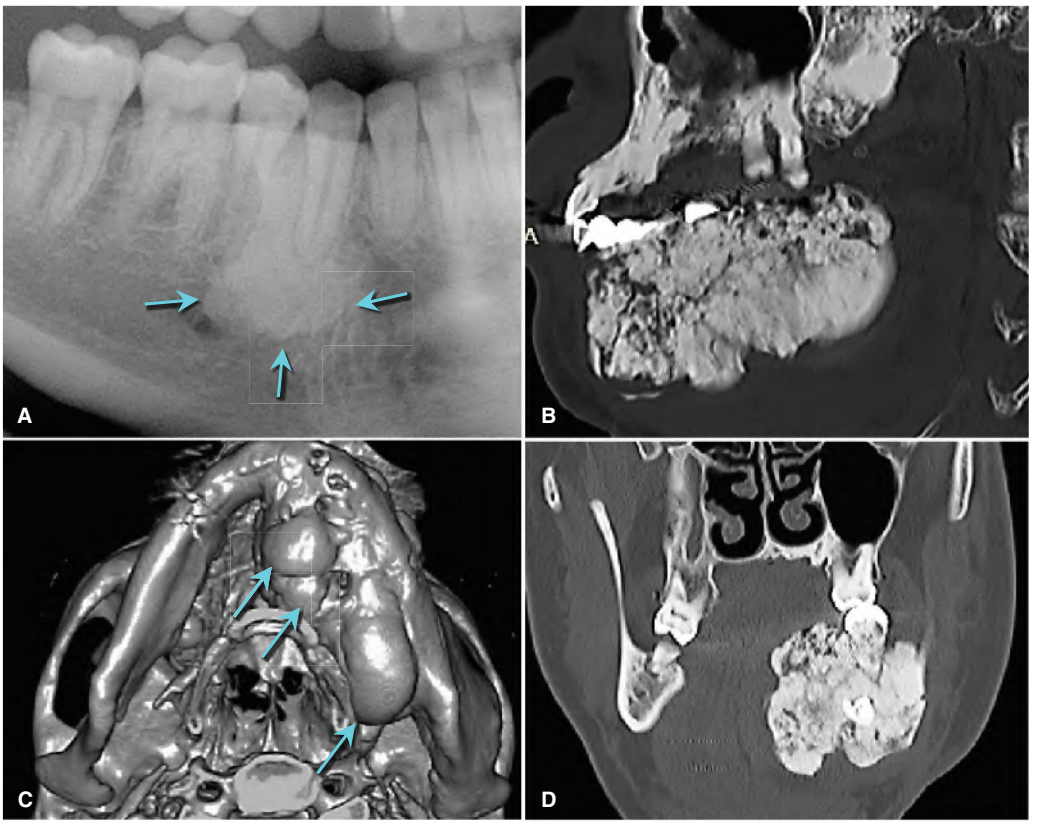

FIGURE 10-20 ***A.*** Panoramic radiographic section demonstrating a case of *idiopathic osteosclerosis* (blue arrows). ***B***. CBCT reconstructions demonstrate a case of *fibrous dysplasia* (polyostotic); coronal view. ***C***. Volumetric 3-D reconstructions. Blue arrows highlight the significant mandibular involvement and growth. ***D***. CBCT Sagittal view. (Courtesy of Dr. Parish P. Sedghizadeh, Los Angeles, CA.)

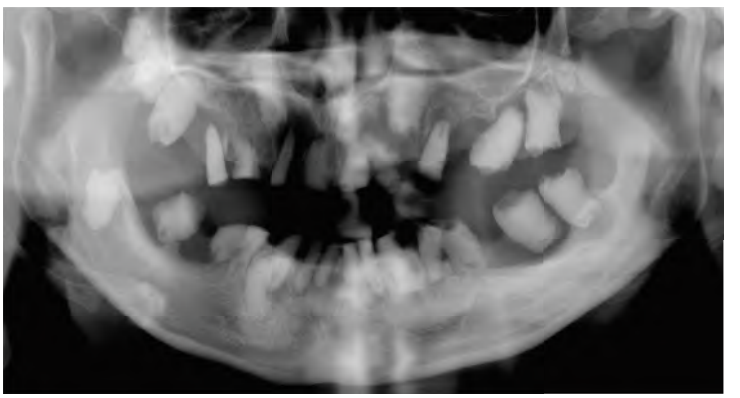

FIGURE 10-21 ***A.*** 14-year old boy with hereditary disease of *ectodermal dysplasia* who also had dermatologic and oral manifestations. Oral findings revealed abnormal tooth development including obliterated pulp canals, and secondary infections of the teeth and periodontium. Note the characteristic developmental presentation of diffuse and bilateral involvement of this condition. (Courtesy of Dr. Parish P. Sedghizadeh, Los Angeles, CA.)

(Figure 10-33), and benign fibro-osseous tumors (Figure 10-34). Examples of malignant neoplasms include primary or metastatic cancers (Figure 10-35).

4. The final step, radiographically, is to formulate a *differential diagnosis* based on the anatomic and pathologic findings in correlation with clinical findings. Figures 10-36 and 10-37 demonstrate two clinical examples of this process. Avoid making a diagnosis based on any single finding, whether it is clinico-pathologic or radiographic. Accuracy in diagnosis increases when multiple factors are being considered instead of only one (Figures 10-38 and 10-39)

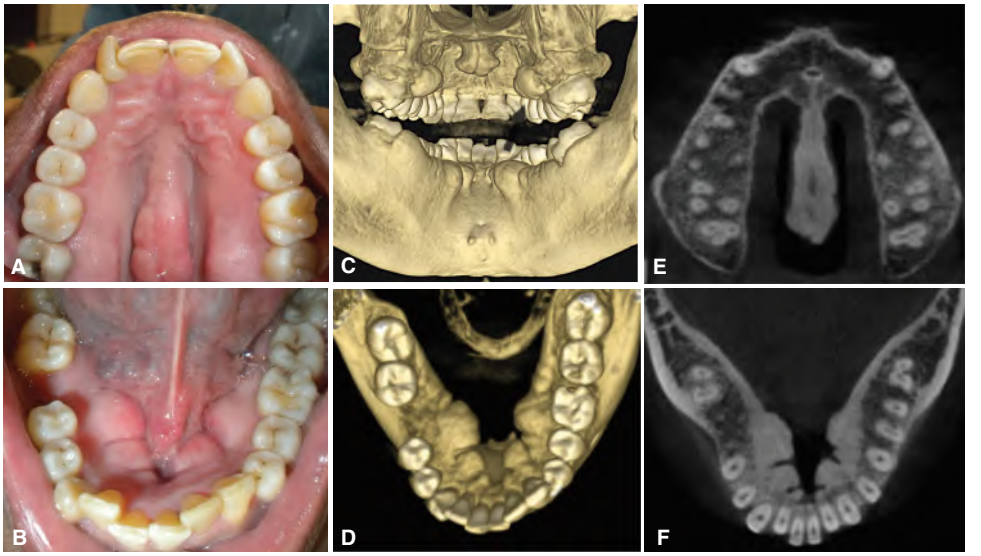

FIGURE 10-22 Developmental *tori*. ***A & B.*** Clinical images. ***C & D.*** volumetric 3-D reconstructions. ***E & F.*** Axial views. Note the midline and symmetric distribution of the palatal torus and the bilateral symmetric distribution of the mandibular tori that may be characteristic findings for developmental conditions. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

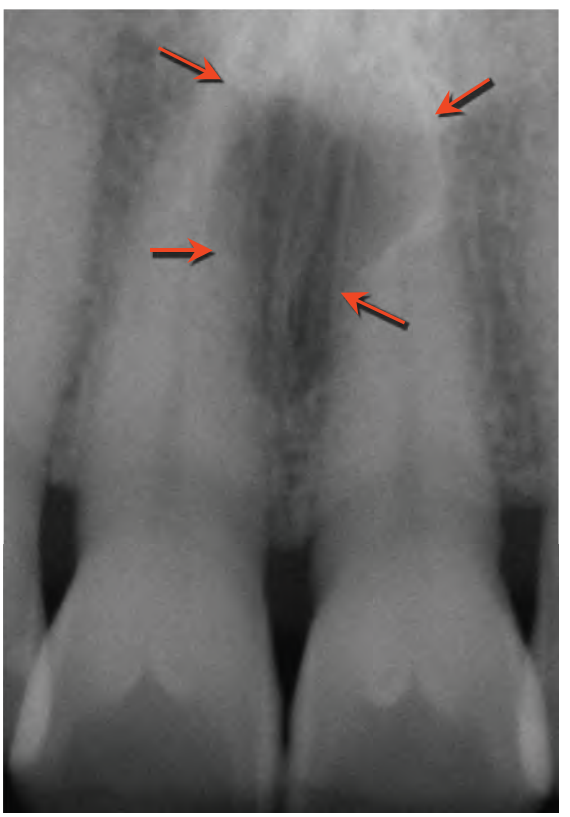

FIGURE 10-23 Periapical radiograph showing an *incisive canal cyst* (red arrows) also known as nasopalatine duct cyst. The midline location and relatively well-defined and symmetric appearance are highly suggestive of a developmental condition associated with normal anatomic structures in this region. These lesions are often described as "pear-shaped" or "heart-shaped" and are usually more than 6mm in diameter. (Courtesy of Dr. Parish P. Sedghizadeh, Los Angeles, CA.)

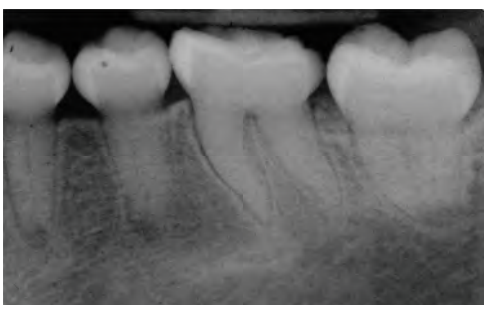

FIGURE 10-24 Periapical radiograph of a patient with *dentinogenesis imperfecta* showing characteristic findings such as bulbous crowns, cervical constriction, and obliterated pulp canals and chambers. (Courtesy of Dr. Carl M. Allen, Columbus, OH.)

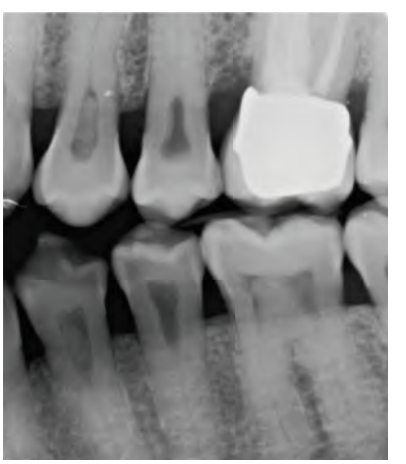

FIGURE 10-25 A bitewing radiograph of a patient with *dentinal dysplasia*, type II (coronal). Note the characteristic 'thistle-tube' appearance of the pulp chambers and presence of pulp stones in several teeth. (Courtesy of Dr. Carl M. Allen, Columbus, OH.)

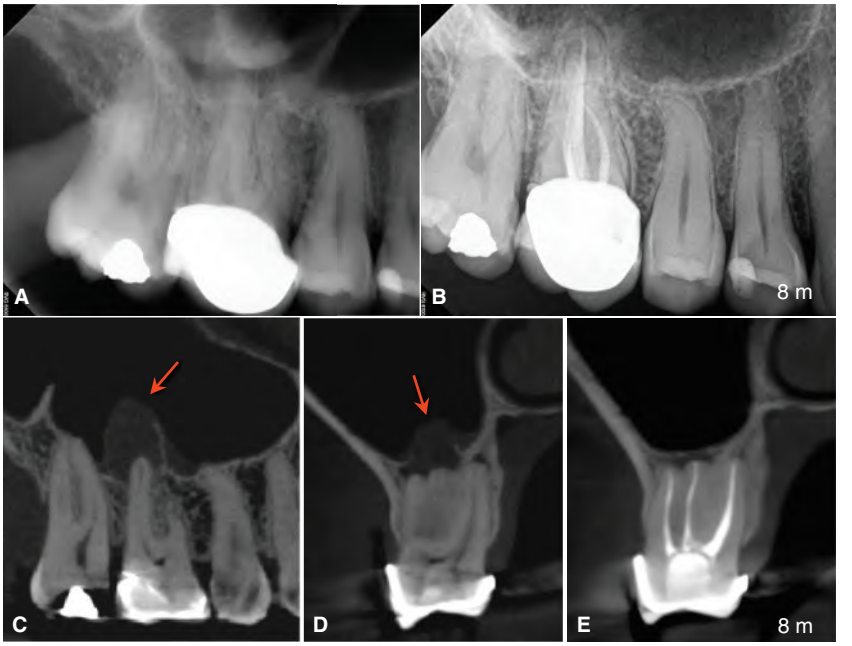

FIGURE 10-26 ***A.*** Periapical radiograph of the maxillary right first molar with asymptomatic *apical periodontitis*. Periapical radiolucency is not visible. The tooth underwent root canal treatment. ***B.*** Eight-month follow-up radiograph. ***C & D.*** 90 µm voxel size CBCT sagittal (C) and coronal (D) views, displaying periapical lesion invading the maxillary sinus (red arrows). ***E.*** Eight-month follow-up CBCT image showing evidence of healing of the lesion. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

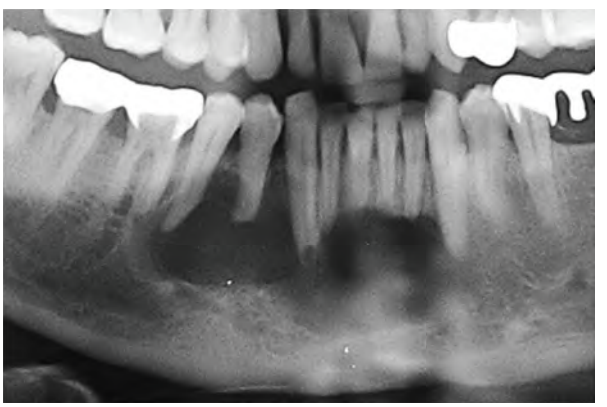

FIGURE 10-27 Panoramic radiographic section showing a large radiolucent lesion of the anterior mandible that was consistent with a large *periapical* (*radicular*) *cyst* diagnosed after surgical enucleation and histopathologic analysis. (Courtesy of Dr. Parish P. Sedghizadeh, Los Angeles, CA.)

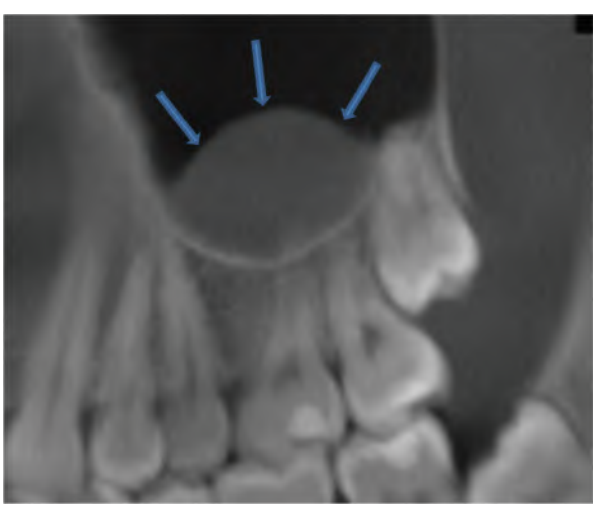

FIGURE 10-28 A section of a panoramic reconstruction from a CBCT scan showing a well-defined, dome-shaped, homogenous and faint radiopacity consistent with an *antral pseudocyst* (aka: sinus mucocele). This is a common radiographic finding in the sinus and is usually clinically inconsequential. Also, in this case, there were no signs or symptoms associated with this lesion and the teeth and pulps were normal. Lack of a corticated rim of bone (arrows) is highly suggestive that this does not represent a periapical cyst. Compare this image to the case of periapical cyst shown in Figure 10-26C & D where a thin rim of corticated bone can be seen at the periphery of the cyst lifting up from the floor of the sinus. Because antral pseudocysts arise from the lining of the sinus they do not alter or lift the antral bone as seen here. (Courtesy of Dr. Parish P. Sedghizadeh, Los Angeles, CA.)

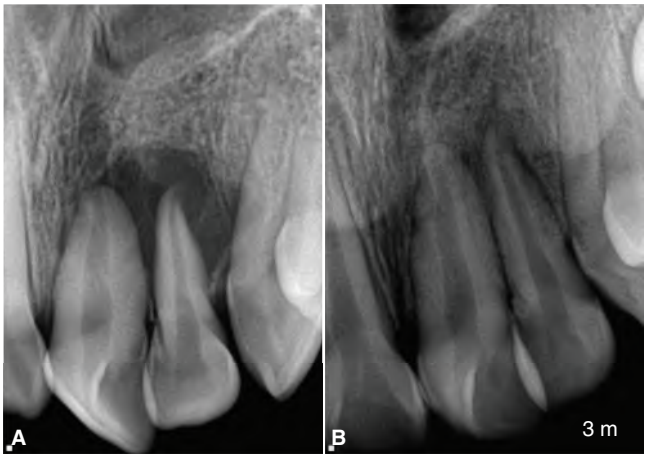

FIGURE 10-29 ***A.*** Periapical radiograph showing an apical radiolucency in a 12-year-old boy. Patient complained of pain and reported a history of trauma to the anterior maxilla. The associated apical radiolucency is most likely consistent with a *traumatic bone cavity* but could represent other conditions such as radicular cyst, periapical granuloma, or an odontogenic tumor (based on radiographic features alone without biopsy) All anterior teeth tested normal to pulp sensibility tests as compared to unaffected control teeth. ***B.*** Periapical radiograph, taken 3 months after initial presentation, showing bone-fill in the region following conservative treatment with antibiotics and close monitoring. The patient was asymptomatic within 2 weeks. On 1-year recall, the area remained healthy. (Courtesy of Dr. Diego Dalla-Bona, Los Angeles, CA.)

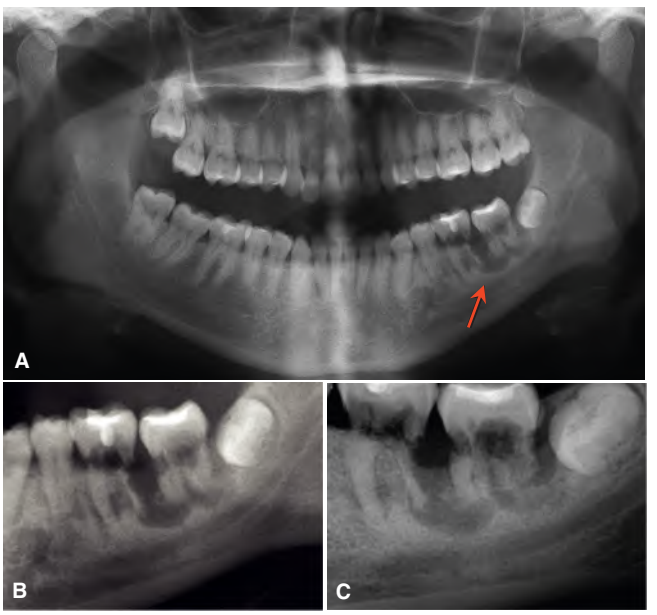

FIGURE 10-30 ***A.*** Panoramic radiograph showing an ill-defined lytic lesion (red arrow) of the left posterior mandible. A diagnosis of *osteomyelitis* (actinomycotic) was confirmed following extraction of the affected molars and histopathologic analysis. ***B & C.*** Higher magnification of the affected area reveals significant tooth and root resorption and irregular bone loss (often consistent with a diagnosis of infection, or sometimes even malignancy). (Courtesy of Dr. Tomoko Wada, Los Angeles, CA.)

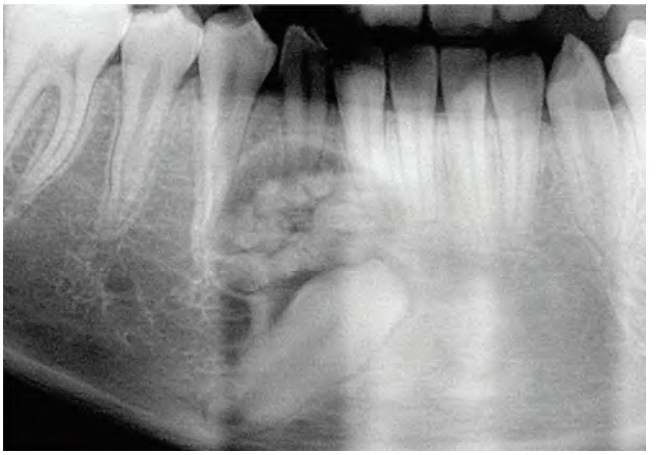

FIGURE 10-31 Panoramic radiographic section showing a well-defined mixed lesion with internal calcifications, circumferential radiolucency, and a thin sclerotic rim or periphery that is highly suggestive of a benign process. An impacted tooth is seen in association with this lesion. After surgical removal and histopathologic analysis, a diagnosis of *compound odontoma* was confirmed. (Courtesy of Dr. Saravanan Ram, Los Angeles, CA.)

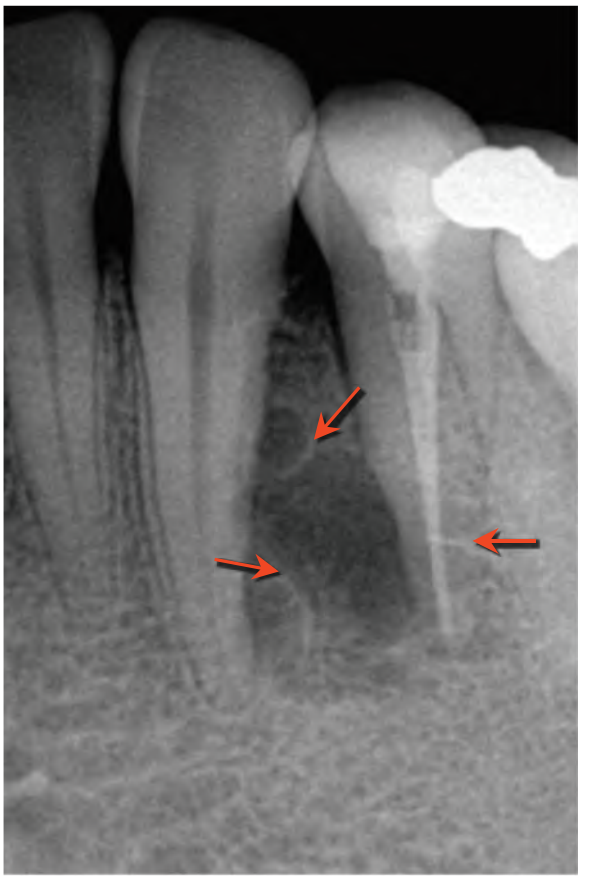

FIGURE 10-32 Periapical radiograph showing a multilocular radiolucency confirmed as *ameloblastoma* following biopsy and histopathologic evaluation. Note loss of lamina dura and potential resorption of adjacent roots and the fine internal septations (red arrows) indicating a multilocular growth process. (Courtesy of Dr. Parish P. Sedghizadeh, Los Angeles, CA.)

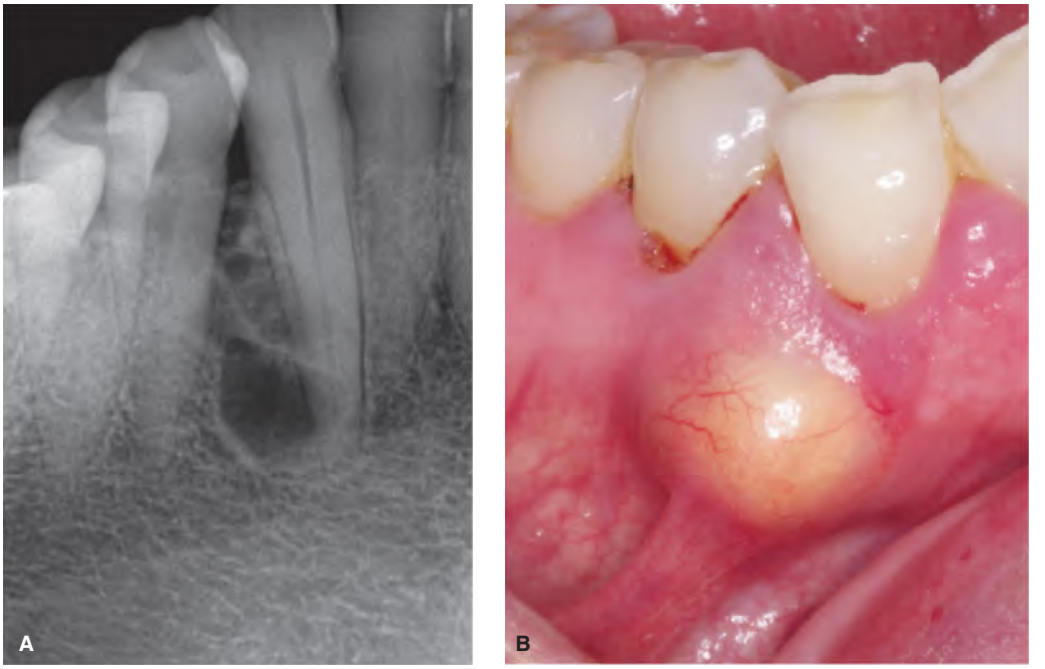

FIGURE 10-33 ***A.*** Periapical radiograph showing a multilocular radiolucency. ***B.*** Clinically, a buccal expansion associated with the intraosseous lesion is noted. Surgical enucleation and biopsy confirmed a diagnosis of *odontogenic myxoma*. Compare this case to the one described in Figure 10-32. The multilocular nature of the process can be suggestive of an odontogenic tumor in general. (Courtesy of Dr. Tomoko Wada, Los Angeles, CA.)

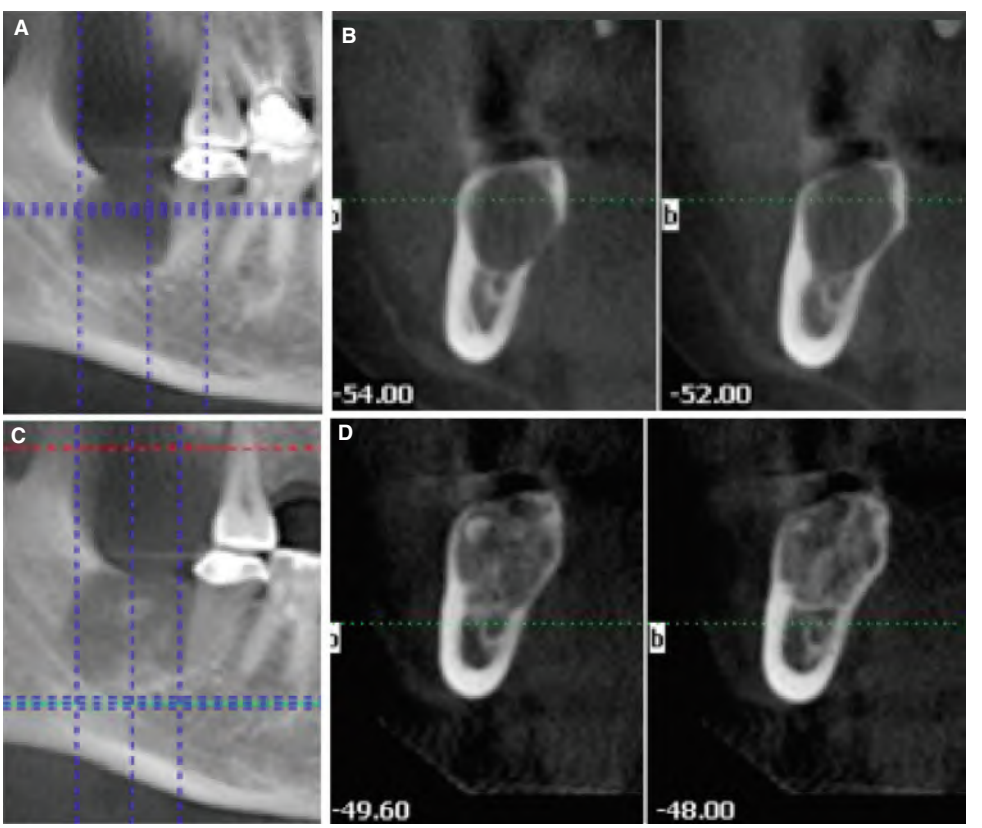

FIGURE 10-34 ***A.*** Panoramic reconstruction from a CBCT scan showing a well-defined radiolucency of the posterior mandible in an asymptomatic patient. ***B.*** Sagittal views reveal the expansile nature of the lesion and thinning of the cortical bone associated with this process. ***C & D.*** CBCT images taken 1 year later, showing internal calcification of the lesion and a more complex appearance than initial findings. Biopsy of the lesion confirmed a *benign fibro-osseous tumor* that explains the mixed radiographic appearance and well-defined nature of the process. (Courtesy of Dr. Yuan-Lung Hung, Los Angeles, CA.)

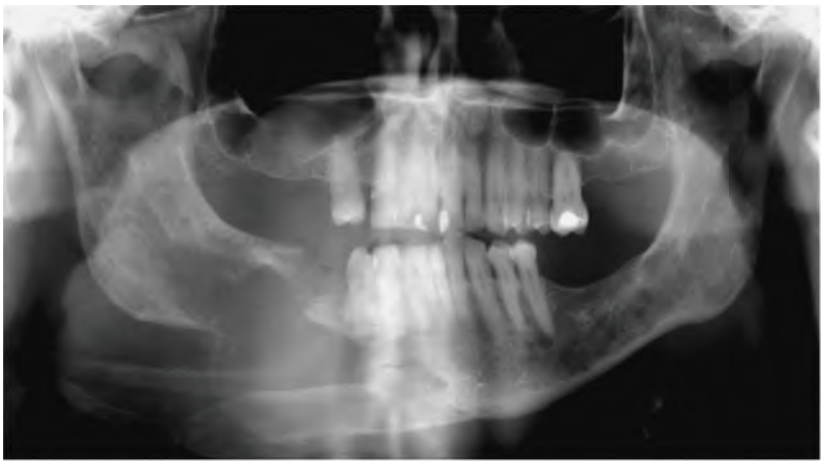

FIGURE 10-35 Panoramic radiograph of a healthy 67-year-old man complaining of pain on the right side of the mandible, presumably from odontogenic origin. The ill-defined radiolucency, or lytic nature of the lesion, and the presence of pathologic fracture is worrisome, warranting immediate biopsy and subsequent histopathologic evaluation that confirmed malignancy of *multiple myeloma*. Note the "punched-out" radiolucent lesions throughout the mandible that can be characteristic (but not pathognomonic) of multiple myeloma. (Courtesy of Dr. Parish P. Sedghizadeh, Los Angeles, CA.)

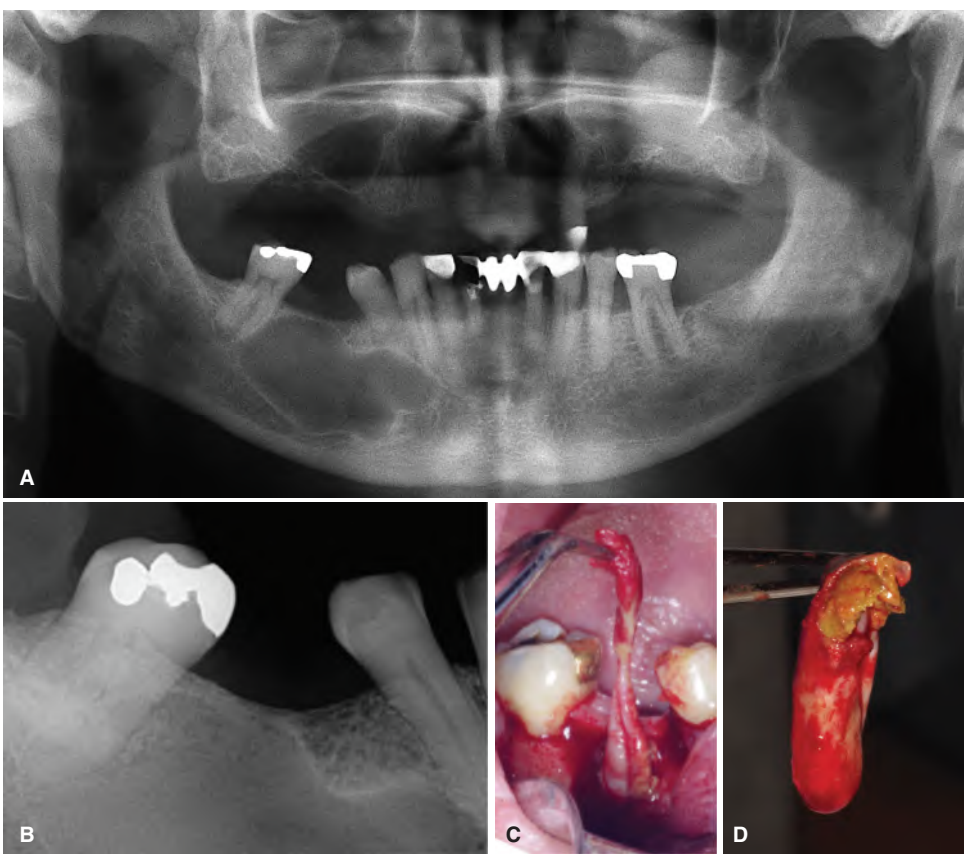

FIGURE 10-36 ***A.*** Panoramic radiograph showing a well-defined radiolucency of the right side of the mandible with a sclerotic rim, or border. The patient exhibited buccal expansion and complained of pain associated with the lesion. ***B.*** Conventional radiograph of the same lesion shows homogenous density in its internal part. ***C.*** Surgical removal of the lesion. Note, cystic appearance of the lesion. ***D.*** The surgical specimen has a sac-like appearance with keratinaceous debris oozing out superiorly. The clinical, surgical, and radiographic findings suggest a benign and cystic process that could include any number of odontogenic cysts or tumors as well as bony cysts or tumors. In this case, histopathologic diagnosis confirmed a *glandular odontogenic cyst*. (Courtesy of Dr. Parish P. Sedghizadeh, Los Angeles, CA.)

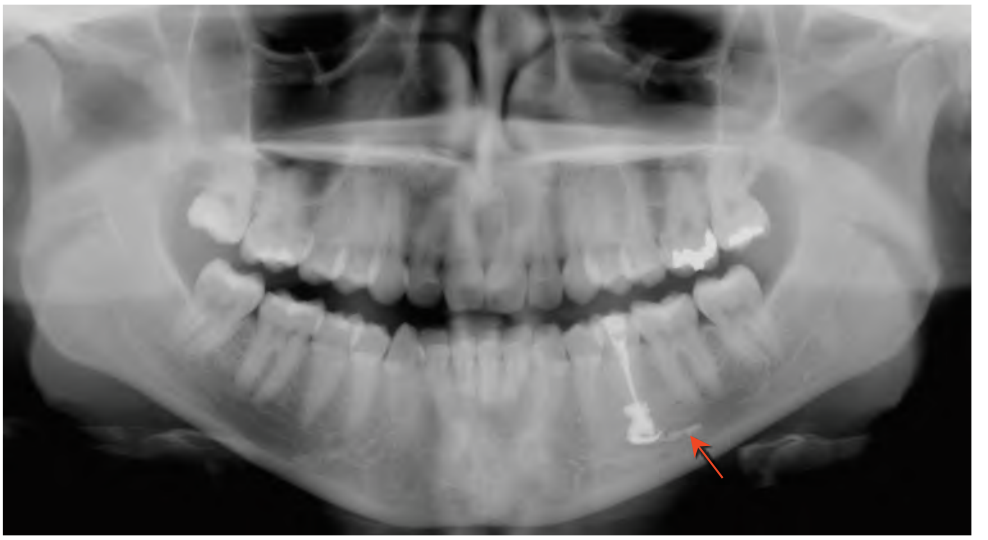

FIGURE 10-37 Panoramic radiograph of a patient complaining of pain and paresthesia/dysesthesia of the left side of the mandible and lower lip, shortly after root canal treatment. Note extrusion of radiopaque material into the inferior alveolar canal, extending several millimeters posteriorly (red arrow). This can explain the patient's chief complaint and neuropathic pain. Clinical symptoms correlated with radiographic findings. (Courtesy of Dr. Parish P. Sedghizadeh, Los Angeles, CA.)

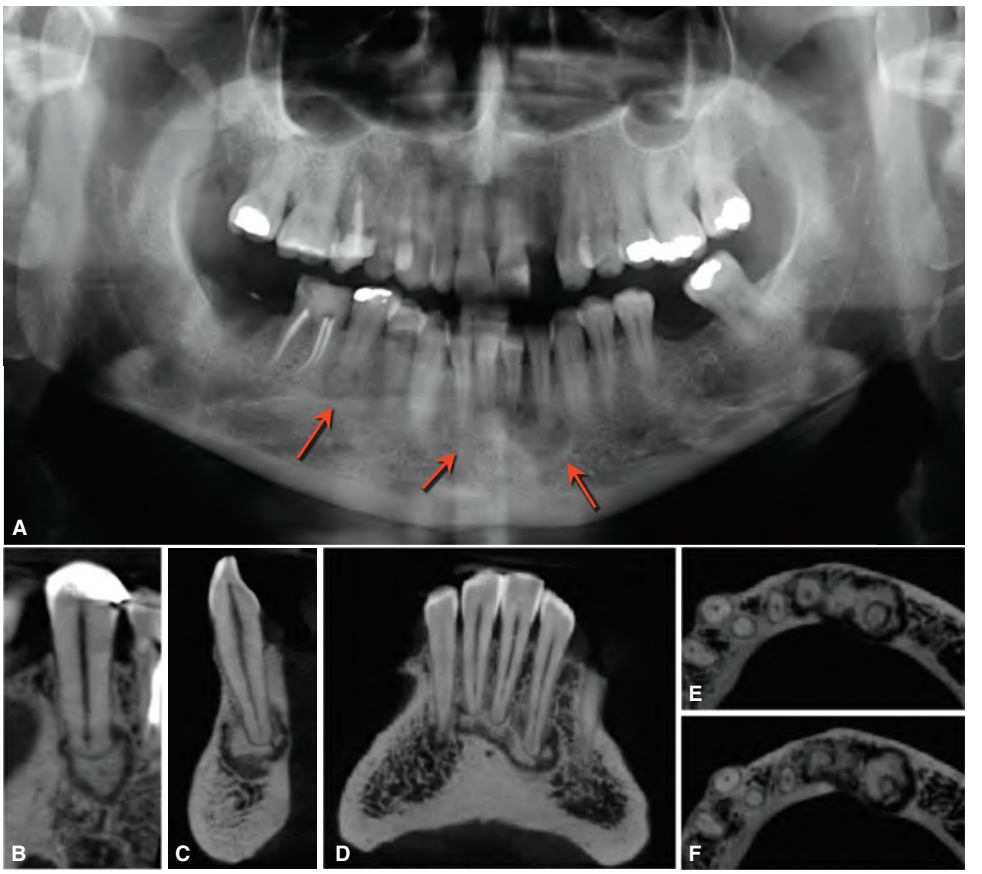

FIGURE 10-38 A case of *cemento-osseous dysplasia*. ***A.*** Panoramic radiograph displaying the radiolucent phase in several teeth (red arrows). All teeth tested normally to pulp sensibility tests. ***B.*** High resolution CBCT sagittal view of the mandibular right second premolar showing a characteristic radiopaque lesion surrounded by a rim of radiolucency. ***C.*** Sagittal view at the level of the mandibular right lateral incisor. ***D.*** Coronal view of the mandibular incisors. ***E & F.*** Axial view of the mandibular incisors displaying expansion of the cortical bone (red arrow). Accurate clinical diagnosis in such cases, requires thorough evaluation of any associated signs and symptoms as well as testing of pulpal responses. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

In summary, the algorithm for image interpretation involves a careful and systematic analysis (Figure 10-40). It is important to recognize, however, that any algorithm for diagnosis and interpretation may fail in some cases since not all lesions behave exactly as expected.[38]

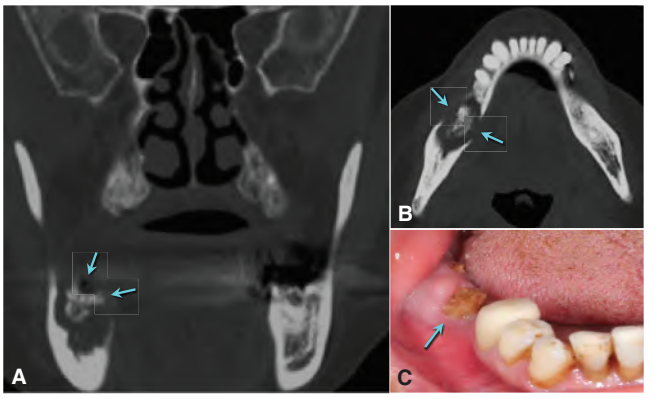

FIGURE 10-39 A case of *medication-related osteonecrosis of the jaw* in an 83-year-old woman who was taking a bisphosphonate drug for osteoporosis therapy for 6 years. ***A.*** Coronal CBCT view shows an ill-defined radiolucency of the right side of the mandible with internal opacity (blue arrows), characteristic of free-floating necrotic bone or "sequestrum." ***B.*** Axial view shows erosion or perforation of the buccal and lingual plates (blue arrows) along with the central sequestrum formation. Note contralateral uninvolved mandible. **C.** Clinical image of the same patient showing exposed necrotic bone (blue arrow) in the oral cavity. Diagnosis of such cases can only be made in conjunction with accurate medical history and clinical factors linked to bisphosphonate drug history. (Courtesy of Dr. Parish P. Sedghizadeh, Los Angeles, CA.)

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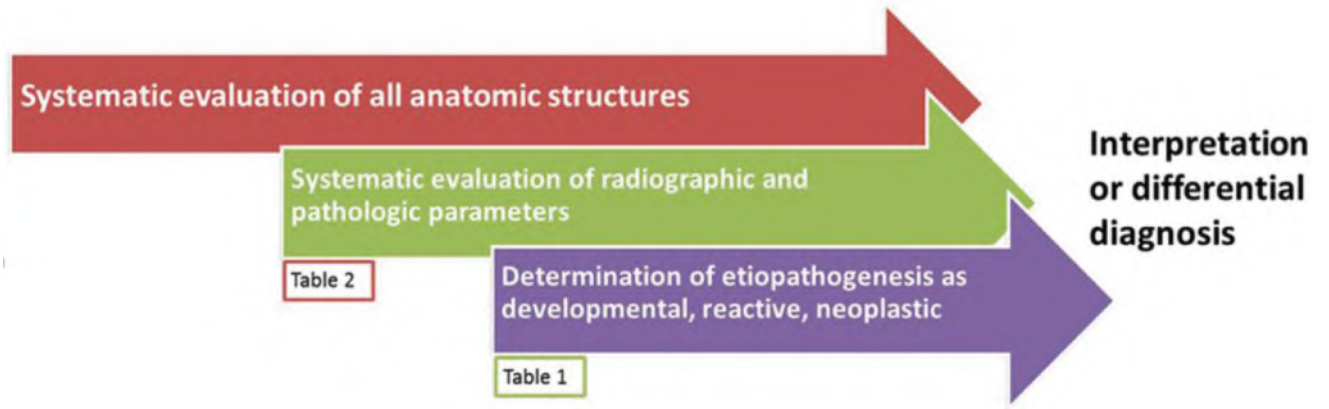

FIGURE 10-40 Diagram showing the integrated steps involved in imaging interpretation.

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Chapter 11

Treatment Planning and Case Selection

Paul A. Rosenberg, Matthew Malek, Katsushi Okazaki

Following the diagnosis of an endodontic problem, the clinician must establish a treatment plan that best meets the oral health needs of the patient. A central question is often whether or not the patient's best interests are served by a specific tooth's retention, or extraction and replacement.

The treatment plan is affected by an array of dental and nondental variables. The patient's health status, endodontic prognosis, motivation, and economic factors all play a role in determining the ultimate treatment plan. Periodontal status, restorability of the tooth, and prognosis of the proposed plan must all be considered.

A complex treatment plan with a questionable prognosis is not appropriate for a patient with limited motivation and a history of infrequent or inadequate dental care. The basic question for the clinician to resolve is, "How can I best serve the patients and meet their oral health needs?"

Today, clinicians have options that did not previously exist. The use of microscopy and improved lighting of the operative field enables clinicians to treat teeth that previously would have been thought to be hopeless. The use of ultrasonics has had a profound effect on post removal and nonsurgical retreatment of failed endodontics. When a tooth has a poor or hopeless endodontic prognosis, an implant can be considered as a replacement. However, retention of the natural dentition remains our primary goal.

This chapter reviews significant factors influencing development of a treatment plan for a patient with a diagnosis of a tooth requiring endodontic treatment. A decision concerning tooth retention, or extraction, can be resolved only after a complete review of the patient's medical, dental, and psychosocial status.

MEDICAL CONSIDERATIONS

Determining the medical status of the patient is a primary consideration before an endodontic treatment plan is established. Texts are available that provide detailed reviews of the impact of specific medical complications on the dental patient. For more details, see Chapter 31. Additionally, the American Academy of Oral Medicine (Edmonds, WA) has a website (www.aaom.com) that is a valuable resource for accessing information concerning medically compromised patients.

The American Society of Anesthesiologists (ASA, Park Ridge, IL) Physical Status Classification System (Table 11-1) is a widely used assessment method for preanesthetic patients. The clinician's responsibility is to go beyond the ASA classification and determine initially when the most recent visit of the patient to his or her physician was and what his or her compliance was with any prescribed medication.

In addition, basic questions to answer at the diagnostic visit include, but are not limited to:

- Date of the most recent medical examination, reason for the visit, and pertinent findings
- History of drug allergies
- History of pertinent information concerning drug interactions, and adverse effects
- Presence of prosthetic valves, joints, stents, and pacemakers
- History of required antibiotic premedication
- Bleeding disorders
- Reaction to local anesthesia with vasoconstrictor
- Assessment of the patient's level of anxiety
- Specific patient concerns

The use of only a written form to collect data about a patient's medical status is not sufficient. Frequently, a patient may not indicate important information on a written form but during a follow-up conversation with the dentist, reveal significant facts about their medical status. A prime example of this is a patient who has a history of a sexually transmitted disease and may not want to disclose that on a written form.

MEDICAL FINDINGS THAT MAY INFLUENCE ENDODONTIC TREATMENT PLANNING

There are several medical conditions that may influence the endodontic treatment planning. For a detailed discussion on this topic, see Chapter 31.

DEVELOPMENT OF THE ENDODONTIC TREATMENT PLAN

The strategic value of a tooth in question should be considered before initiating endodontic treatment. Some decisions may be straightforward, while others can be challenging as the clinician weighs multiple factors that will play a role in determining the ultimate success or failure of the case.

TABLE 11-1 American Society of Anesthesiologists Physical Status Classification System

ASA PS Classification	Definition	Examples, Including, But Not Limited to:
ASA I	A normal healthy patient	Healthy, nonsmoking, 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), active hepatitis, alcohol dependence or abuse, implanted pacemaker, moderate reduction of ejection fraction, ESRD undergoing regularly scheduled dialysis, premature infant PCA < 60 weeks, history (>3 months) of MI, CVA, TIA, or CAD/stents.
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, ongoing cardiac ischemia or severe valve dysfunction, severe reduction of ejection fraction, sepsis, DIC, ARD or ESRD not undergoing regularly scheduled dialysis
ASA V	A moribund patient who is not expected to survive without the operation	Examples include (but not limited to): ruptured abdominal/thoracic aneurysm, massive trauma, intracranial bleed with mass effect, ischemic bowel in the face of significant cardiac pathology or multiple organ/system dysfunction
ASA VI	A declared brain-dead patient whose organs are being removed for donor purposes	

From https://www.asahq.org/resources/clinical-information/asa-physical-status-classification-system

Referral of the patient to a specialist should be considered when the complexity of a procedure is beyond the ability of a clinician. Factors that affect endodontic prognosis, including periodontal and restorative considerations, must be considered. The decision concerning endodontic therapy and tooth retention, or extraction and implant placement, should be based on the best available evidence. The patient is usually best served when the natural dentition can be preserved. However, if endodontic treatment has a poor prognosis, an extraction and replacement by an implant is an appropriate consideration.

ENDODONTIC PROGNOSIS

Studies concerning endodontic prognosis identified a number of preoperative factors affecting the outcome of primary endodontic treatment. In a systematic review, it was determined that the absence of a periapical radiolucency significantly improves the outcome of root canal treatment.[1] In the same study, it was found that tooth vitality does not have an impact on endodontic prognosis as long as the periapex is healthy.[1]

It has also been found that the size of the radiolucency may affect the outcome of treatment.[1,2] Another study determined that existence of a sinus tract, narrow but deep periodontal probing depth, pain and discharging sinus have a significant effect on outcome of nonsurgical root canal treatment.[3] Preoperative pain is not only one of the most important predictors for postoperative pain, it may also have an impact on tooth survival after endodontic treatment.[4,5]

These findings suggest that preexisting signs and symptoms may affect the prognosis of treatment. Endodontic prognostic factors and factors affecting other disciplines, such as periodontics, 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 explained to the patient before initiation of the treatment as well. For more details, see Chapters 33 and 34.

Endodontic Retreatment

There is a lack of agreement concerning the endodontic prognosis of retreatment cases. Differences in research methodology may explain the variation in research findings. In a systematic review, it was suggested that the outcome of retreatment cases should be similar to treatment cases as long as access to the apical infection can be re-established (Figure 11-1).[1] However, there is some evidence indicating that the incidence of postoperative pain and flare up is higher in retreatment cases in comparison to treatments.[6] The presence of a preoperative periapical lesion, apical extent of root filling, and quality of coronal restoration have been shown to significantly affect the outcome of retreatment cases.[7]

Important questions to be considered before initiating 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 procedural problem that can be corrected?
- Is the canal system readily accessible for reentry?

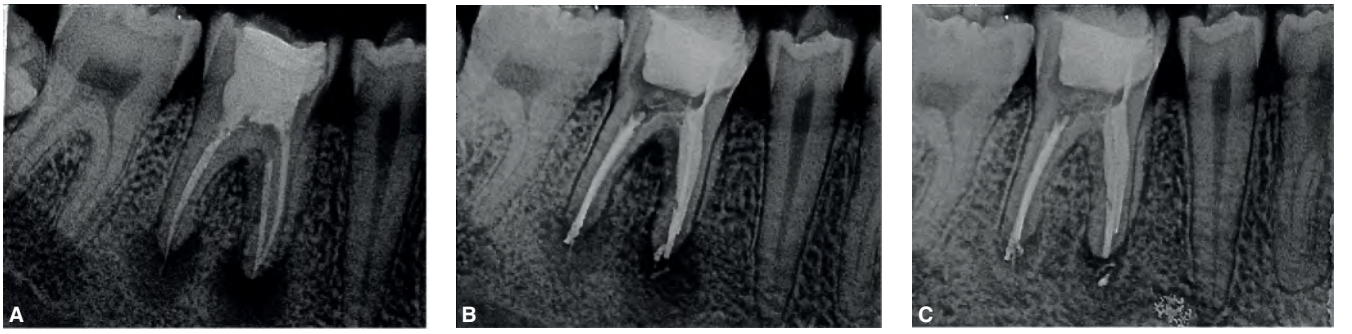

FIGURE 11-1 Potential for healing should not be underestimated. ***A.*** Preoperative radiograph. ***B.*** The mandibular right first molar was treated nonsurgically. ***C.*** Follow-up examination at 6 months indicates healing in progress.

- Are there additional factors (other than endodontic) that may have contributed to the failure?
- Is the tooth critical to the treatment plan?
- Does the patient understand the prognosis for the tooth and want to attempt retreatment?

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) (Figure 11-2). Retreatment cases may require surgical endodontics in combination with nonsurgical retreatment. Referral to a specialist is often helpful when planning treatment for complex cases (Figure 11-3). If retreatment (with or without surgery) for a tooth with a new restoration is being considered, it must be weighed against the possibility of an implant. Many variables must be considered before a reasonable conclusion can be reached. For more details, see Chapter 23.

Evaluation of Endodontic Treatment Outcome

It is essential that teeth having had endodontic treatment receive periodic follow-up examinations. This is particularly important in teeth with apical periodontitis. Apical lesions may heal within months or take years to resolve. Only through periodic review of the case can the clinician determine the course of healing. Numerous possibilities exist. There may be complete healing, partial healing, or failure of the treatment. The ideal outcome is the absence of clinical signs or symptoms and complete radiographic healing of preoperative apical periodontitis. Following endodontic treatment no further treatment may be required or apical surgery with or without retreatment is necessary.

A number of variables affect the decision concerning how best to approach the case requiring further treatment. The variables include quality of the previous instrumentation and obturation, periodontal status, quality of the restoration, and the patient's systemic health. However, the central issue always comes down to preventing microbial microleakage from the canal(s) space into the periradicular tissues.

Guidelines published by the European Society of Endodontology, state that the first follow-up examination should be made after 1 year.[8] If the original lesion is only diminished it should be categorized as "questionable and should be followed for up to 4 years. The treatment plan must be modified if clinical symptoms develop, or if the lesion increases in size.[8]

Single-Visit Versus Multiple-Visit Treatment

There is an ongoing debate concerning single visit endodontics. Some clinicians take an extreme approach and treat all cases in a single visit, regardless of the patient's symptoms or complexity of the case. Others believe that many vital cases are suitable for single-visit treatment. The number of roots, time available, and the clinician's skills are factors to be considered.

Severity of the patient's symptoms is another important consideration. For example, a patient with a necrotic pulp and in severe pain, with or without swelling, is not a candidate for single visit therapy. Treatment in such cases should be directed at alleviating pain, with filling of the canal deferred for a subsequent visit. There are advantages in having a patient return for a second visit, after initially presenting with an endodontic emergency due to pain and/or swelling with or without a stoma. The second visit allows the clinician to determine the effect of the treatment on inflamed and infected tissues.

The best available evidence in considering a single- or multiple-visit approach is frequently contradictory. Although some studies have reported less posttreatment pain in single-visit cases, a systematic review found that the incidence of postobturation discomfort was similar in the single- and multiple-visit approaches.[9–12] In another systematic review, it was concluded that there was a lack of compelling evidence indicating a significantly different prevalence of post treatment pain/flare-up with either single- or multiple-visit root canal treatment.[13] Differences in research methodology may explain the conflict in their findings.

Regarding the healing rate of single- and multiple-visit cases, a systematic review found that there was no detectable difference in the effectiveness of root canal treatment in terms of "radiographic success" between single- and multiple-visits.[10] A more recent systematic review also found that the healing rate of single- and multiple-visit root canal treatment is similar for infected teeth.[12]

Teeth with nonvital pulps and apical periodontitis pose a microbiological problem for the clinician. There is no

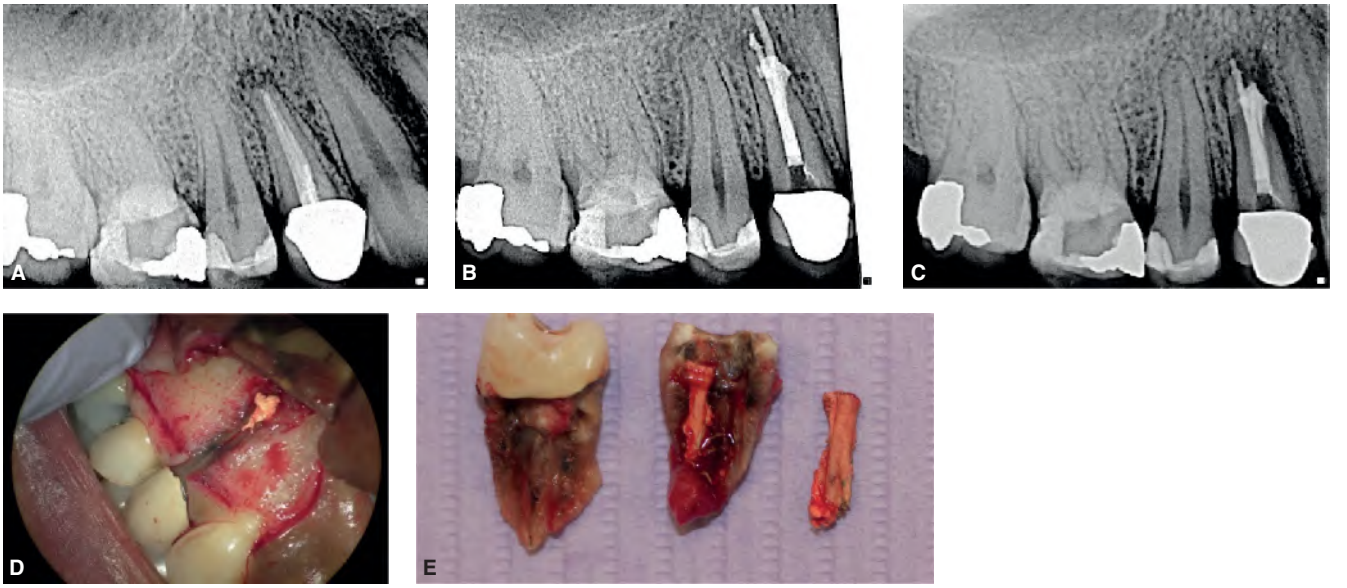

FIGURE 11-2 Retreatment without accurately determining the cause of failure. ***A.*** Preoperative radiograph showing periapical radiolucency associated with the maxillary right second premolar that is symptomatic to biting. A deep isolated pocket is present along the buccal root surface. ***B.*** Postoperative radiograph showing extensive overfilling. ***C.*** 6-month follow- up radiograph indicates that retreatment has failed. Since the cause of failure was not determined preoperatively, the clinician proceeded to periapical surgery. ***D.*** Intraoral image after flap reflection indicates a split in the root. The tooth is hopeless and requires extraction. ***E.*** The tooth following extraction.

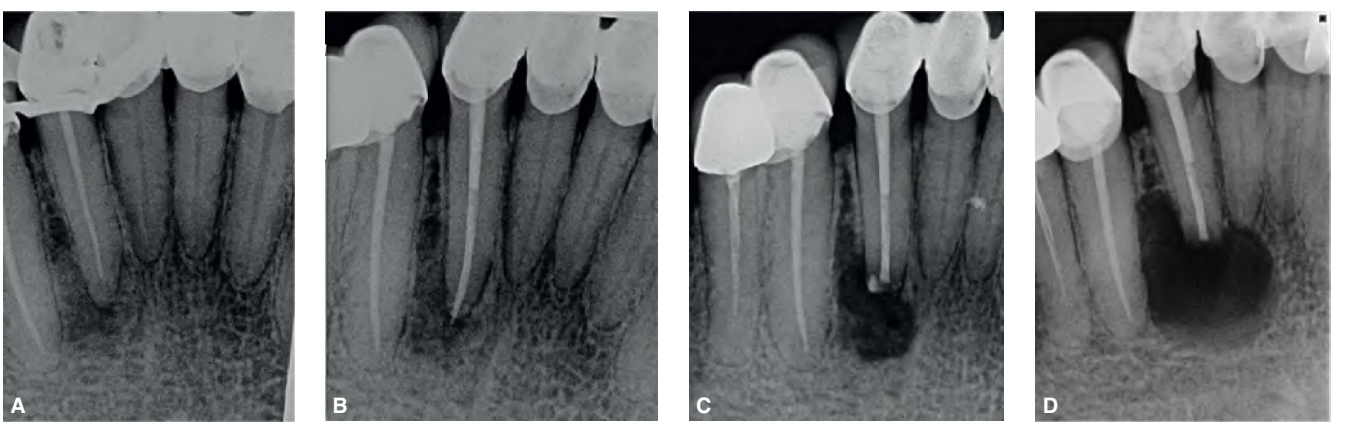

FIGURE 11-3 Treatment challenge in a mandibular lateral incisor. ***A.*** Preoperative radiograph. ***B.*** The tooth was retreated nonsurgically. ***C.*** At 12 months, treatment was deemed a failure. An apicoectomy was performed. ***D.*** One-year follow-up radiograph shows that the apicoectomy also failed. Possible reasons for failure include: absence of adequate seal, existence of a second canal, or vertical root fracture. Treatment options are retreatment (nonsurgically and surgically), or extraction. How would you treat this case?

consensus regarding 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.[14–16] In contrast, other researchers have not found any statistically significant difference in success when using the single-visit or multiple-visit approach to the nonvital tooth with apical periodontitis.[2,4,10,12,17–22].

A systematic review found that, single-visit root canal treatment appeared to be slightly more effective than multiple-visit treatment (6.3% higher healing rate). However, the difference between these two treatment regimens was not statistically significant.[21] This is a complicated issue because the inability to detect differences between groups might also be due to variations in research methodology, including sample size, duration of follow-up, and treatment methods.

It is possible that total elimination of microorganisms 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 disinfection of the root canal system is critical.[22] For more details, see Chapter 3.

Treatment planning for an endodontic case should be based on biologic considerations. Patients, who present with acute symptoms, present a different set of biologic issues than those with an asymptomatic tooth. Swelling and/or acute pain are signs of pathologic processes that must be addressed immediately if the tooth is to be retained. The ultimate treatment plan can be fully developed after the symptoms are under control.

Although in most cases, the clinical procedures required to complete endodontic treatment can be accomplished in a single-visit that does not mean it is the best approach. What *can be done* and what *should be do*ne represent two very different approaches to endodontic treatment planning. The patient's systemic health, level of anxiety, and symptoms, as well as the complexity of the root canal system, are among the factors that should be considered.

The author of a study, concerning outcome of initial endodontic treatment of apical periodontitis, 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 use of effective modifiers of any of these three factors might significantly improve the outcome of treatment".[2]

INTERDISCIPLINARY TREATMENT PLANNING

Periodontal Considerations

Severe periodontal lesions may complicate endodontic prognosis. Lesions with combined endodontic and periodontal components may necessitate consultation with an endodontist and/or periodontist in order to gather more information about the tooth's prognosis.

In a 4-year-retrospective study, it was found that attachment loss and periodontal status affected endodontic prognosis of endodontically treated molars.[23] 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 (Figure 11-4) 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 treatment plan for a tooth with an endodontic/periodontal lesion.

In primary endodontic disease, the pulp is nonvital (Figure 11-5), whereas in primary periodontal disease the pulp retains vitality. True combined endodontic–periodontal disease occurs less frequently. The combined lesion is usually 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.[24] The radiographic appearance of combined endodontic–periodontal lesions may sometimes be similar to that of a vertically fractured tooth. Therapy for true combined lesions requires both endodontic and periodontal therapy.

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 favorable. 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.[24]

Pathogenesis of the lesion can be better understood after pulp 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. For more details, see Chapter 36.

Surgical Considerations

Biopsy is the definitive means of diagnosing osseous pathoses that may mimic a lesion of endodontic origin. When retreatment is being considered, the clinician must determine

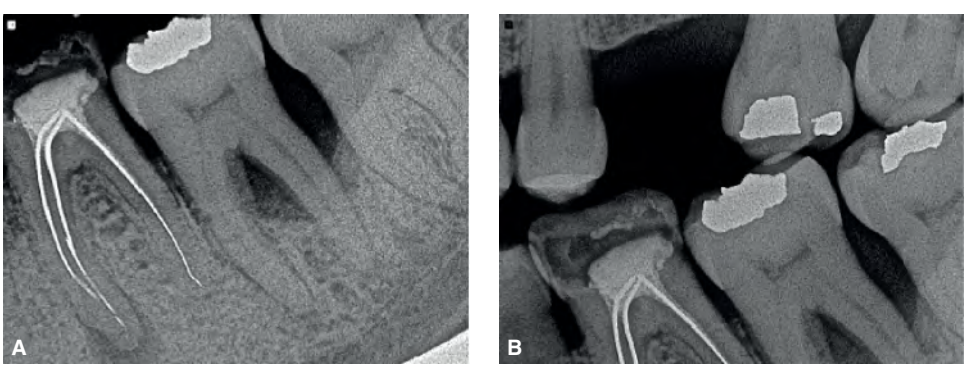

FIGURE 11-4 The pulp of the mandibular left second molar is nonvital, possibly due to root fracture. ***A***. Periapical radiograph ***B***. Bitewing radiograph. Severe destruction of intraproximal bone is indicative of primary periodontal involvement. Periodontal probing reached the apical third of the distal root. The prognosis is questionable. Extraction is indicated and should be done as soon as possible to prevent further damage to the bone associated with the adjacent teeth. Implant site preservation is another consideration in treatment planning for this case.

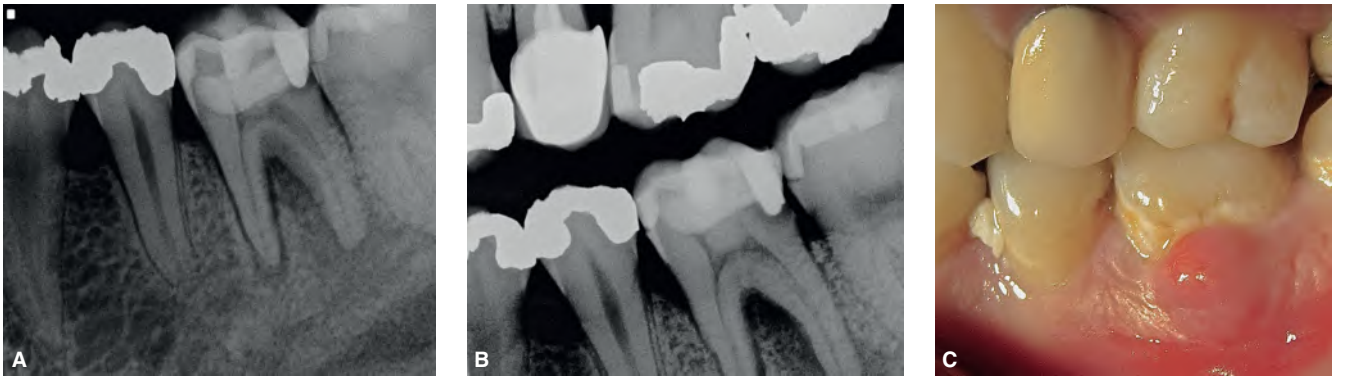

FIGURE 11-5 Primary endodontic lesion with secondary periodontal involvement in a mandibular left first molar. ***A.*** Periapical radiograph showing a deep coronal restoration with distal caries. ***B.*** Furcation bone loss is visible on bitewing radiograph. ***C.*** Clinically, a swelling is visible on the buccal attached gingiva of the affected tooth. Pulp sensibility test was negative. Periodontal probing reached the apical third of the distal root. The prognosis of endodontic treatment depends also on the healing of the periodontal lesion.

whether nonsurgical, surgical, or combined treatment is appropriate. This decision is influenced by the presence of complex restorations, posts, and radiographic assessment, including cone-beam computed tomography (CBCT) of prior endodontic therapy.

Endodontic surgery is most often performed in an attempt to improve the apical seal of unsuccessful nonsurgical treatment. Microbial leakage, due to an inadequate apical seal, is the primary cause of failure. Prior to surgery, it is important that the clinician determine the cause of microbial leakage. For example, a deficient restoration, or recurrent decay, will result in microleakage into the root canal space. Unless that issue is addressed, neither apical surgery nor retreatment will be successful. When a deficient restoration is identified it must be replaced in order to prevent continued microbial penetration.

Endodontic surgery may also be performed as a primary procedure when there are complications, such as calcific metamorphosis, preventing penetration of the canal. In those cases, by electing surgery as the initial treatment, the clinician can preserve 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 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 healing potential after endodontic surgery is an important consideration in the management of post treatment disease.[25] Numerous studies have examined the outcome of apical surgery and the results vary considerably.[26–28] This variability may reflect actual outcome differences or variations in case selection techniques, recall periods, and methodology.

In a prospective study, it was found that there is an increased odds ratio for disease persistence in teeth with large pretreatment lesions and pretreatment root canal filling of adequate length.[27] In another study, it was 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 having no pain at the initial examination.[26]

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.[29,30] Moreover, it has been reported that isolated endodontic lesions have much higher success rates (95.2%) at the 1- to 5-year follow-up, in comparison with endodontic–periodontal combined lesions (77.5%).[31]

Dramatic changes in surgical techniques and materials have occurred. The advent of microscopy, endoscopy, and ultrasonics, as well as improved root-end filling materials represents important modifications of surgical techniques. In an outcome study comparing traditional root-end surgery (TRS) and endodontic microsurgery (EMS), it was found that the probability of success for EMS was 1.58 times the probability of success for TRS.[23,32] In another study, comparing the EMS techniques with and without the use of higher magnification, it was found that the difference of probability of success between the groups was statistically significant for molars, but not for the premolar or anterior teeth.[33]

CBCT has proved to be of value diagnostically in surgical cases. It produces three-dimensional images of a tooth, lesions, and adjacent anatomical structures (Figure 11-6). It is beneficial in localizing the mandibular canal, mental foramen, maxillary sinus, and nasal cavity.[34–37] For more detials, see Chapter 24.

Restorative and Prosthodontic Considerations

A restoration may be jeopardized by a number of complications. 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 outcome. These complicating factors must

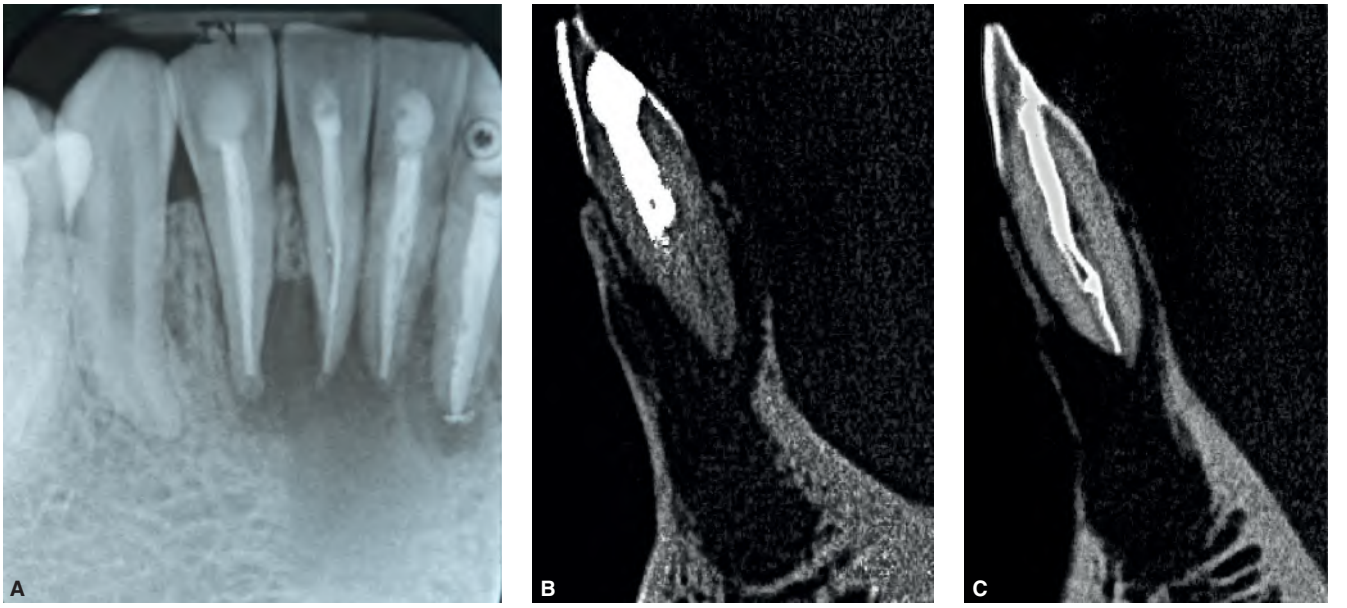

FIGURE 11-6 ***A.*** Periapical radiograph of the mandibular anterior region. ***B & C.*** CBCT reveals thin labial and lingual cortical bony plates that were not visible with conventional radiography. It also provides an accurate image of the extent of the periapical pathosis. This information is critical prior to endodontic surgery.

be recognized before endodontic treatment is initiated. For complex cases, a restorative treatment plan should be determined before initiating endodontic treatment. Some teeth may be endodontically treatable but nonrestorable without extensive additional treatment (Figure 11-7).

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 full coverage restorations to be compromised during endodontic access. Whenever possible, restorations should be removed before endodontic treatment.

It is possible that the complexity of a treatment plan involving endodontics, periodontics, including crown lengthening, and a post and crown may not be financially prudent. A patient should never be "pushed" into a treatment plan. A treatment plan and its alternatives should always be discussed with the patient weighing the risks and rewards of retaining the tooth versus a replacement.

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[36]:

- 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

In another systematic review, it was found that the odds for healing of apical periodontitis increase with both adequate root canal treatment and adequate restorative treatment.[37] 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.[37] These findings suggest that the quality of the coronal restoration is as important as the quality of the root canal treatment. Therefore, in order to increase the success of the treatment, it is strongly suggested to 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 Treatment and Dental Implants

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 treatment for a tooth with a questionable prognosis or extract and use a single-tooth implant as a replacement. Numerous studies have evaluated both nonsurgical endodontic therapy and endosseous dental implants.[3,7,38–45]

It is not possible to compare endodontic/implant outcome studies because of variations in research methodologies, follow-up periods, and criteria associated with determining success or failure. A review of current 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

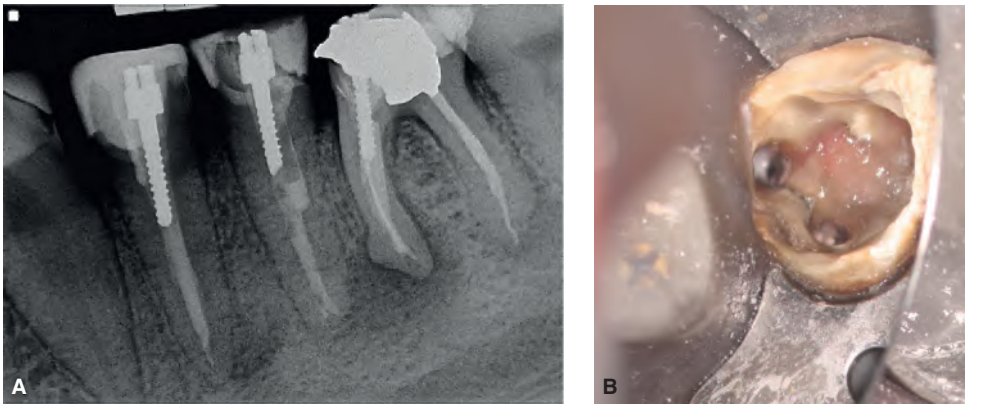

FIGURE 11-7 Restorability of teeth prior to initiating root canal therapy is an important consideration. ***A.*** The mandibular left second premolars and first molar were referred for evaluation and possible retreatment. Severe destruction of tooth structure in all three teeth is evident. This finding eventually leads the clinician to the conclusion that the patient would be better served by extraction and replacement with implants. ***B.*** Intraoral image after removal of the restoration and post on the mandibular molar. Note perforation on the floor of the pulp chamber.

planned and implemented. A study assessed clinical and radiographic success of initial endodontic therapy of 510 teeth over a period of 4 to 6 years. It was found that 86% of teeth healed and 95% remained asymptomatic and functional.[38] Another study considered the outcomes of endodontic treatment on 1,462,936 teeth. More than 97% of teeth were retained after 8 years.[46] 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.[39] A recent 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."[47] However, the number of patients included in this study were sustantially lower than those used in the other large epidimiological studies.

The American Dental Association'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%.[42] With such 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. Most current studies indicate no significant difference in the long-term prognosis between restored endodontically treated teeth and single-tooth implants.[44]

A retrospective cross-sectional comparison of initial nonsurgical endodontic treatment and single-tooth implants suggested that restored endodontically treated teeth and single-tooth implant restorations have similar failure rates (Figure 11-8).[48]

A review summarized the best available evidence concerning factors influencing the preservation of a tooth with endodontic therapy or replacement by a single-tooth implant.[44] Factors considered included prosthetic restorability of the natural tooth, quality of bone, esthetic concerns, cost-to-benefit ratio, systemic factors, potential for adverse effects, and patient preferences. 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."[44]

Aside from treatment outcome, 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.[49] In a prospective comparative study, it was also found that patients perceive both nonsurgical root canal treatments and single-tooth implants with high degrees of satisfaction and with minimal pain and discomfort.[50] One study compared endodontic molar retreatment and fixed partial dentures to implant–supported restorations. Despite its high survival rate, an implant was found to be the least cost-effective treatment option.[51] In light of this finding, patient's demographic and socio-economic status (SES) should be considered in desiging a treatment plan. In a related study, it was found that older patients were six times more likely than younger patients to receive dental implants. Male patients were 1.3 times more likely than female patients. Insured patients were 1.6 times more likely to have received root canal treatment compared with uninsured patients. The odds of patients with higher SES receiving dental implants was 2.4 times greater than those from low SES.[52]

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."[53]

Implants require a surgical procedure that may not be adjustable due to the patient's medical status. Diabetes mellitus can be a complicating factor. However, it has been

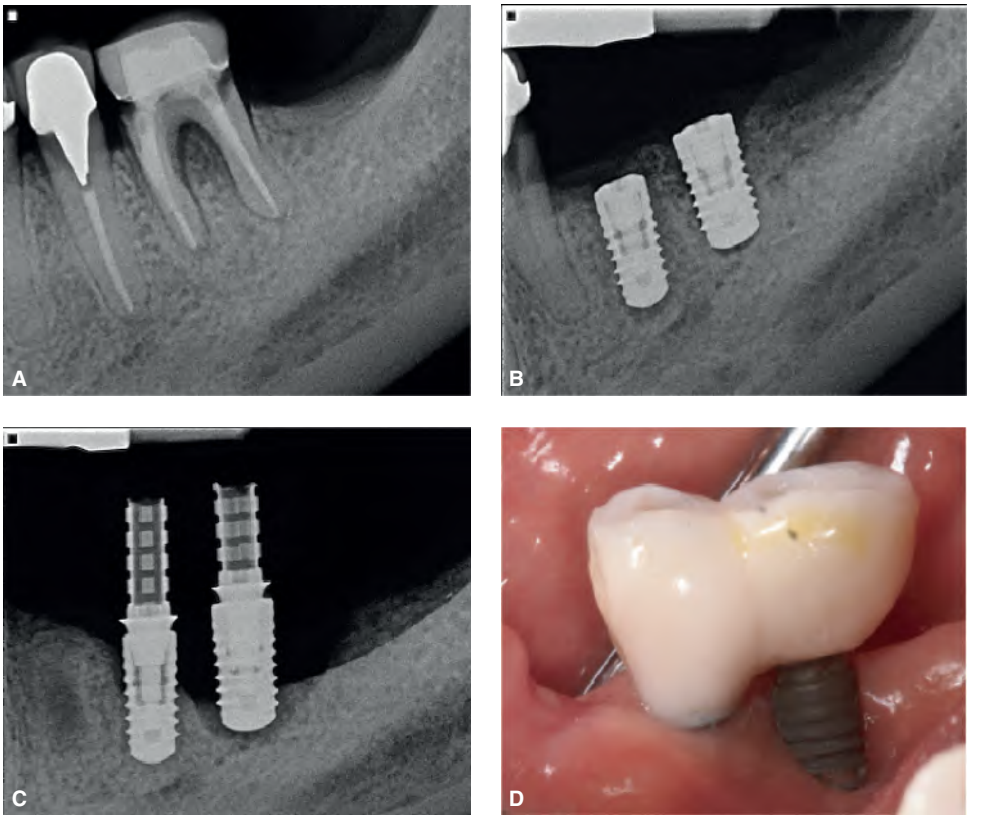

FIGURE 11-8 Implant placement may successfully address a problem if planned and executed appropriately. ***A.*** Periapical radiograph of the mandibular left second premolar and molar with questionable prognosis. ***B.*** The teeth were extracted and replaced with two implants. ***C & D.*** Eighteen months later, the implants are failing.

shown that dental implant osseointegration can be accomplished in these subjects as long as they have good glycemic control.[54]

It seems clear that the patient is 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. Similarly, it is also not reasonable for a patient to invest in root canal therapy, a post and 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 the rapid return of the patient's compromised dentition to full function and aesthetics. This 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 tooth retention or implant replacement.

OTHER FACTORS THAT CAN INFLUENCE ENDODONTIC CASE SELECTION

A variety of factors may complicate proposed endodontic therapy. Calcifications (Figure 11-9), dilacerations, or resorptive (Figure 11-10) defects may compromise endodontic treatment of a tooth with potentially strategic value. The inability to isolate a tooth efficiently is also a problem and may result in microbial contamination of the root canal system. Additional roots and canals pose a particular anatomic challenge that conventional radiographs do not always reveal. A bitewing radiograph is useful in providing an accurate image of the pulp chambers of posterior teeth (Figure 11-11). A CBCT is of particular value in providing a three dimensional image of a tooth's anatomy and an associate lesion. For more details, see Chapter 9.

Another consideration is the stage of maturity of the tooth. Primary and immature permanent teeth may have 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 self-esteem of the patient.

Some clinicians use a simple formula for determining those endodontic cases they treat and that 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 American Association of Endodontists (AAE) developed guidelines for assessing endodontic case difficulty (available at *www.aae.org*). 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

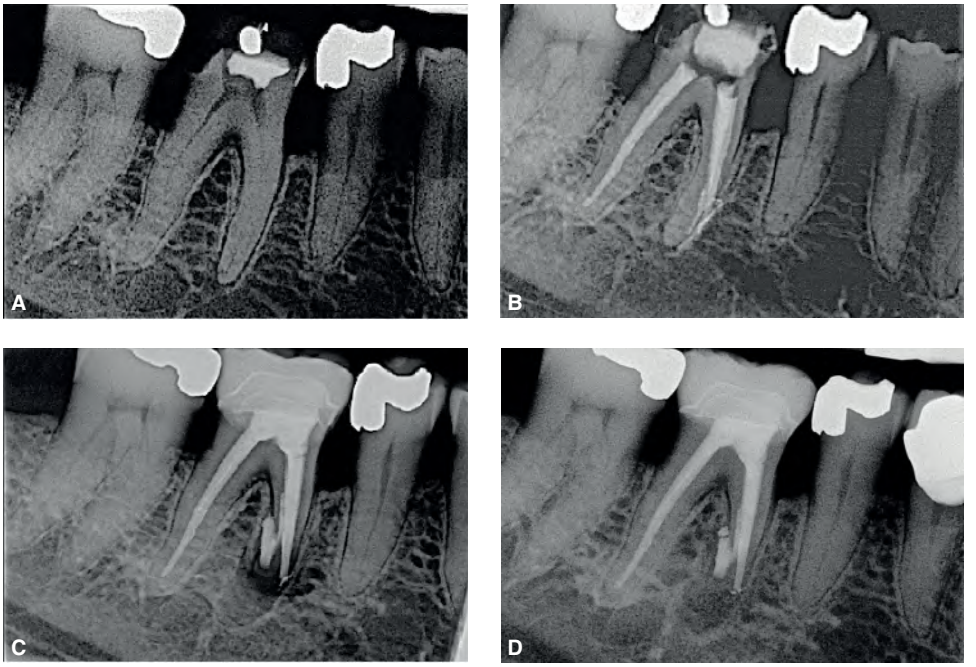

FIGURE 11-9 Calcification may complicate root canal treatment. ***A.*** Radiograph showing calcific metamorphosis in the coronal third of the root of the mandibular right first molar. The tooth is symptomatic to percussion. ***B.*** The clinician was not able to negotiate the mesial canals curvatures and perforated the distal side of the mesial root. The tooth is still symptomatic. ***C.*** Apical surgery was attempted to treat the defect. The retrograde filling is not optimum. ***D.*** Twelve-month follow-up radiograph showing signs of bony healing. The tooth is no longer symptomatic.

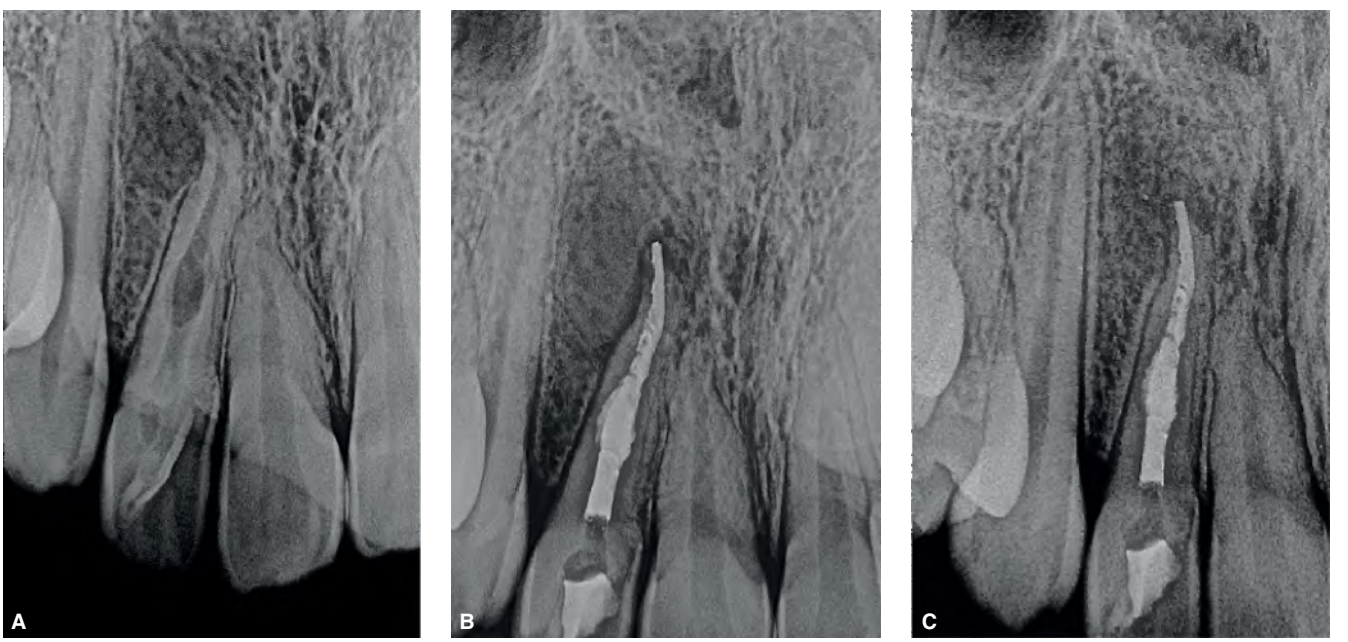

FIGURE 11-10 Some resorptive defects can be successfully treated endodontically. Early intervention, before perforation occurs, is essential. ***A.*** Preoperative radiograph showing a maxillary right lateral incisor with internal resorption and associated periapical rediolucency. ***B.*** Immediate postoperative radiograph. ***C.*** Twelve-months follow-up showing evidence of resolution of the periapical radiolucency. Regretfully, the tooth was not permanently restored.

lists criteria that can be used to identify cases that should be referred to a specialist. The use of surgical operating microscopes (Figure 11-12), endoscopes (Figure 11-13), CBCT, and ultrasonics enables the clinician to predictably diagnose and treat teeth that would not have been treatable previously.

Anxiety

Anxiety presents a problem at many levels of dental care and often results in the avoidance of dental treatment. This avoidance appears to be associated with significant deterioration of oral and dental health.[55] Severe anxiety may cause confused diagnostic results.[56] For more details, see Chapters 18 and 31.

Scheduling considerations

Scheduling of visits during endodontic treatment is an important component of treatment planning. If a vital case is to be treated using a multiple-visit approach, it is suggested that the clinician waits 5 to 7 days between canal instrumentation and obturation in order to allow periradicular tissues to recover. This, often, prevents filling a case while tenderness

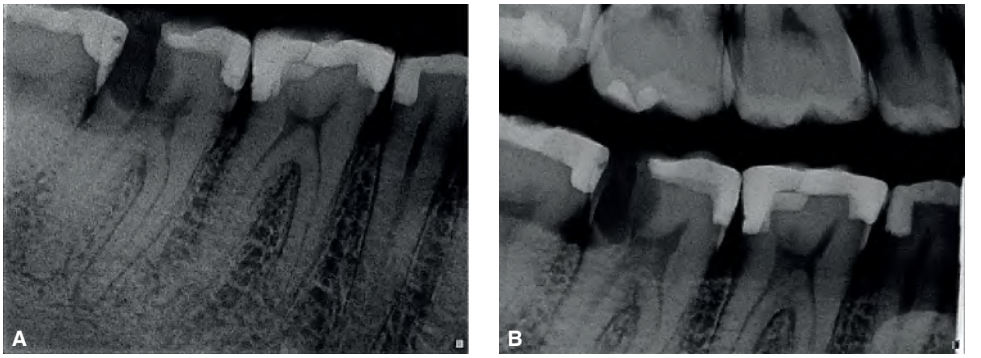

FIGURE 11-11 ***A.*** Periapical radiograph showing a madibular right second molar with large coronal destruction. ***B.*** A bitewing radiograph provides a clearer image of the pulp chamber and surrounding structures. It indicates that the tooth may require crown lengthening prior to restoration. This should be factored into the patient's treatment plan.

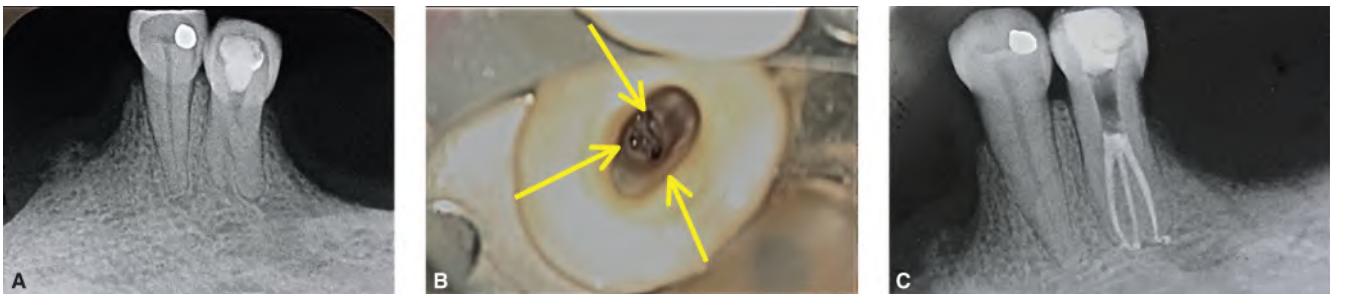

FIGURE 11-12 Enhanced visibility with use of the surgical microscope allows the clinician to accurately locate the third root of the mandibukar left first premolar. ***A.*** Preoperative radiograph. ***B.*** Three orifices are visible at their point of splitting from the main canal using 4.8× magnification (yellow arrows). ***C.*** Immediate postoperative radiograph.

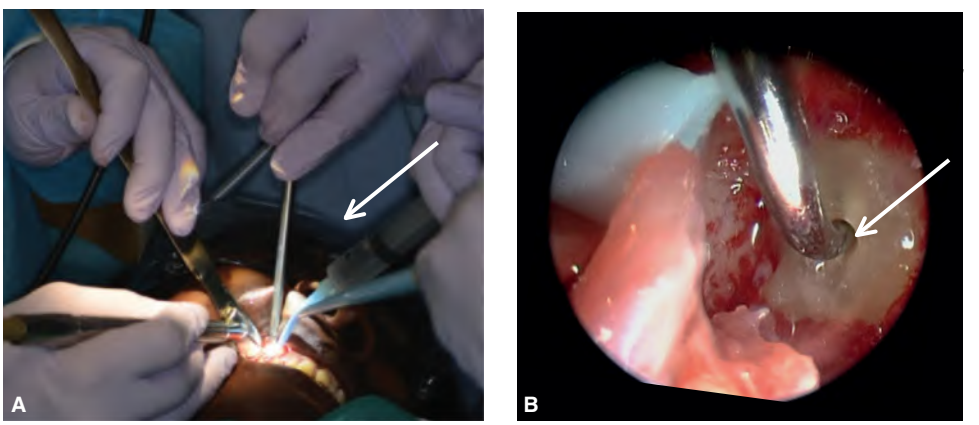

FIGURE 11-13 The endoscope provides high magnification images in areas that may be difficult to view with a microscope. ***A.*** Use of the endoscope during apical surgery. Note, white arrow points to the user of the endoscope during surgery. ***B.*** Retrograde preparation with ultrasonic device during apical surgery as seen on an endoscope monitor.

persists following instrumentation. When a vital case is to be treated in a single visit, adequate time must be scheduled so the clinician can comfortably complete the procedure. Since profound inferior alveolar nerve block anesthesia can require approximately 15 to 20 minutes, it is wise to include that time when scheduling a patient's appointment.

When indicated, appointments to obturate nonvital cases should be scheduled approximately 1 week after instrumentation in order to maximize the antimicrobial effect of intracanal medicaments and to allow time for the apical tissues to recover following instrumentation.[14,15]

Acute (pain and/or swelling) associated with nonvital cases should be seen every 24 to 48 hours in order to monitor the patient's progress and bring the acute symptoms under control. Further cleaning, shaping, and irrigation are important components of the treatment as the clinician seeks to eliminate persistent microorganisms from the canal system. Long delays between visits contribute to the development of resistant microbial strains and should be avoided.

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Chapter 12

Endodontic Considerations in Dental Trauma

Martin Trope

Traumatic injuries to the teeth result in damage to many dental and periradicular structures. Therefore, the management and consequences of these injuries are multifactorial, and knowledge of the interrelating healing patterns of these tissues is essential.

The focus of this chapter is the dental pulp and how damage to the dentinopulpal complex can contribute to complications after a traumatic injury and how correct diagnosis and treatment can result in favorable healing after an injury. More information on management of dental trauma can be obtained from the Website of the International Association of Dental Traumatology: <http://www.iadt-dentaltrauma.org>.

ASPECTS UNIQUE TO DENTAL TRAUMA

Most dental trauma occurs in the 7- to 10-year-old age group owing to falls and accidents near home or school.[1,2] It occurs primarily in the anterior region of the mouth, affecting the maxilla more than the mandible.[3]

Thus, in many cases after a dental traumatic injury, endodontic therapy is provided to caries-free, single rooted, **young** permanent teeth. Therefore, maintenance of the tooth is particularly important in these young individuals. Luckily, the potential for a successful endodontic outcome is very good if timely and correct treatment is provided soon after the injury.

CROWN FRACTURES

The primary aim from an endodontic point of view is to **maintain pulp vitality** after fractures involving the crowns of teeth.

Crown infractions—"incomplete fracture or crack of enamel without loss of tooth structure"[4]—and **uncomplicated crown fractures**—"fractures of the enamel only or enamel and dentin without pulp exposure"[4]—are injuries that have little danger of resulting in pulp necrosis. In fact, the biggest danger to the health of the pulp is iatrogenic, caused during esthetic restoration of these teeth. Therefore, meticulous follow-up, over at least a 5-year period, is an important endodontic preventive measure. Endodontic intervention should be considered at any follow-up time, when reactions to sensibility tests change, apical periodontitis develops, the root appears to have stopped development, or the pulp is obliterating.

Complicated Crown Fractures

By definition, complicated crown fractures involve enamel, dentin, and pulp[4] and occur in 0.9% to 13% of all dental injuries.[5–7] A crown fracture involving the pulp, if left untreated, will always result in pulp necrosis.[8] However, the manner and time sequence in which the pulp becomes necrotic allow a great deal of potential for successful intervention in maintaining pulp vitality.

The first reaction after an injury is hemorrhage and local inflammation. Subsequent inflammatory changes are usually proliferative but can be destructive. A proliferative reaction is favored in traumatic injuries since the fractured surface is usually flat, allowing salivary rinsing with little chance of impaction of contaminated debris. Therefore, unless impaction of contaminated debris is obvious, it is expected that within the first 24 hours after the injury, a proliferative response with inflammation will extend no more than 2 mm into the pulp (Figure 12-1).[9–11] In time, however, microbial infection will result in local pulp necrosis and a slow apical spread of pulpal inflammation.

Treatment

Treatment options are (1) **vital pulp therapy** comprising pulp capping, partial pulpotomy, and full pulpotomy or (2) **pulpectomy**. The choice of treatment depends on the stage of development of the tooth, time between the accident 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 and difficult. Of more importance, however, is the fact that necrosis of an immature tooth leaves it with thin dentinal walls that are susceptible to fracture both during and after an apexification procedure.[12] Therefore, every effort must be made to keep the pulp vital at least until the apex and cervical root have completed development. Pulpectomy in a mature tooth has a high success rate,[13] but vital pulp therapy (rather than removal) on a mature tooth performed under optimal conditions can also be carried out successfully.[14,15] Therefore, vital pulp therapy can be an option under certain circumstances, even though a pulpectomy is the treatment affording the most predictable success. **In an immature tooth, vital pulp therapy should be attempted, if at all feasible, because of the tremendous advantages of maintaining the vital pulp.**

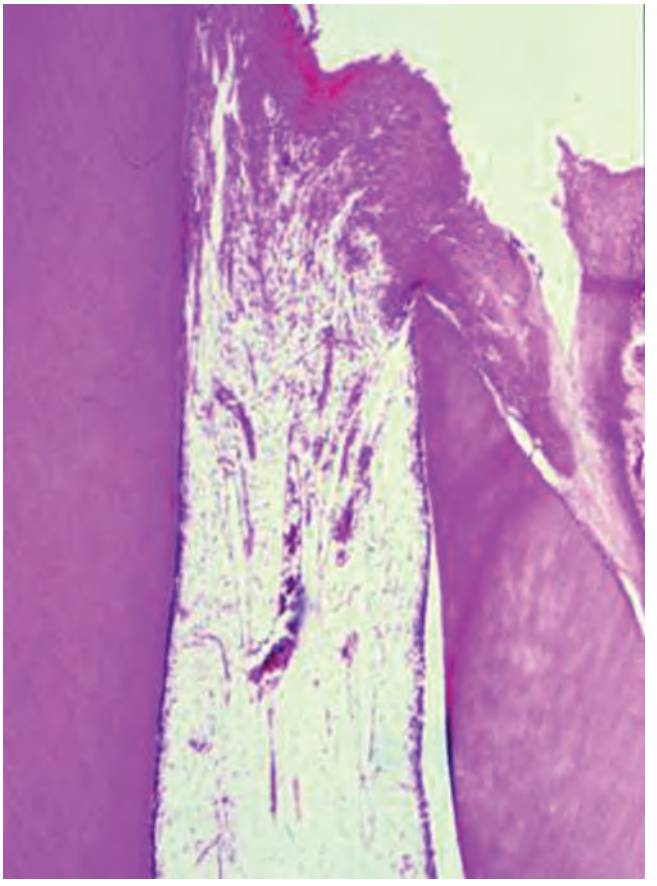

FIGURE 12-1 Histologic appearance of the pulp within 24 hours of a traumatic exposure. The pulp proliferates over the exposed dentinal tubules. There is approximately 1.5 mm of inflamed pulp below the surface of the fracture.

Time Between the Accident and Treatment

For 48 hours after a traumatic injury, the initial reaction of the pulp is proliferative, with a depth of no more than 2 mm of pulpal inflammation (Figure 12-1). After 48 hours, chances of direct microbial contamination of the pulp increase as the zone of inflammation progresses apically.[16] Thus, with time, the chance of success in maintaining a healthy pulp decreases.

Concomitant Attachment Damage

A concomitant periodontal injury will compromise 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.[17,18]

Restorative Treatment Plan

In a mature tooth, pulpectomy is a viable treatment option, unlike an immature tooth, in which the benefits of maintaining vitality of the pulp are great. However, if performed under optimal conditions, vital pulp therapy after traumatic exposures can be successful. Thus, 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 consideration. If a more complex restoration is to be placed (e.g., a crown), pulpectomy would be the more predictable treatment method.

VITAL PULP THERAPY FOR TRAUMATIC DENTAL INJURIES

Requirements for Success

Vital pulp therapy in traumatically injured teeth has a very high success rate if the following requirements are adhered to:

1. **Treatment of a healthy pulp** has been shown to result in predictable success.[19,20] Vital pulp therapy of the inflamed pulp, affords a less predictable success rate.[19,20] Therefore, the optimal time for treatment is within the first 24 hours, when pulp inflammation is superficial. As time increases between the time of injury and therapy, pulp removal must be extended apically to attempt to reach a noninflamed pulp area.
2. **A microbial-tight seal** is the most critical factor for successful treatment.[20] Challenge by microorganisms during the healing phase will cause failure.[21] On the other hand, if the exposed pulp is effectively sealed from microbial access, successful healing of the pulp with a hard tissue barrier will occur, independent of the dressing placed on the pulp, and in some cases where the capped pulp is inflamed.
3. **A proper pulp dressing** is important, and, presently, calcium hydroxide dressing is commonly used for vital pulp therapy for traumatized teeth. Its advantages are that it is antibacterial[22,23] and will disinfect the superficial pulp. Pure calcium hydroxide (pH 12.5) will cause necrosis of about 1.5mm of the pulp tissue[24,25] involving the superficial layers of inflamed pulp if present (Figure 12-2). The toxicity of the calcium hydroxide appears to be neutralized as the deeper layers of pulp are affected, causing coagulative necrosis at the junction of the necrotic and vital pulp, resulting in only a mild irritation. This mild irritation will initiate an inflammatory response and in the absence of microrganisms[24] will heal with a hard tissue barrier (Figure 12-3).[25,26] Hard-setting calcium hydroxide does not cause necrosis to the superficial layers of pulp but has been shown to initiate healing with a hard tissue barrier as well.[27,28] A major disadvantage of calcium hydroxide is that it does not seal the fractured surface. Therefore, an additional material must be used to ensure that the pulp is not challenged by microorganisms, particularly during the critical healing phase.

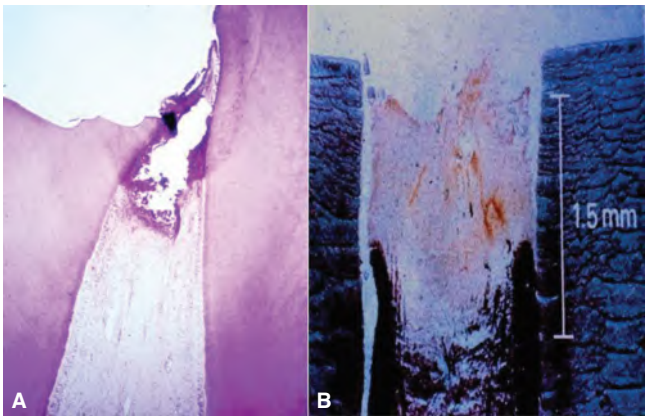

FIGURE 12-2 Pulp necrosis (1.5 mm) as a result of the high pH of calcium hydroxide.

FIGURE 12-3 Hard tissue barrier developed after calcium hydroxide partial pulpotomy. Histologic appearance of replacement odontoblasts and a hard tissue barrier.

Other materials, such as zinc oxide–eugenol,[20] tricalcium phosphate,[29] and composite resin,[30] have been proposed as materials for vital pulp therapy. They have not afforded the predictability of calcium hydroxide used in conjunction with a well-sealed coronal restoration.

Today bioceramic materials are considered the best materials for vital pulp therapy.[31,32] They have a high pH, similar to calcium hydroxide, when unset[32] and after setting will create an excellent bacteria-tight seal.[33] They also set hard enough to act as a base for a final restoration.[34] Mineral trioxide aggregate (MTA) was the first bioceramic used for this purpose. However, certain formulations may cause discoloration of the tooth which is a particular problem in traumatized teeth that are usually in the esthetic zone. Second generation bioceramics like premixed bioceramic putty (Endosequence, [Brasseler, Savannah, Georgia], or TotalFill [FKG Dentaire, La Chaux- De- Fonds, Switzerland]) do not have this disadvantage. For more details, see Chapter 27.

TREATMENT METHODS

Pulp Capping

Pulp capping implies placing a dressing directly onto the pulp exposure. As indicated by the success rate of this procedure (80%) compared with that of partial pulpotomy (95%),[35] it appears that a superficial pulp cap should not be considered after traumatic pulp exposures. The lower success rate is not difficult to understand since superficial inflammation develops soon after the traumatic exposure. Thus, 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 microbial-tight coronal seal is much more difficult to achieve in superficial pulp capping since there is no cavity depth to create this microbial-tight seal, as there is with a partial pulpotomy.

It should be noted that the success rates mentioned previously are of studies performed in the 1970s[20] with calcium hydroxide as the pulp capping agent and a restoration above with a high probability of leakage. Recent case series studies[34] on capping the inflamed pulp suggest that the bioceramic materials offer a better success rate than previously thought. Studies particularly on vital pulp therapy on traumatized teeth with inflamed pulps are yet to be performed but there is a good chance these too will show an improved success rate if bioceramic base is used before the final restoration.

Partial Pulpotomy

Partial pulpotomy implies the removal of coronal pulp tissue to the level of healthy pulp tissue. In traumatic injuries, this level can be accurately determined owing to the knowledge of the reaction of the pulp after a traumatic injury. This procedure is commonly called the "Cvek pulpotomy." For more details, see Chapter 27.

Full Pulpotomy

This procedure involves removal of the entire coronal pulp to the level of root orifices. This level of pulp amputation is chosen arbitrarily because of its anatomic convenience. Therefore, since the inflamed pulp sometimes extends past the canal orifices into the root pulp, many "mistakes" are made, resulting in treatment of an inflamed rather than a noninflamed pulp. This procedure is indicated when it is predicted that the pulp is inflamed to deeper levels of the coronal pulp or with symptoms of irreversible pulpitis. Traumatic exposures after 72 hours and a carious exposure with symptoms of irreversible pulpitis are two examples in which this type of treatment may be indicated. While the more predictable procedure would be pulpectomy in mature teeth, the benefits outweigh the risks for this treatment in immature teeth with incompletely formed apices and thin dentinal walls. For more details, see Chapter 27.

Pulpectomy

Pulpectomy implies removal of the entire pulp to the level of the apical foramen. The indications are for complicated crown fracture of mature teeth (if conditions are not ideal for vital pulp therapy). This procedure is not different from root canal treatment of a vital, nontraumatized tooth.

TREATMENT OF NONVITAL PULPS

Immature Tooth: Apexification

Apexification is indicated for teeth with open apices and thin dentinal walls in which standard instrumentation techniques cannot create an apical stop to facilitate effective root canal filling (Figure 12-4). For more detail, see Chapter 28.

PULP REVASCULARIZATION

Regeneration of a necrotic pulp has been considered possible only after avulsion of an immature permanent tooth. The advantages of pulp revascularization are the possibility

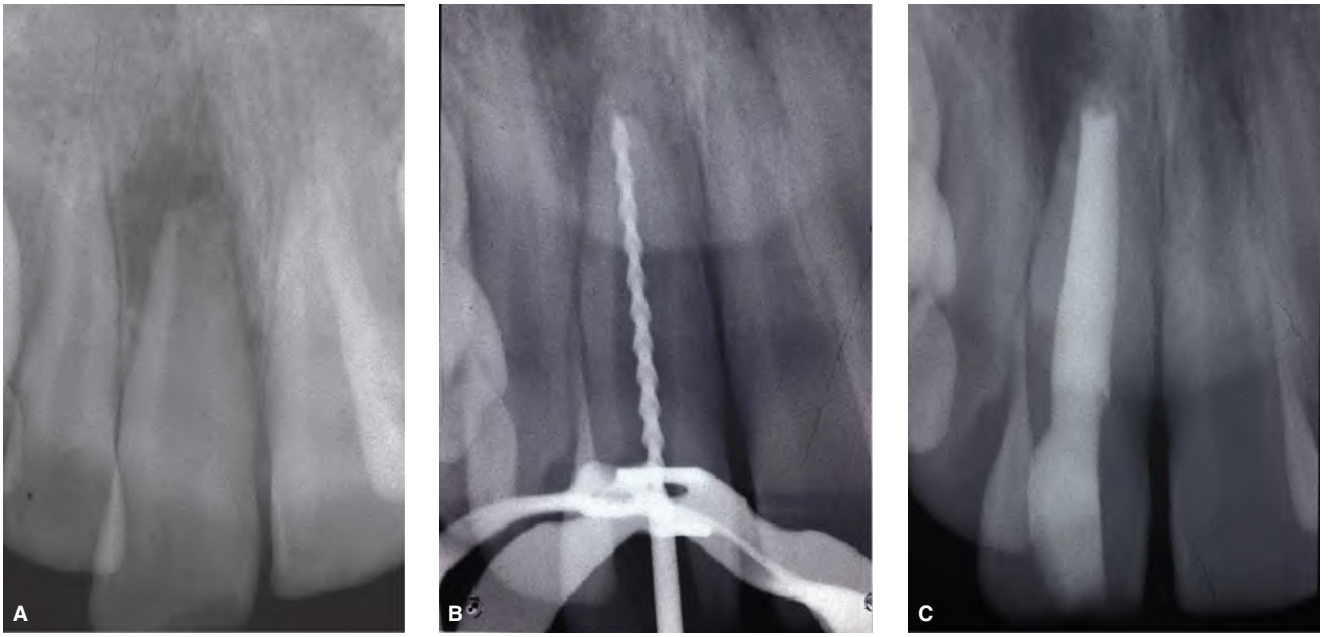

FIGURE 12-4 Apexification with calcium hydroxide. ***A.*** Maxillary central incisor with a history of crown fracture and subluxation. The pulp was infected and necrotic and there was an apical lesion of endodontic origin. The canal was cleaned and disinfected and a preparation of calcium hydroxide placed. ***B.*** Radiograph taken 3 months later showing a file used to test the presence of an apical barrier (apexification). ***C.*** The root canal was obturated with gutta-percha and sealer. The tooth was asymptomatic and the patient monitored for continued healing. (Courtesy of the Endodontic Clinic, Loma Linda University, School of Dentistry, CA.)

of further root development, reinforcement of dentinal walls by deposition of hard tissue, and thus strengthening the root against fracture. After replantation 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, that allows new tissue to grow into the pulp space relatively quickly. The pulp is necrotic but usually not degenerated and infected, so it will act 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 replantation proliferate coronally, replacing the necrotized portion of the pulp.[36–39] In addition, the fact that, in most cases, the crown of the tooth is intact and caries-free, ensures that bacterial penetration into the pulp space through cracks[40] and defects, will be a slow process. Thus, the race between the new tissue and infection of the pulp space favors the new tissue.

Regeneration of pulp tissue in a necrotic infected tooth with apical periodontitis has been thought until now to be impossible. However, if it were possible to create a similar environment as described previously for the avulsed tooth, regeneration should occur. Thus, if the canal is effectively disinfected, a matrix into which new tissue could grow is provided, and the coronal access is effectively sealed, regeneration should occur as in an avulsed immature tooth. A report by Banchs and Trope reproduced results in cases reported by others that indicate that it may be possible to replicate the unique circumstances of an avulsed tooth so as to revascularize the pulp in infected necrotic immature roots[41] (Figure 12-5). For more details, see Chapter 29.

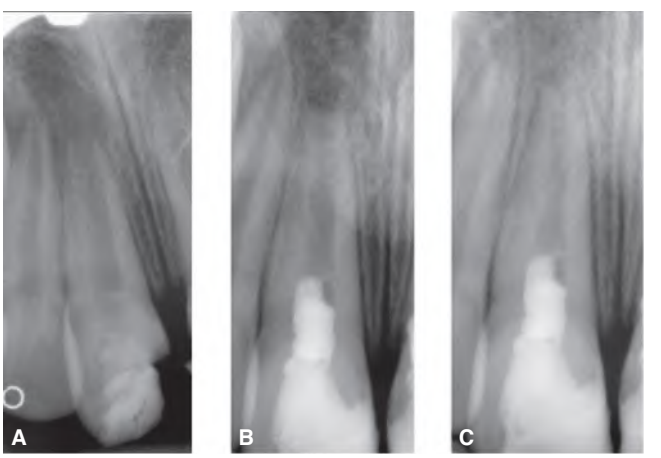

FIGURE 12-5 ***A.*** Immature tooth with a necrotic infected canal associated with apical periodontitis. The canal is disinfected with copious irrigation of sodium hypochlorite and a triantibiotic paste is placed. After 4 weeks, the triantibiotic paste is removed and a blood clot is created in the canal space. The access is filled with a mineral trioxide aggregate base and bonded resin above it. ***B.*** At 7 months, the patient is asymptomatic. The apex shows some signs of closure and healing of the apical periodontitis is noted. ***C.*** At 12 months, further apical healing is evident and root wall thickening has occurred, indicating that the root canal has been revascularized with vital tissue. (Courtesy of Dr. Blayne Thibodeau, Saskatoon, Canada.)

Mature Teeth

Traumatized mature teeth are routinely treated endodontically in the same manner as nontraumatized teeth.

CROWN-ROOT FRACTURES

These fractures are first treated periodontally to ensure that a good margin for restoration is possible. If the tooth can be maintained from a periodontal point of view, the pulp is treated as a crown fracture.

HORIZONTAL ROOT FRACTURES

This injury implies fracture of the cementum, dentin, and pulp. They are relatively infrequent injuries, occurring in less than 3% of all dental injuries.[42] Incompletely formed roots with vital pulps rarely fracture horizontally.[42] 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. Pulp necrosis develops in the coronal segment owing to its displacement but occurs in only about 25% of cases.[43] The clinical presentation 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 extrusive luxation (cervical fracture). Radiographic examination for root fractures is extremely important. Since root fractures are usually oblique (facial to palatal), one periapical radiograph can easily miss its presence. It is imperative to take at least three angled radiographs (45, 90, and 110°) so that at least at one angulation the X-ray beam will pass directly through the fracture line to make it visible on the radiograph (Figure 12-6).

Today cone-beam tomography (CBCT) has made the diagnosis of horizontal root fractures less of a challenge and allowed a much better assessment in terms of treatment strategy and prognosis prediction. CBCT is still relatively rare in the armamentarium of the people who see these injuries, so the periapical radiographic strategies for diagnosis remain extremely important.

Treatment

Emergency treatment involves repositioning of the segments in as close a proximity as possible and splinting to adjacent teeth for 2 to 4 weeks with a functional splint.[44,45] This splinting protocol has recently changed from the 2 to 4 months of rigid splinting that had been recommended for many years.[44,45] If a long time has elapsed between the injury and treatment, it will likely not be possible to reposition the segments close to their original position, compromising the long-term prognosis of the tooth.

TREATMENT OF ROOT FRACTURE 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 such treatment, and, in fact, if the coronal segment is below the attachment level and adequately splinted, the chances of healing do not differ from those of midroot or apical fractures.[43]

Midroot and Apical Root Fractures

Pulp necrosis occurs in 25% of root fractures.[43–45] In the vast majority of cases, the necrosis occurs in the coronal segment only with the apical segment remaining vital. Therefore, endodontic treatment is indicated in the coronal root segment only unless a periapical lesion is seen in the apical

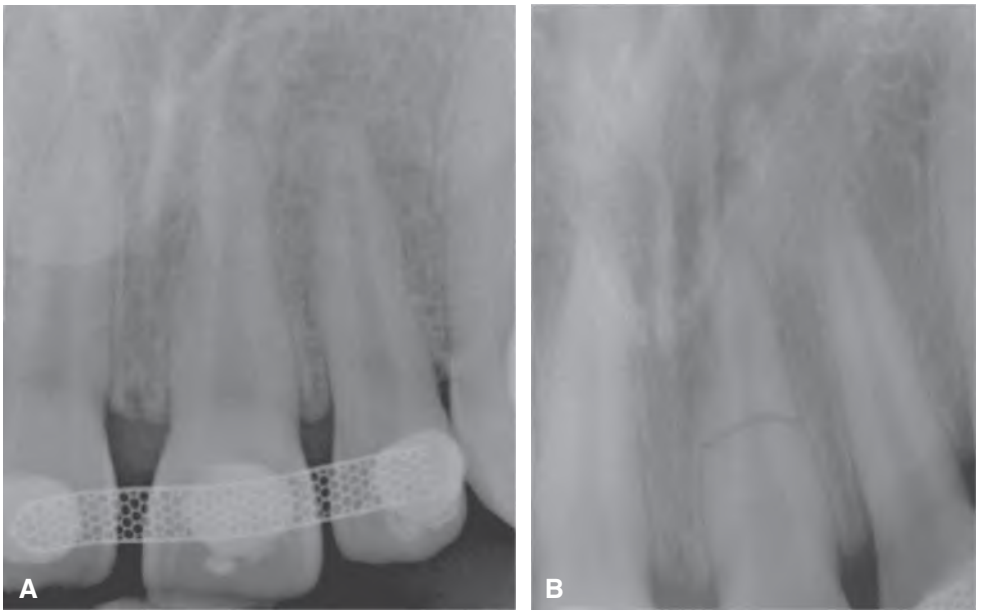

FIGURE 12-6 Radiographic technique for finding diagonal root fractures. ***A.*** Conventional 90° periapical radiograph of a maxillary central incisor with a diagonal root fracture that is easy to miss since the fracture does not show radiographically. ***B.*** A steep occlusal view (approximately 45°) clearly identifies the fracture because the X-ray beam can travel parallel to the fracture in the root. (Courtesy of the Endodontic Clinic, Loma Linda University, School of dentistry, California.)

segment. In most cases, the canal lumen is wide at the apical extent of the coronal segment, so long-term calcium hydroxide treatment or an Bioceramic apical plug is indicated. The coronal segment is root-filled after a hard tissue barrier has formed apically in the coronal segment and periapical healing has taken place (Figure 12-7). If Bioceramic is used, the root filling will be placed before healing is seen, making follow-up visits essential in these cases.

In rare cases, when both the coronal and apical pulp areas are necrotic, treatment is more complicated. Endodontic treatment through the fracture is extremely difficult and should be avoided. Endodontic manipulations, medicaments, and filling materials all have a detrimental effect on healing of the fracture site. In more apical root fractures, necrotic apical segments can be surgically removed. This is a viable treatment if the remaining root is long enough to provide adequate periodontal support. Removal of the apical segment in midroot fractures leaves the coronal segment with a compromised attachment, and the crown to root ratio must be assessed before the decision to maintain the tooth is made.

After the splinting period is completed, follow-up is conducted at the same intervals as all dental traumatic injuries: at 3, 6, and 12 months and yearly thereafter.

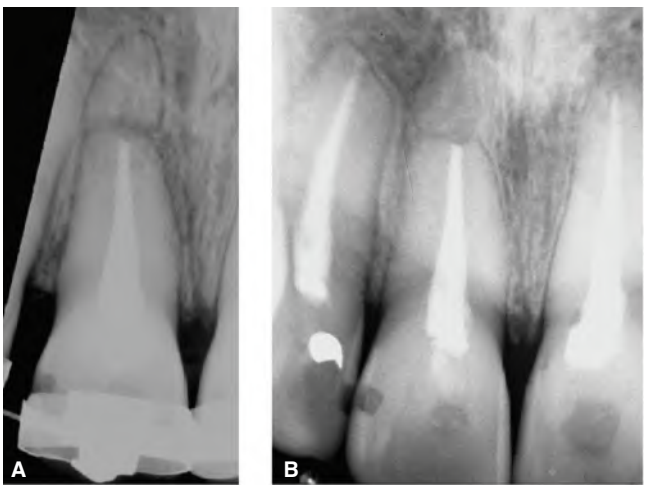

FIGURE 12-7 The coronal segment (only) is root-filled after a hard tissue barrier has formed at the most apical part of the coronal segment.

Factors Influencing Repair

1. The degree of dislocation and mobility of the coronal fragment are extremely important in determining outcome.[46,47] Increased dislocation and coronal fragment mobility result in a decreased prognosis.
2. Immature teeth are seldom involved in root fractures, but when they are, the prognosis is good.[48,49]
3. Quality of treatment: prognosis increases with early treatment, close reduction of the root segments, and semirigid splinting for 2 to 4 weeks.[44,45]

Complications are as follows: (1) pulp necrosis that can be treated successfully[48,50] by treating the coronal segment with adequate disinfection and long-term calcium hydroxide or Bioceramic and filling when a hard tissue barrier has formed; (2) root canal obliteration is not uncommon if the root segment (coronal or apical) remains vital (Figure 12-8).

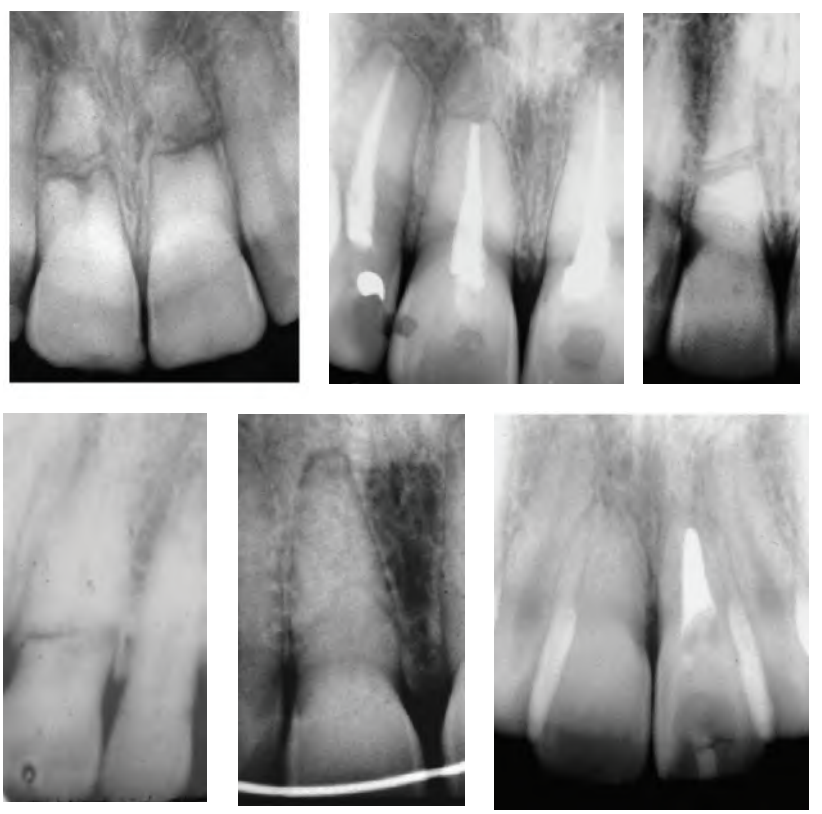

FIGURE 12-8 Examples of root canal obliteration in root-fractured teeth. It is common for the segments that maintain vitality to obliterate.

Endodontic Essentials

If the coronal segment is reduced quickly and a functional (nonrigid) splint is placed for 2 to 4 weeks, pulp necrosis is remarkably low. It will occur only 25% of the time, and in the vast majority of cases, the necrosis will occur only in the coronal segment. Root canal obliteration of both segments is very common. When necrosis does occur, disinfection of the coronal segment only should be initiated, followed by initiation of a physiologic barrier with long-term calcium hydroxide or a physical barrier with Bioceramic. Root canal obliteration is usually not treated endodontically.

LUXATION INJURIES

Definitions

1. **Concussion** implies no displacement, normal mobility, sensitivity to percussion.
2. **Subluxation** implies sensitivity to percussion, increased mobility, no displacement.
3. **Lateral luxation** implies displacement labially, lingually, distally, or incisally.
4. **Extrusive luxation** implies displacement in a coronal direction.
5. **Intrusive luxation** implies displacement apically into the alveolus.

Definitions 1 through 5 describe injuries of increasing magnitude in terms of the intensity of the injury and subsequent sequelae.

Luxation injuries result in damage to the attachment apparatus (periodontal ligament and cemental layer), the severity of which is dependent 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 altered or total loss of pulp vitality in the tooth. If the luxation injury is mild, the inflammatory response to the external root surface will be small and the pulp will maintain vitality. Inflammation owing to the trauma will thus be self-limiting, and cemental (favorable) healing will result.

If, on the other hand, the luxation injury is severe, the damage to the external root surface may be so diffuse that healing with new cementum alone may not be possible. If bone attaches directly to the root (ankylosis), the root will eventually be replaced by bone through physiologic resorption and apposition of the bone (osseous replacement). In addition and of special interest to the endodontist, with a severe luxation injury, the neurovasculature tissues of the pulp may be severed, resulting in a necrotic pulp. If this necrotic pulp becomes infected, the microbial toxins will pass through the tubules and the damaged cemental covering of the root, resulting in potentially catastrophic periradicular inflammation with bone and root resorption. For more details, see Chapter 15.

There are two types of trauma-related resorption in which the pulp plays an essential role:

1. In **external** inflammatory root resorption, the **necrotic, infected pulp** provides the stimulus for periodontal inflammation. If the cementum has been damaged and the intermediate cementum penetrated, as when the tooth undergoes a severe traumatic injury, the inflammatory stimulators in the pulp space are able to diffuse through the dentinal tubules and stimulate an inflammatory response over large areas of the periodontal ligament. Owing to the lack of cemental protection, periodontal inflammation will include root resorption and the expected bone resorption.
2. In **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. It is believed that coronal necrotic infected pulp provides a stimulus for pulpal inflammation in the more apical parts of the pulp. If in "rare" cases, the inflamed pulp is adjacent to a root surface that has lost its predentin protection, internal root resorption will result. Thus, both the necrotic infected pulp and the inflamed pulp contribute to this type of root resorption.

CONSEQUENCES OF APICAL NEUROVASCULAR DAMAGE

Pulp Canal Obliteration

Pulp canal obliteration is common after luxation injuries, if not severe enough to sever the neurovascular supply apically. The frequency of pulp canal obliteration appears inversely proportional to that of pulp necrosis. The exact mechanism of pulp canal obliteration is not known. It is theorized that the sympathetic or parasympathetic control of blood flow to odontoblasts is altered, resulting in uncontrolled reparative dentin formation.[43,50] Another theory is that hemorrhage and blood clot formation in the pulp after injury are a nidus for subsequent calcification if the pulp remains vital.[43,50] Pulp canal obliteration can usually be diagnosed within the first year after injury[43,50] and was found to be more frequent in teeth with open apices (>0.7 mm radiographically), in teeth with extrusive and lateral luxation injuries, and in teeth that have been rigidly splinted.[51]

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 > immature apex).[52] Pulp necrosis can lead to infection of the root canal system, with external inflammatory root resorption as the consequence. To develop pulp space infection, the pulp must first become necrotic (either partially or totally). In trauma, necrosis is usually due to displacement of the tooth, resulting in severing of the apical blood vessels. In mature teeth, pulp regeneration cannot occur, and usually by 3 weeks, the necrotic pulp will become infected. For more details, see Chapter 3.

Because serious injury is required for pulp necrosis, it is usual that areas of cemental covering of the root are also affected, resulting in the loss of its protective (insulating) quality. Now microbial toxins can pass through the dentinal tubules and stimulate an inflammatory response in the corresponding 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 (microorganisms in the pulp space) is removed (Figure 12-9).[53,54] Radiographically, the resorption is observed as progressive radiolucent areas of the root and adjacent bone (Figure 12-10). For more details, see Chapter 15.

Treatment

The attachment damage owing to the traumatic injury is often the focus of an emergency visit that, unfortunately, rarely involves the endodontist. The clinician's attention to pulp space infection should ideally be within 7 to 10 days after the injury.[55] Root canal therapy either removes the necrotic pulp as a nidus for microbes, or removes the stimulus (microbes already present) to the periapical inflammation. Without microbial stimulation, resorption will not occur or will heal.[55,56] In most cases, a new cemental attachment will form, but if a large area of root is affected, osseous replacement (ankylosis) can result by the mechanism already described. Treatment principles include prevention of pulp space infection or elimination of the microorganisms 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 microorganisms, and external inflammatory root 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.[57,58] However, even under the best conditions, revascularization will fail to occur on many occasions. Thus, a diagnostic dilemma results. If the pulp revascularizes, external root resorption will not occur and the root will continue to develop and strengthen. However, if the pulp becomes necrotic and infected, the subsequent external inflammatory root resorption that develops could result in the 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 since by that time, the teeth not revascularized could be lost to the resorption process. The laser Doppler flowmeter has been shown to be an excellent diagnostic tool for the detection of revascularization in immature teeth. These devices appear to accurately detect the presence of vital tissue in the pulp space by 4 weeks after the traumatic injury.[58]

   b. ***Prevent root canal infection by 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.[55,56] This is also a convenient time because this is the time

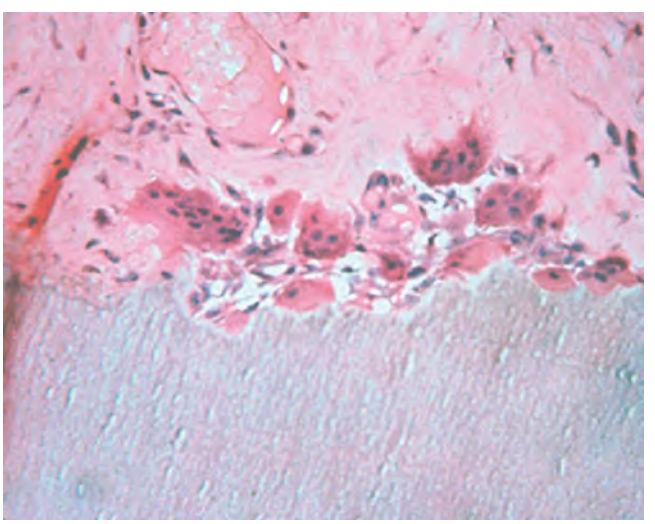

FIGURE 12-9 Histologic appearance of multinucleated osteoclasts (dentinoclasts) resorbing the radicular dentin.

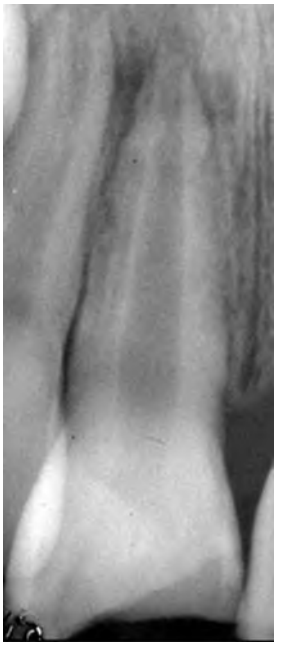

FIGURE 12-10 Inflammatory root resorption due to pulp space infection. Note the radiolucencies in the root **and** surrounding bone. (Courtesy of Dr. Frederic (Fred) Barnett, Philadelphia, PA.)

period in which the functional splint is removed and chemotherapeutic medicaments to limit the initial inflammatory response are stopped.[79]

From a theoretical point of view, the teeth treated at this time can be considered equivalent to the treatment of a tooth with a vital pulp. Therefore, the endodontic treatment could be completed in one visit. However, efficient treatment is extremely difficult so soon after a serious traumatic injury, and it is beneficial to start the endodontic treatment with pulpectomy and canal preparation followed by an intracanal dressing with a creamy mix of calcium hydroxide.[55,56] The practitioner can now fill the canal at his or her 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. Notwithstanding, in a compliant patient, the calcium hydroxide can be applied for a longer term (up to 6 months).[56]

2. **Elimination of 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 a long-term dressing with densely packed calcium hydroxide.[56] Calcium hydroxide can affect an alkaline pH in the surrounding dentinal tubules, kill bacteria, and neutralize endotoxin, a potent inflammatory stimulator.

The first visit consists of the microbial control phase with instrumentation of the canal and placement with a lentulo-spiral of a creamy mix of calcium hydroxide as an intracanal antibacterial agent. 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 since the radiodensity of calcium hydroxide in the canal is usually similar to that of the surrounding dentin (Figure 12-11). A radiograph is then taken at 3-month intervals. At each visit, the tooth is tested for symptoms of periodontitis. In addition, healing of the resorptive process and the presence or absence of the calcium hydroxide (i.e., calcium hydroxide washout) are assessed. Since the root surface is so radiodense as to make the assessment of healing difficult, the adjacent bone healing is assessed. If the adjacent bone has healed, it is assumed that the resorptive process has stopped in the root as well, and the canal can be permanently root-filled (Figure 12-12). If it is determined that additional healing would be beneficial before root filling, the need for replacing the calcium hydroxide in the canal is assessed. If the canal still appears calcified radiographically, there is no need to replace the calcium hydroxide. If, on the other hand, the canal has regained its lucent appearance, the calcium hydroxide should be repacked and reassessed in another 3 months.

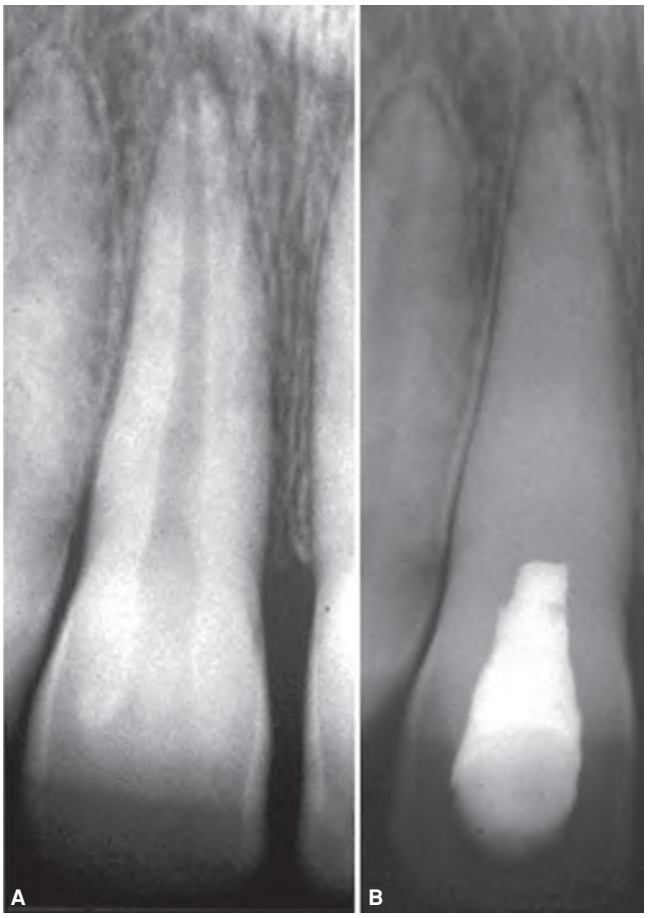

FIGURE 12-11 Root canal that "disappears" after placement of a thick mix of pure calcium hydroxide. (Courtesy of Dr. Frederic (Fred) Barnett, Philadelphia, PA.)

TREATMENT OF ESTABLISHED EXTERNAL ROOT RESOPTION IN A YOUNG PATIENT

Arresting external inflammatory root resorption owing to pulp space infection is achieved by disinfecting the pulp space. This is particularly difficult after a luxation injury since the cemental covering is also damaged. This results in the microbes and their by-products moving deeper into the dentinal tubules to stimulate and maintain the inflammation in the surrounding periapical tissues.

A triantibiotic paste introduced by Hoshino et al.[59] has been used in the revascularization process of necrotic teeth with open apices and apical periodontitis.[59] The antibiotic paste comprising metronidazole, ciprofloxacin, and minocycline has been shown to have an excellent antimicrobial spectrum for pulp space microbes and an excellent ability to penetrate the dentinal tubules. Since many cases of external inflammatory

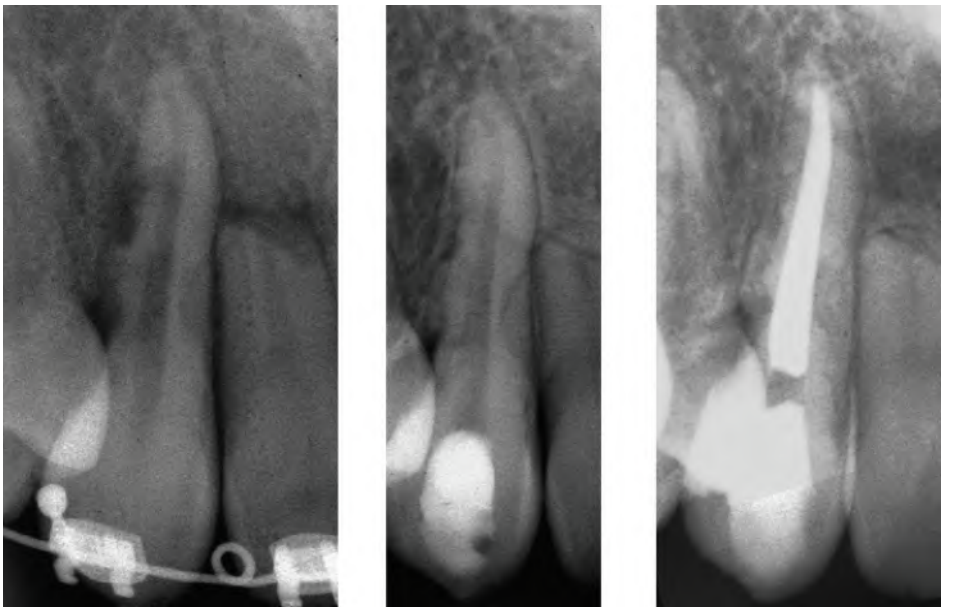

FIGURE 12-12 The root canal is filled when the adjacent bone has healed, indicating that root resorption has stopped. **Left.** Active root resorption with lucencies in the root and adjacent bone. **Middle.** After long-term calcium hydroxide treatment, the adjacent bone has healed. **Right.** The canal is now obturated.

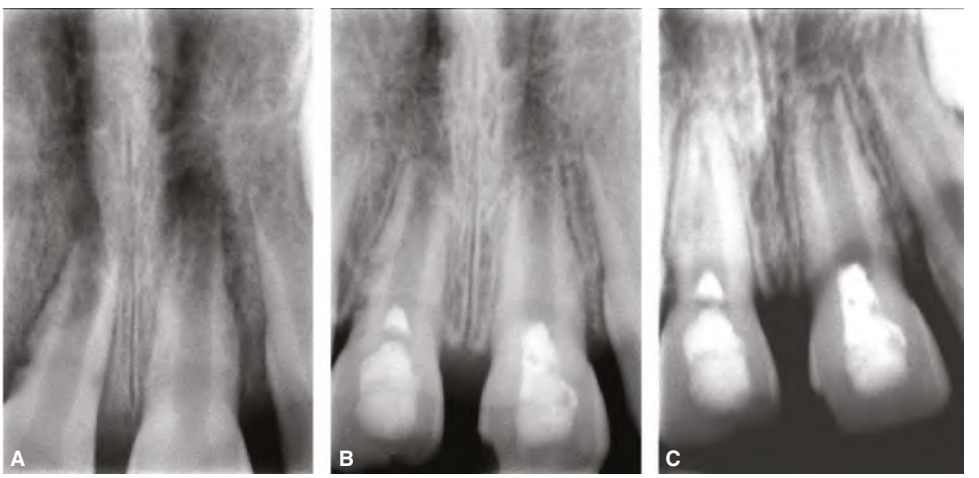

FIGURE 12-13 Immature tooth with a necrotic infected pulp, apical periodontitis, and external inflammatory root resorption. Treatment is as described in Figure 12-5. Note the healing of the external root resorption and the continued root development. (Courtesy of Dr. Linda Levin, Durham, NC.)

resorption owing to pulp space infection occur in immature teeth or teeth with apical resorption, the same procedure for the healing of external resorption and revascularization should work in these cases also. With this antimicrobial medicament, the potential exists to arrest the external inflammatory root resorption and revascularize the root canal (Figure 12-13).

The canal is disinfected without mechanical instrumentation but with copious irrigation with sodium hypochlorite and the use of a mixture of antibiotics for at least 4 weeks.[41,60] A blood clot is produced by mechanically irritating the periradicular tissues to the level of the cementoenamel junction to provide a matrix for the ingrowth of new tissue followed by a deep coronal restoration to provide a microbial-tight seal. The patient is followed every 3 months until evidence of healing of the external root resorption and thickening of the dentinal walls are seen (Figures 12-5 and 12-13).

An early study has confirmed the potent antibacterial properties of the triantibiotic paste used in this case,[61] and further studies are described in Chapter 29. The procedure described here can be attempted in most cases, and if after 3 months no signs of resorption repair and regeneration are present, the more traditional treatment methods may be initiated.

ENDODONTIC ESSENTIALS

Concussion and Subluxation

Since both of these injuries imply no noticeable displacement of the tooth in its socket, pulp consequences should not be expected. However, baseline and follow-up diagnostic tests are essential to pick up late complications such as pulp canal obliteration (most likely) or pulp necrosis.

Teeth that give a positive pulp test response at the initial examination cannot be assumed to be healthy and continue to give a positive response over time. Teeth that yield a negative response or no response, however, cannot be assumed to have necrotic pulps because they may give a positive 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 testing returns.[62]

The transition from a negative to a positive response at a subsequent test may be considered a sign of a healthy pulp. The repetitious finding of positive responses may also be taken as a sign of a healthy pulp. The transition from a positive to a negative response may be taken as an indication that the pulp is probably undergoing degeneration. The persistence of a negative response would suggest that the pulp has been irreversibly damaged, but even this is not absolute.[62]

Thermal and electric 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 3 weeks; 3, 6, and 12 months; and yearly intervals following the accident. 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 (–78°C) or dichlordifluoromethane (–40°C) placed on the incisal third of the facial surface gives more accurate responses than does a water ice pencil.[63,64] The intense cold seems to penetrate the tooth and splints or restorations and reach the deeper areas of the tooth. In addition, dry ice does not form ice water, that could disperse over adjacent teeth or gingiva to give a false-positive response. The electric pulp test relies on electric impulses directly stimulating the nerves of the pulp. These tests have limited value in young teeth but are useful in cases in which 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 electric pulp test becomes important.

Lateral Luxation, Extrusive Luxation

The tooth should be repositioned as soon as possible and a functional splint placed. The endodontic focus takes place at 7 to 10 days after the emergency visit. The clinician must evaluate the potential for revascularization of the pulp and therefore not initiate root canal treatment, or if revascularization is not likely, root canal treatment should be initiated at this 7- to 10-day time period.

The potential for revascularization depends primarily on the width of the apical constriction at the time of repositioning. If the width is 1.0 mm or more, revascularization is considered a possibility. The wider the apical opening is, the higher the likelihood of revascularization.[65] If revascularization is considered possible, a regular follow-up regimen must be scheduled. Since pulp testing is so erratic in these types of teeth, radiographic follow-up at short intervals with similar angulations is undertaken.

A small amount of radiographically appearing root resorption should be expected owing to the initial inflammation. This should be self-limiting and not increase in size after 3 weeks. If the radiographic resorption increases or any other signs of pulp infection are present, root canal therapy should be quickly initiated. If the apical opening is < 1 mm, root canal treatment should be started immediately. If the patient does not present to the clinician within 7 to 10 days postinjury, or if radiographic evidence of active external resorption is present, long-term calcium hydroxide treatment or a triantibiotic regimen should be initiated.

Intrusive Luxation

Intrusive luxation is the most destructive traumatic injury. Attachment damage is diffuse, and pulp survival is considered impossible. It is possible that in immature roots, spontaneous re-erupt and revascularization may occur, but in mature teeth, pulp necrosis is a certainty.[4] Thus, at the first chance of accessing the pulp space, root treatment should be initiated.

THE AVULSED TOOTH

The conditions at the emergency visit will have an important effect on the steps taken at the second visit. Ideally, the tooth should be replanted as soon as possible after the avulsion and functionally splinted. If unable to replant the tooth, it should be placed in a physiologic storage solution to allow for an extended extraoral time with fewer resorption complications after reimplantation. At the emergency visit, the root is prepared depending on the maturity of the tooth (open vs. closed) and the extraoral **dry time**. A dry time of 60 minutes is considered the point at which survival is unlikely for periodontal ligament cells.

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. If the tooth has a closed apex, revascularization is not possible,[65] 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 where periodontal healing would be expected.[66–68]

A continuing challenge is the treatment of the tooth that has been dry for more than 20 minutes (periodontal cell survival is ensured) but less than 60 minutes (periodontal survival unlikely). In these cases, logic suggests that the root surface consist of some cells with the potential to regenerate and some that will act as inflammatory stimulators. Exciting new strategies are under investigation that may be extremely valuable in these cases. The use of Ledermix placed in the canal space at the emergency visit has been found to be valuable in cases that were considered hopeless in the past[69] (see the following), and this medicament may prove extremely valuable in the 20- to 60-minute dry time period.

Open Apex

Soak in doxycycline or cover with minocycline for 5 minutes, gently rinse off debris, and replant. In an open-apex tooth, revascularization of the pulp and continued root development are possible (Figure 12-14). Cvek et al.[70] 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 revascularization. This result was confirmed in dogs by Yanpiset et al.[71] Ritter et al.[72] found that covering the root with minocycline that attaches to the root for approximately 15 days further increased the revascularization rate in dogs. Although these 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 will occur in humans as well. As with the tooth with the closed apex, the open-apex tooth is then gently rinsed and replanted.

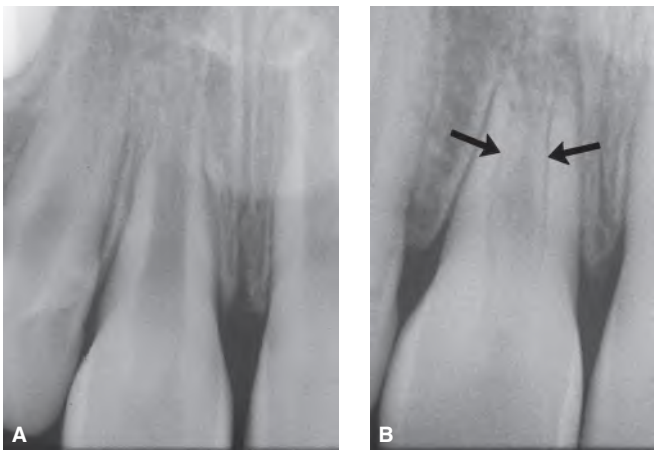

FIGURE 12-14 Revascularization of an immature root. A tooth with an open apex was replanted soon after the avulsion. A shows the radiographic picture immediately after replantation B. The follow up taken The follow-up radiograph 1 year later suggests that revascularization had taken place. In this case, it appears that new periodontal tissue has formed with a lamina dura and bone growth within the pulp canal (black arrows). (Courtesy of Dr. Cecilia Bourguignon, Paris, France.)

EXRAORAL DRY TIME MORE THAN 60 MINUTES

Closed Apex

Remove the periodontal ligament by placing it in etching acid for 5 minutes, soak it in fluoride, and replant. When the root has been dry for 60 minutes or more, survival of the periodontal ligament cells is not expected.[66,73] In these cases, the root should be prepared to be as resistant to resorption as possible (attempting to slow the ankylotic osseous replacement process). These teeth should be soaked in an acid for 5 minutes to remove all remaining periodontal ligament and thus remove the tissue that will initiate the inflammatory response on replantation. The tooth should then be soaked in 2% stannous fluoride for 5 minutes and replanted.[74,75] Alendronate was found to have resorption slowing effects similar to those of fluoride when used topically,[76] but further studies need to be carried out to evaluate whether its effectiveness is superior to fluoride and if this justifies its added cost. Some studies have found that Emdogain (enamel matrix protein) may 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.[77,78] Another study has not confirmed the beneficial effect of Emdogain.[79]

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, the endodontic treatment, if performed after replantation, involves a long-term apexification procedure. In these cases, completing the root canal treatment extraorally, where 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 free of bacteria.

Open Apex

Replant? If yes, treat as described with a closed-apex tooth. Endodontic treatment may be performed out of the mouth. Since these teeth are in young patients in whom facial development is usually incomplete, many pediatric dentists consider the prognosis to be so poor and the potential complications of an ankylosed tooth so severe as to recommend that these teeth not be replanted. However, considerable debate exists as to whether it would be beneficial to replant the root even though it will inevitably be lost owing to osseous replacement. If the patients are followed carefully and the root is submerged at the appropriate time,[80,81] the height and, more importantly, the width of the alveolar bone will be maintained. This allows for easier permanent restoration, when the facial development of the child is complete. Studies are ongoing to evaluate whether the present recommendations should be changed.

ADJUNCTIVE THERAPY

Systemic antibiotics given at the time of replantation and prior to endodontic treatment are effective in preventing microbial invasion of the necrotic pulp and therefore subsequent inflammatory resorption.[82] Tetracycline has the additional benefit of decreasing root resorption by affecting the motility of the osteoclasts and reducing the effectiveness of collagenase.[83] The administration of system antibiotics is recommended beginning at the emergency visit and continuing until the splint is removed.[84] For patients not susceptible to tetracycline dentin staining, doxycycline twice daily for 7 days at an appropriate dose for patient age and weight[83,84] is the antibiotic of choice. Penicillin V 1000 mg and 500 mg every 6 hours for 7 days has also been shown to be beneficial. The microbial 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, chlorhexidine rinses for 7 to 10 days are useful.

Bryson et al.[69] found benefit in removal of the pulp contents at the emergency visit and placing Ledermix (not available in the United States) into the root canal. This product contains a tetracycline corticosteroid combination that has been shown to move through the dentinal tubules. Based on the results of the study, the use of the medicament was able to shut down the inflammatory response after replantation to allow for more favorable healing compared with those teeth that did not have the medicament.

It appears that any corticosteroid alone (without tetracycline) is equally effective in shutting down the inflammatory response and decreasing the incidence of osseous replacement.[85]

The need for analgesics should be assessed on an individual case basis. Mild analgesics are usually adequate, or a nonsteroidal anti-inflammatory drug may be recommended. The patient should be sent to a physician for consultation regarding a tetanus booster within 48 hours of the initial visit.

Second Visit

This visit should take place 7 to 10 days after the emergency visit. At the emergency visit, emphasis is placed on the preservation and healing of the attachment apparatus. The focus of this 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 and bone and root resorption. Also at this visit, the course of systemic antibiotics is completed, the chlorhexidine rinses can be stopped, and the splint is removed.

ENDODONTIC TREATMENT

Extraoral Time Less Than 60 Minutes

Closed Apex

Initiate endodontic treatment at 7 to 10 days. In cases in which endodontic treatment is delayed or signs of resorption are present, treat with calcium hydroxide until evidence of healing is present, such as redevelopment of the periodontal ligament space. The root canal can then be filled and the crown restored.

Open Apex

Avoid endodontic treatment and look for evidence of revascularization. At the first indication of an infected pulp, initiate the apexification procedure.

Extraoral Time More Than 60 Minutes

Closed Apex

These teeth are treated endodontically in the same way as those teeth that had an extraoral time of <60 minutes.

Open Apex

If endodontic treatment was not performed out of the mouth, initiate the apexification procedure. In these teeth, the chance of revascularization is extremely poor.[55,86] Therefore, no attempt is made to revitalize these teeth. An apexification procedure is initiated at the second visit if root canal treatment was not performed at the emergency visit. If a root canal filling was placed 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 sealing ability. A depth of at least 4 mm is recommended and a cotton pellet need not be placed; the temporary restoration is placed directly onto the calcium hydroxide in the access cavity. Calcium hydroxide should first be removed from the walls of the access cavity because it is soluble and will wash out when it comes in 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 and the residual resin is removed at a later appointment. At this appointment, healing is usually sufficient to perform a detailed clinical examination on the teeth surrounding the avulsed tooth. The sensibility tests, reaction to percussion and palpation, and periodontal probing measurements should be carefully recorded for reference at follow-up visits.

ROOT FILLING VISIT

The root canal can be filled at the practitioner's convenience or, in the case of long-term calcium hydroxide therapy, when an intact lamina dura can be traced. If the endodontic treatment was initiated 7 to 10 days after the avulsion and clinical and radiographic examinations do not indicate pathosis,

filling of the root canal at this visit is acceptable, although the use of long-term calcium hydroxide is a proven option for these cases. On the other hand, if endodontic treatment was initiated more than 7 to 10 days after the avulsion or if active resorption is visible, the pulp space must first be disinfected before root filling. Traditionally, the reestablishment of a lamina dura (Figure 12-12) is a radiographic sign that the canal infection has been controlled. When an intact lamina dura can be traced, root filling can take place. The canal is cleaned, shaped, and irrigated under strict asepsis. After completion of the instrumentation, the canal can be filled by any acceptable technique, with special attention to an aseptic technique and the best possible seal of the filling material.

Permanent Restoration

Much evidence exists that coronal leakage caused by defective temporary and permanent restorations results in a clinically relevant amount of microbial contamination in the root canal after root filling. Therefore, the tooth should receive a permanent restoration at or soon after the time of filling of the root canal. As with the temporary restoration, the depth of restoration is important for its seal; therefore, 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 with the addition of dentin bonding agents are usually recommended in these cases. They have the additional advantage of internally strengthening the tooth against fracture if another traumatic event should occur.

Follow-up Care

Follow-up evaluations should take place at 3 months, at 6 months, and yearly for at least 5 years. If osseous replacement (ankylosis) is identified (Figure 12-15), timely revision of the long-term treatment plan is indicated. In the case of inflammatory root resorption (Figure 12-10), a new attempt at disinfection of the root canal space by standard retreatment can reverse the process. Teeth adjacent to and surrounding the avulsed tooth or teeth may show pathologic changes long after the initial accident. Therefore, these teeth should be tested at recall and the results compared with those collected soon after the accident.

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.[4] External resorption, that is much more common, is often misdiagnosed as internal resorption. Internal root resorption is characterized by resorption of the internal aspect of the root by multinucleated giant cells adjacent to granulation tissue in the pulp. For more details, see Chapter 15.

Internal root resorption is usually asymptomatic and is first recognized clinically through routine radiographs. Pain may be a presenting symptom if perforation of the crown occurs and the metaplastic tissue is exposed to the oral fluids.

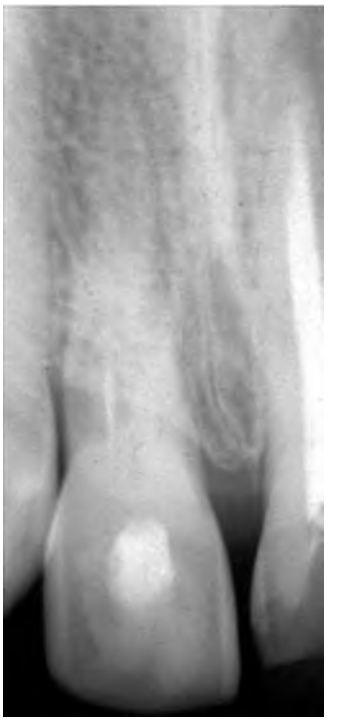

FIGURE12-15 Radiographic appearance of osseous replacement. The root acquires the radiographic appearance of the surrounding bone (without a lamina dura). Note that radiolucencies typical of active inflammation are **not** present.

For internal resorption to be active, at least part of the pulp must be vital, so a positive response to pulp sensitivity testing is possible. The coronal portion of the pulp is often necrotic, whereas the apical pulp that includes the internal resorptive defect remains 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 pathognomonic of internal root resorption. The pink color is due to the granulation tissue in the coronal dentin undermining the crown enamel (Figure 12-16). The pink tooth can also be a feature of cervical subepithelial external inflammatory root resorption that must be ruled out before a diagnosis of internal root resorption is made.

Radiographic Appearance

The usual radiographic presentation of internal root resorption is a fairly uniform radiolucent enlargement of the pulp canal (Figure 12-17A). Because the resorption is initiated in the root canal, the resorptive defect includes some part of the root canal space. Therefore, the original outline of the root canal is distorted. Only on rare occasions, when the internal resorptive defect penetrates the root and impacts the periodontal ligament, does the adjacent bone show radiographic changes.

Histologic Appearance

Similar to other inflammatory resorptive defects, the histologic picture of internal resorption is granulation tissue with multinucleated giant cells. An area of necrotic pulp is found coronal to the granulation tissue. Dentinal tubules

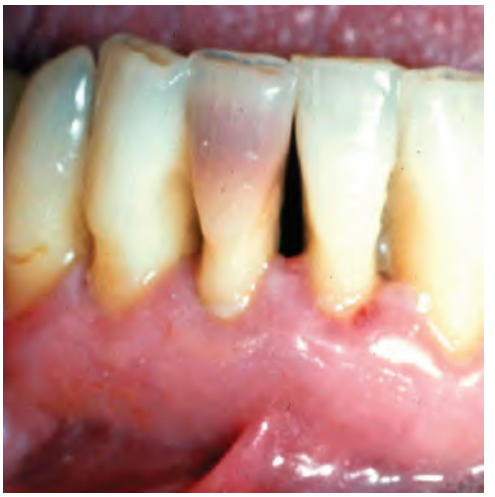

FIGURE 12-16 Pink spot associated with internal root resorption.

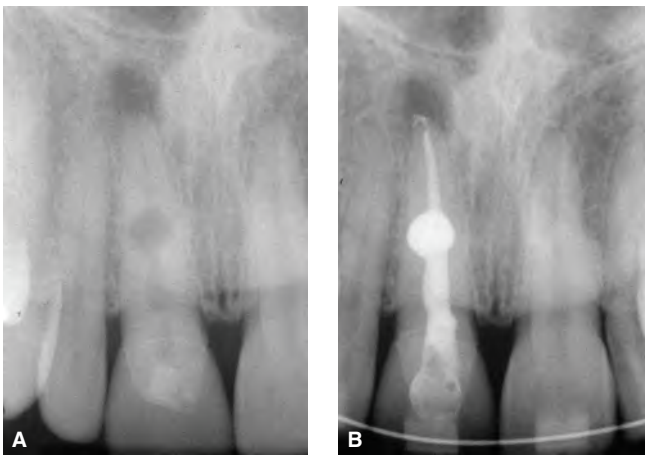

FIGURE 12-17 Internal root resorption. ***A.*** Radiograph showing the typical uniform radiolucency of internal resorption in the middle of the root of the left maxillary incisor. ***B.*** The area of internal resorption has been filled with softened gutta-percha and sealer. (Courtesy of Dr. Steven G. Morrow, Loma Linda, CA.)

containing microorganisms and communicating between the necrotic zone and the granulation tissue can sometimes be seen.[87,88] Unlike external root resorption, the adjacent bone is not affected with internal root resorption.

Endodontic Treatment

Treatment of internal root resorption is conceptually very easy. Since 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 is 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 instrumented 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 soft root filling technique (Figure 12-17B).

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Chapter 13

Crown-Originating Dental Fractures

Leif K. Bakland

A confusing area in dental diagnosis and treatment planning is that involving teeth with various types of fractures. These conditions are associated with many different patient histories, complaints, symptoms, clinical and radiographic findings, treatment options and preferences, and treatment outcomes.

Efforts have been made to categorize the various dental fracture entities. An early effort by Talim and Gohil[1] was rather complex and Silvestri and Singh[2] simplified it to a system based on complete and incomplete fractures. This was followed by the American Association of Endodontists (AAE) identifying five types of cracks in teeth.[3] No system has yet gained universal acceptance.

With the current emphasis on practicing evidence-based dentistry, there is recognition of the need for evidence-based guidelines for diagnosis, prevention, and treatment of cracks in teeth.[4] To that end, developing a useful classification of dental fractures would be beneficial. Developing such a classification system may take time, but the effort to accomplish that is needed. For a step in that direction, one may consider using the term "*Dental Fractures*" as an umbrella term to cover all the various entities of fractures that can occur in a tooth. Under that umbrella one may then divide into three categories fracture situations that have commonalities with each other (Table 13-1): *Crown-originating fractures* (COFs), *vertical root fractures* (VRFs), and *trauma-related fractures.*

COFs often seem to occur spontaneously and their etiology can be difficult to establish. The fractures usually start in the tooth crowns and progress into the roots in an apical direction (Figure 13-1). This category will be described in more detail.

VRFs are primarily found in endodontically treated teeth (Figure 13-2);[5] reports of VRFs in teeth with vital pulps have been published, but seem to be limited to a specific Asian population with some unusual chewing habits.[6,7] When VRFs occur in endodontically treated teeth, they frequently appear to originate in the apical segment of the root and progress coronally; they may, however, also occasionally

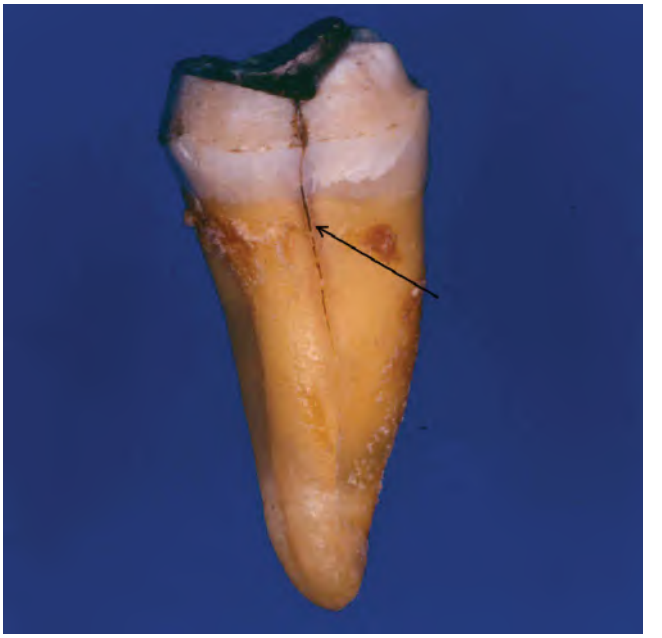

FIGURE 13-1 Extracted tooth shows common example of a crown-originating fracture (arrow). These fractures originate in the tooth crowns and propagate in an apical direction.

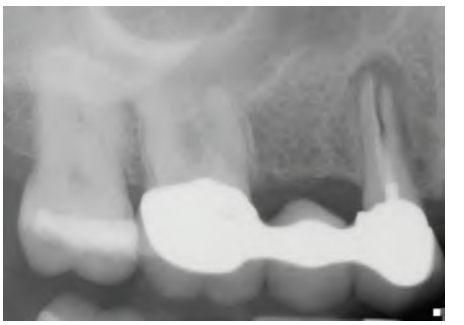

FIGURE 13-2 Radiograph showing a maxillary first premolar with a vertical root fracture. Characteristically, such fractures occur in endodontically treated teeth and the fracture orientation is bucco-palatally/lingually, thus they are usually readily apparent on radiographs.

TABLE 13-1 Three Subcategories of Dental Fractures. Each Subcategory Contains Clinical Situations That Have Commonalities With Each Other

Categories	Characteristics
Crown-originating fracture (COF)	Spontaneous fracture originating in the crown and progressing into the root in an apical direction.
Vertical root fracture (VRF)	A root-originating fracture that may begin anywhere in the root; it occurs primarily in endodontically treated teeth.
Trauma-related fracture	Tooth fracture of acute nature that may involve the crown or the root, or both.

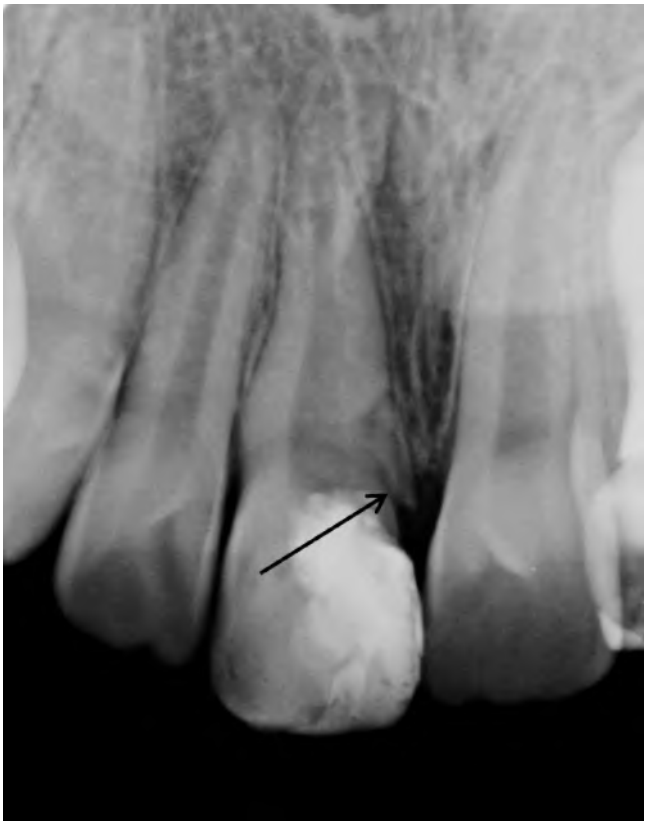

FIGURE 13-3 Radiograph showing a right central incisor with a cemental tear in the mesio-cervical area. The tooth had suffered a traumatic injury 2 years prior.

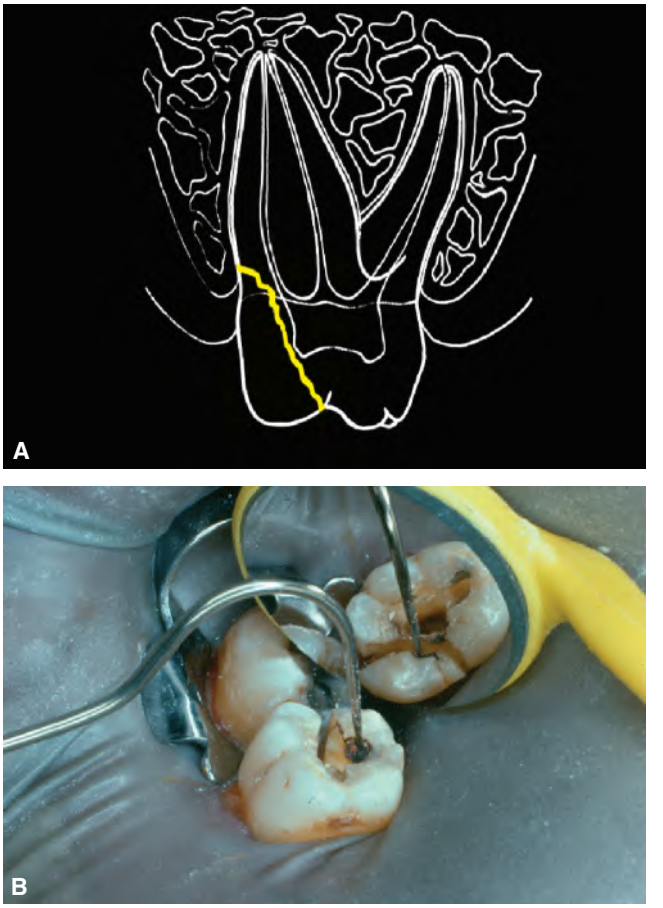

FIGURE 13-4 Cuspal fractures. ***A.*** Graphic illustration showing a common fracture orientation in cuspal fractures. The fracture line often skirts the pulp without invading it. ***B.*** Clinical example of a cuspal fracture; if the pulp is not directly involved, endodontic therapy may not be necessary.

develop in the cervical areas or mid-root and spread longitudinally.[5,8–10] Diagnosis is usually based on history (root canal treatment), radiographic appearance, and clinical findings. With few exceptions, treatment requires extraction. For more details, see Chapter 14.

Trauma-related fractures are, as the term implies, the result of acute traumatic events, such as may occur in sports, at work, during physical assault or in other accidental situations. These fractures are categorized as crown fractures, crown/root fractures, and root fractures. In addition a type of fracture not often addressed is what is referred to as cemental tears (Figure 13-3); they may have a traumatic etiology even if not always an acute traumatic event.[11]

CROWN-ORIGINATING FRACTURES

A possible term for fractures that typically originate in the crowns of teeth has been suggested.[12] The term COF could be considered a general term for the many others that have been used and would include both symptomatic and asymptomatic teeth. Many of the terms that have been used are fairly specific for certain fracture situations; it is however, often difficult to distinguish clinically among the various types of cracks.

Teeth have been subject to fractures for as long as humans have existed; a 6500 year old skull, recently discovered, was found to not only have a cracked tooth, but the crack had been filled with beeswax, probably to alleviate symptoms.[13] More recently, Gibbs[14] coined the term *cuspal fracture odontalgia* for those situations where teeth had become symptomatic and subsequently were found to have cracks extending under cusps and often resulting in the cusps fracturing off (Figure 13-4). The term *cracked tooth syndrome* (CTS) was suggested by Cameron[15] and it immediately became accepted by the profession for symptomatic teeth with the presence of fractures in the crowns. While generally accepted, using the term "syndrome" for such cases may be questionable. A syndrome is considered a combination of signs and symptoms that are indicative of a particular disease or disorder.[16] In teeth with CTS, there is a presumptive diagnosis based on the presence of consistent symptoms of pain to biting and cold temperature stimuli. Whether CTS qualifies as a "syndrome" is not certain, but it has become so well recognized over the past 50 years that it probably has to be accepted as a recognized term for symptomatic teeth with COFs.

Another term that has been used by many, including Brynjulfsen et al.[17] is *infraction*. This is a very descriptive term that indicates an incomplete fracture without

displacement of the fragments.[16] Similar terms are hairline fractures,[18] incomplete tooth fractures,[19,20] green stick fractures,[21,22] cracked teeth,[3] crown-root fractures,[23] and longitudinal tooth fractures.[3,24]

Two other terms represent the extremes of COFs. *Craze lines* are visible fractures that only involve enamel[4] (Figure 13-5). They may extend into subjacent dentin, but that is usually difficult to determine. They are not associated with symptoms and treatment is limited to esthetic concerns. The other extreme is the *split tooth*[3,21,23] that describes a fracture that extends through both marginal ridges usually in a mesio-distal direction, splitting the tooth completely into two separate segments (Figure 13-6). For convenience in this chapter, the term fracture will generally be used for these various COFs.

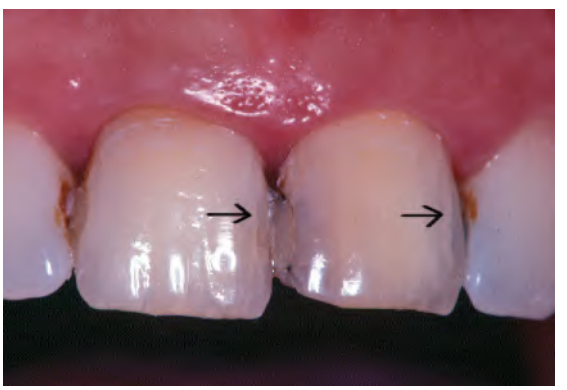

FIGURE 13-5 Maxillary central incisors with many craze lines (arrows).

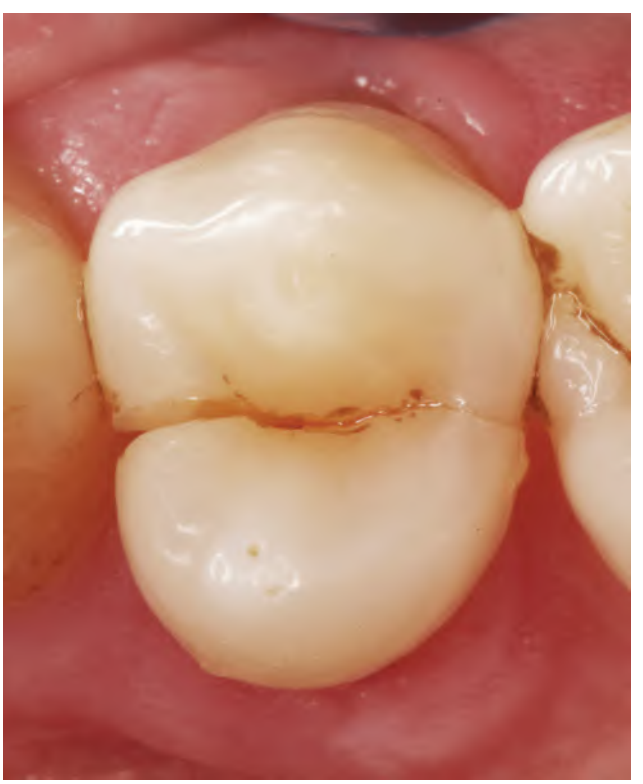

FIGURE 13-6 Maxillary premolar with a mesio-distal split.

CHARACTERISTICS OF CROWN-ORIGINATING FRACTURES

COFs are progressive in nature and may be described as discontinuity in the integrity of a tooth's hard tissues. They originate in the tooth crown on the pulpal side and propagate toward the dentino-enamel junction, and then further apically in to the root[25–27] (Figure 13-7). Some COFs are the cause of cuspal fractures and these often do not directly involve the pulp. Fractures that more centrally located on the occlusal surface do involve the pulp.[17] Some fractures are associated with symptoms while others are not.[28] As pointed out by Ritchey et al.[19] even the symptomatic teeth are difficult to localize since the symptoms are vague—discomfort to chewing and elevated sensitivity to cold food and drinks. Also puzzling is the observation that teeth with infractions, even if sensitive to biting, are often not distinguishable by percussion.[29] Diagnosis will be covered later in this chapter.

Incidence

The incidence of tooth COFs has not been extensively reported. Two studies[30,31] indicate that 2% to 3% of dental patients have infractions in their molars and premolars; most are asymptomatic, so the incidence of symptomatic teeth may be less than 1%. Other studies[28,32] show a more ubiquitous presence of teeth with cracks, though most are asymptomatic. A reasonable estimate would be five fractured teeth per 100 adults per year.[28]

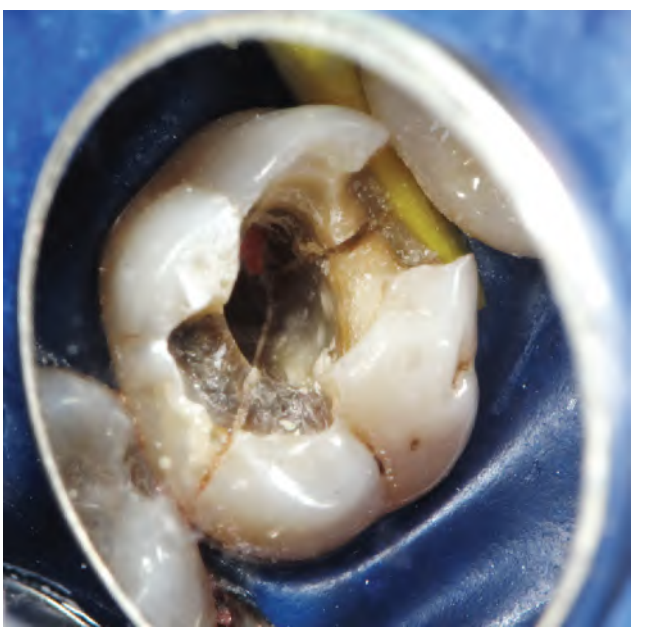

FIGURE 13-7 A molar with a very visible crown-originating fracture that has propagated from the pulpal aspect of the tooth to the external surfaces on both the mesial and distal sides of the tooth.

Molars and premolars appear to be the teeth most frequently involved in COFs.[15,17,31,33–35] The location of fractures is overwhelmingly in the mesio-distal direction (Figure 13-8), with only a few in the bucco-lingual direction (Figure 13-9), and some in a combination of both directions.[15,17,26,33,34] Brynjulfsen et al.[17] noted that 10% of the infractions were centrally located, indicating likely pulpal involvement. It is interesting that in their study, in maxillary teeth, the majority (70%) of fracture lines were situated toward the buccal tooth surface, while in the mandibular teeth they were inclined to be located toward the lingual surface, also at 70%.

Many reports appear to point to mandibular teeth, in particular the second molar as being most frequently involved,[15,33,36] but other studies identify maxillary molars more frequently.[17,35] Both of the latter studies involved specific groups of patients; the first group of patients had long histories of undiagnosed infractions and the second group was exclusively Korean. The influence of various factors on the incidence is not entirely understood. In a study where the findings of 12 previous studies were compiled, the following results emerged: 48% of cracked teeth were mandibular molars, 28% were maxillary molars, 16% were maxillary premolars, 6% were mandibular premolars, and about 2% were other teeth.[4]

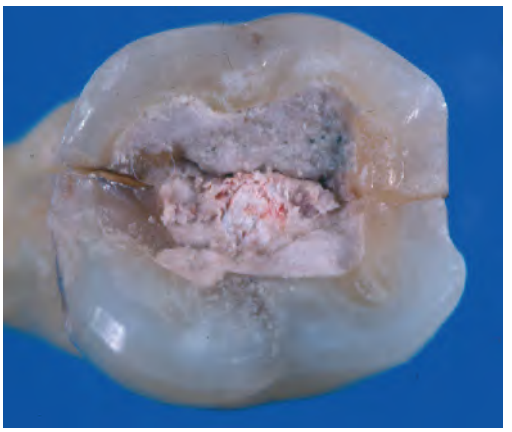

FIGURE 13-8 An extracted mandibular molar demonstrating the typical mesio-distal direction of crown-originating fractures. This is more common in mandibular teeth.

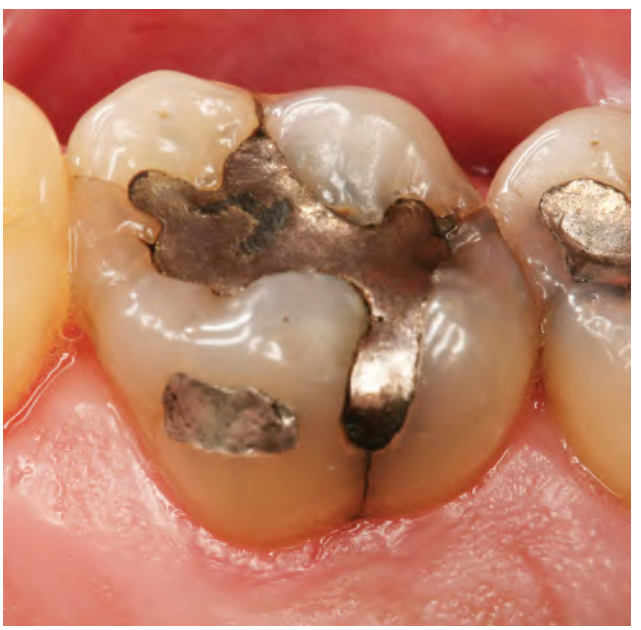

FIGURE 13-9 Maxillary molar with a crown-originating fracture running in a bucco-palatal direction; such fracture direction is seen more in maxillary teeth than mandibular ones.

In the rare case when an anterior tooth is involved with an infraction, it probably is the result of an injury from a sudden traumatic blow to the tooth.[37] (Figure 13-10)

One might intuitively expect teeth with restorations to be more prone to fracture than those without. The available evidence is not conclusive. Most investigators agree that teeth with restorations are more likely to develop infractions,[26,27,30,31,38–41] but there are also reports about nonrestored teeth with infractions.[21,34,35] Hiatt,[34] however, selectively chose teeth with no proximal restorations and Roh and Lee[35] studied a group of elderly Korean patients in which the majority of teeth were nonrestored. They speculated that diet and racial anatomical characteristics contribute, as well as restorative procedures, to the development of infractions. Age may also be a factor due to less elasticity and more brittleness in the teeth.[21]

An unusual type of COF has been described by Berman and Kuttler.[42] They used the term "fracture necrosis" for teeth with cracks that extended from the occlusal surface into the pulp and progressed to an external root surface. Pulp necrosis was found in the absence of restorations, caries, or luxation injuries, and was likely caused by longitudinal fractures extending from the occlusal surface and into the pulp.

When looking at the total picture of nontrauma related fractured teeth, COFs and VRFs, it appears that clinicians encounter VRFs more often than COFs.[6,43] However, because of the rather minimal epidemiological evidence, one cannot be certain about which type of fracture is more common.

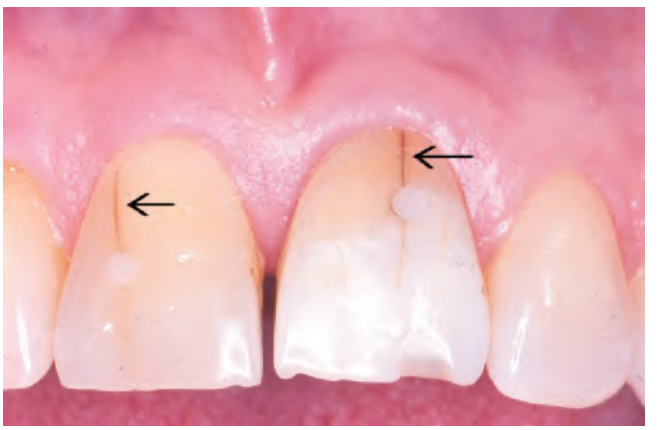

FIGURE 13-10 Two central incisor teeth with crown-originating fractures, probably resulting from a traumatic injury to the teeth years earlier.

Pain Characteristics

Pain is generally a complaint that draws patients' attention to problem teeth. That is true with respect to COFs as well.[14,15,17,19,22,26,27,34,44–47] As these authors have pointed out, pain to chewing is the most common finding in symptomatic tooth fractures.

Brännström[45] proposed that masticatory pain is due to sudden movement of dentinal fluid when the fractured tooth portions move independently, activating myelinated A-delta fibers in the pulp and creating a rapid, acute pain response. Stimulation of the A-delta fibers does not require inflammation of pulp tissue that explains why chewing discomfort occurs in the very early stages of infraction and appears to be a symptom common to all fractures, both cuspal fractures and those directly involving the pulp. Sutton[22] similarly suggested that the occasional sharp, momentary lancinating masticatory pain was due to bending and rubbing of dentin along fracture lines.

A unique pain response to chewing, experienced by many patients, is the pain that occurs when patients release the pressure of biting, variously referred to as "rebound" or "relief" pain.[26,27,47] This can be used diagnostically as suggested by Ailor[26] by having the patient bite on a moist cotton roll, (Figure 13-11) and if "rebound" pain occurs on release, there is a higher likelihood that one of the two opposing teeth, maxillary or mandibular, has an infraction.

Pain that is generated by temperature changes, particularly with application of cold stimuli, is also a common feature of teeth with infractions, but less common than chewing discomfort.[15,19,26,27,29,33,34,45,48] Ritchey et al.[19] noted an elevated response to cold, a condition likely due to pulpal inflammation caused by the infractions that contain bacteria and which communicate with the pulp.[27,29,45,48,49] Brännström[45] proposed that the inflammation sensitizes the pulpal C-fiber-receptors that respond to temperature changes. While hot stimuli are not often mentioned as associating with symptoms, they, no doubt, can cause pain, particularly as pulpal inflammation increases, making C-fiber receptors more sensitive and lowering threshold levels to both cold and hot stimuli.

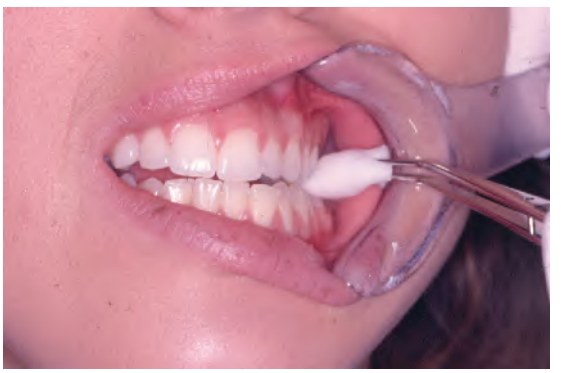

FIGURE 13-11 Biting test performed using a moist cotton roll. The patient bites as hard as possible until experiencing pain. If on release, the patient experiences a sudden, sharp pain that is what is described as "rebound" or "relief" pain, characteristic of crown-originating fractures.

It would be reasonable to expect teeth that are painful to mastication to also be hypersensitive to percussion stimuli. This does not appear to be the case with tooth infractions.[35] Swepson and Miller[46] have suggested that sensitivity to percussion occurs when the pulp becomes involved, which is not always the case in tooth infractions, at least not initially. Rosen[29] pointed out that until a fracture has propagated all the way from the pulp to the periodontal ligament (PDL), localization of an infracted tooth by percussion is very difficult. Thus, in cases of cuspal fractures in which the pulp is not directly involved, percussion may not produce a painful response.

It is well known that teeth with COFs may present with unusual pain conditions.[15,17,18,19,27,29] Ritchey[19] described the patients' chief complaint as vague discomfort to mastication, a description echoed by Cameron,[15] who included the observations that in addition to being vague, the pain complaint could mimic other conditions, such as trigeminal neuralgia. This was also reported by Caufield.[18] Further confounding the pain description in teeth with fractures is that the resultant pulpal inflammation can refer pain sensations to other areas served by the fifth cranial nerve with the result that pain can be experienced in other noninvolved teeth, and adjacent anatomical locations.[29] For more details, see Chapter 7.

Both Kahler et al.[27] and Brynjulfsen et al.[17] have made a point of the fact that tooth infraction pain can be associated with protracted histories of pain. The latter group reported a range of 3 months to 11 years in a group of 32 patients with undiagnosed long-standing orofacial pain from tooth infraction. They indicated that such protracted histories could lead to development of chronic orofacial pain with hyperalgesia. Another observation by Brynjulfsen et al.[17] was that, in some cases, the symptoms of pain were not provoked by chewing or application of cold stimuli, but rather were spontaneous pain experiences involving diffuse orofacial areas. Often, the pain would be felt anterior to the tooth responsible for the symptom when not referred to an anatomic area apart from teeth. The pattern of pain referral was that pain from mandibular teeth was frequently distributed to maxillary teeth, the neck, the ear, muscles of mastication, and temporomandibular joint (TMJ). Pain from maxillary teeth seldom projected to the mandible. An important observation was that the longer the duration of pain, the more diffuse it became and often led to headaches.[17]

Pain response to sweets in teeth with fractures has been reported[27,34,41,44]; this is probably related the presence of microorganisms located in the tooth fracture causing pulpal inflammation.[49] Since pain responses in teeth with COFs can mimic many other conditions, such as cariously affected teeth, it is prudent to maintain a high degree of suspicion when a general dental examination yields symptoms and examination findings that are contradictory.

Etiology

There are two primary factors predisposing teeth to cracks: natural predisposing features such as occlusal anatomy

and bruxism, and iatrogenic causes such as instruments used and cavity preparations.[4] Considering that restored teeth are often associated with COFs,[26,27,30,31,38–41] it has led many investigators to evaluate a number of restorative factors for clues to the etiology of tooth infractions. In fact several aspects of restorative dentistry have been identified as potential culprits. Most often-mentioned are excessively large and incorrectly designed restorations.[26,29,30,38,39–50] Seo et al.[24] showed that the use of nonbonded inlay restoration materials such as gold or amalgam increased the occurrence of longitudinal tooth fractures.

Another contributing factor has been the use of pins for supporting large restorations, especially self-threading and friction-lock pins.[29,38,50–52]

Abrasion, erosion and caries may also contribute to the development of fractures,[40] along with age changes in dentin,[26] oral habits,[26,29] and thermal cycling.[53,54] The latter is suspected of initiating infractions near the dentinoenamel junction and leading to a breakdown of the dentin-enamel bond. Resisting these damaging forces is the dentin's collagenous structure which prevents fractures from forming and propagating. As the dentin loses the ability for plastic deformation, it becomes more inclined to develop infractions, evidenced by the increase in infractions with age.[27]

In addition to cavity designs and size, the act of preparing a tooth for a restoration can also be hazardous. It is suspected that both the use of high-speed hand pieces[52] and course diamond burs[55] can lead to infractions. When course diamond burs are used, subsurface cracks develop from enamel into dentin. It is not clear if such infractions are more likely to progress further than the frequently observed enamel infractions. These craze lines can range from 1 to 20 μm in size and are usually filled with organic material that appears to stain quite readily.[54]

The act of chewing is also implicated in the development of fractures.[6,27,29,34,35,36,41,47,50] Masticatory forces from repeated occlusal loads and perhaps certain types of foods can contribute to this problem.[6,27,35,36,41,47,50] Certainly accidental biting on hard objects has resulted in tooth infraction and VRFs.[29,47] Bruxism and clenching often combined with a wedging effect of cusps in opposing fossae can be stressful on teeth.[6,34,35,36] Considering a biting force of up to 90 kg on first molars[41] it is obvious that these teeth are subjected to extensive stress. Yeh[36] coined the term "fatigue root fracture" to describe the stress on teeth combined with a course diet.

Acute trauma to teeth can also be responsible for tooth fractures.[29] Makkes and Folmer[37] described a situation in which injury to a central incisor in a child resulted in a VRF that was not discovered until years later.

Teeth are subjected to a variety of traumatic and stressful events, many resulting in damage such as COFs. In contrast to bone where repair of fractures can take place, repair in dentin and enamel fractures does not occur.

DIAGNOSIS OF CROWN-ORIGINATING FRACTURES

One of the difficulties in making a diagnosis of COF is that the cracks may be hidden below restorations;[17,24] (Figure 13-12) further, fractured teeth may not always be symptomatic or they may alternate between being symptomatic and asymptomatic.[24] Clinically, symptomatic tooth fractures are confusing because they often mimic symptoms from other conditions such as earache, sinusitis, temporomandibular joint dysfunction, and even trigeminal neuralgia and other neurological pain conditions.[17,18,30,33] When the problem is difficult to identify, it can lead to a long standing pain condition that becomes more diffuse, making localization of the offending tooth even more difficult.[17,27] Many reported cases of long standing histories of orofacial pain have been associated with the finding of cracks and infractions in teeth.[17,27,33,37]

Diagnosis of symptomatic tooth fractures is accomplished based on symptoms, clinical and radiographic findings, and history. A suspicion of COF should be considered when symptoms of a possible toothache are not readily connected to evidence of caries, periodontal disease, or recent trauma. The careful practitioner will proceed cautiously when a definitive diagnosis is difficult to establish to avoid acting on an incorrect diagnosis. Adding to these complications is the notion pointed out by Brynjulfsen et al.[17] that the longer it takes to make a diagnosis the more diffuse the pain becomes.

Symptoms

The most common symptoms associated with symptomatic tooth fractures are pain to chewing,[14,15,22,26,27,33,35,41] followed by pain to exposure to cold food and liquids,[15,19,26,27,35,41,48] and in some cases to sweets.[27,41,44] It is interesting to note the common absence of heat induced sensitivity.[21] This is probably related to early stages of pulpitis that may be present when teeth with COFs become symptomatic.

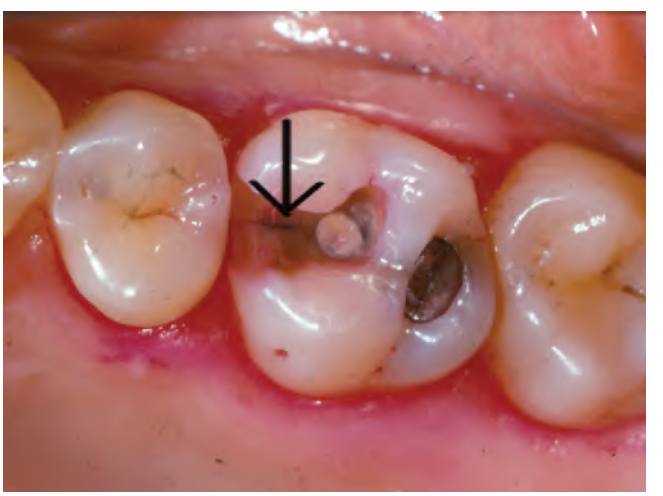

FIGURE 13-12 Maxillary molar with suspected crown-originating fracture. The mesio-occlusal amalgam had to be removed to identify the infraction (arrow) that has been highlighted by using red dye.

As has been noted, not all teeth with infractions are symptomatic.[31] Symptoms probably develop when fractures become populated with microorganisms that stimulate pulpal inflammation.[27,46,48,49] Localization may be possible when infractions reach the PDL, usually in the area of the crestal ligament where proprioceptive nerve fibers are present while not present in the pulp.[27,29]

Patients' histories of symptoms can be helpful in establishing a diagnosis of COF. Diffuse, protracted pain not associated with more easily diagnosed conditions of caries-induced pulpitis should at least attract a suspicion of tooth fracture.[17,27,29,30,33] Considering that symptomatic COF is in fact not a common dental condition, it should be considered when the more common conditions are ruled out.

Clinical Examination

Developing a definitive diagnosis of symptomatic COFs requires obtaining adequate information, both from the patient's history and from clinical examination. It usually begins with a chief complaint of pain to chewing, possibly elevated sensitivity to cold food and sweets. The absence of an obvious carious etiology should trigger a suspicion of a possible fracture and in most cases the following clinical examination procedures should help identify teeth with fractures.

Visual examination can reveal many instances of infractions, especially when aided by the use of transillumination[17,26] (Figure 13-13) and a dye such as methylene blue or red dye.[17,26,33,56] (Figure 13-12) The use of transillumination is dependant on the part of the tooth to be examined to be without restoration in order to allow the light beam to pass through the tooth; when it encounters a fracture line in the dentin, the light beam will bend and not pass through and the tooth structure opposite to the fracture line will be dark.[26] Additionally, to aid in visualization, the use of a microscope can be very valuable,[17,26] although magnification alone may not be a predictable way of detecting fractures.[57] However, in combination with the application of a dye, infractions become quite visible.[17]

Since existing restorations may hide infractions, it is often necessary to remove the filling to reveal the fracture lines.[17,26] (Figure 13-12) This becomes an important step in the clinical examination when the symptoms are vague and location of pain is diffuse and not localized. Brynjulfsen et al.[17] methodically removed restorations, one tooth at a time, to locate teeth with infractions in patients with long standing, undiagnosed orofacial pain.

An important step in the clinical examinations is the application of a **biting test.** Various techniques have been recommended, such as biting on Burlew wheels,[33] rubber wheels,[25] cotton tip applicators,[25] moist cotton rolls,[26] (Figure 13-11) and commercial biting applicators such as ToothSlooth (Professional Results Inc., Laguna Nigel, CA) and Fractfinder (Denbur, Oak Brook, IL). Ailor[26] has suggested that to differentiate between biting pain from restorations with microleakage and pain from tooth infraction, the ToothSlooth can be positioned so that pressure is placed on the filling and then the tooth cusps to determine the source of the symptoms (Figure 13-14).

A very significant response to biting is when pain is experienced on release of biting pressure[26,27,47] and referred to as either "rebound pain"[27] or "relief pain."[47] Kahler et al.[27] explain the pain associated with release of pressure results from fluid movement as the crack rapidly closes.

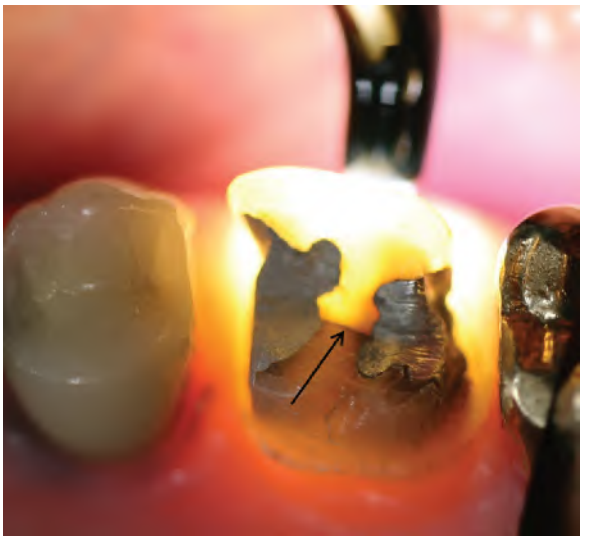

FIGURE 13-13 Example of transillumination highlighting a mesio-distal infraction (arrow) in a maxillary molar being prepared for a crown.

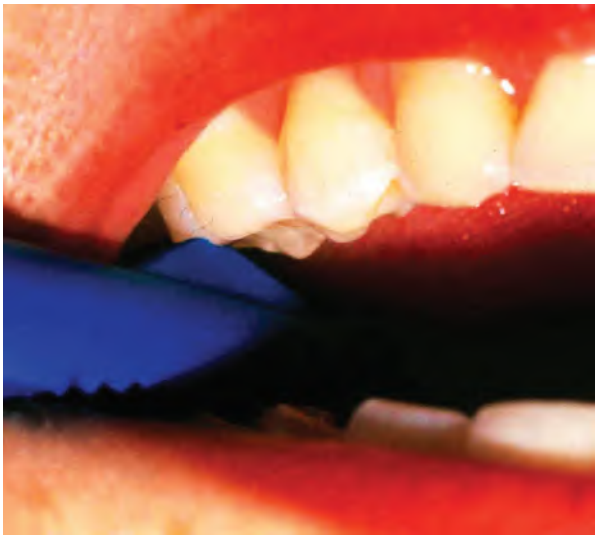

FIGURE 13-14 Illustration of the use of a ToothSlooth to attempt to differentiate between a fractured cusp versus a leaky restoration as the source of pain during mastication. The point of the ToothSlooth is being placed alternately on a cusp or on the surface of a restoration.

The moist cotton roll has been very effective for the author in stimulating rebound pain, providing a good clue to the tooth in question.

Cold stimulus application and ***electric pulp testing*** (EPT) will provide information about the status of the pulp and there is evidence that teeth with fractures respond at lower threshold levels to cold and EPT stimulation compared to nonfractured teeth.[19,48] Since any tooth with pulpal inflammation is likely to have lowered pain thresholds, it is not particularly significant that teeth with infractions also do. But it can add to the total information about the tooth.

Cameron[33] has suggested the use of a thin sharp **explorer** to probe around the cervical circumference of teeth suspected of having infractions, particularly in interproximal areas not readily visible if the tooth crown has a large or full coverage restoration (Figure 13-15). Both the "click" of the explorer tip encountering the fracture, and perhaps the patient's response, as in touching a sensitive spot on the root surface, can provide a clue. Another clue can be found by using a sharp explorer at margins of large restorations—a sharp pain may be elicited if a crack is present.[21]

Percussion sensitivity in teeth with infractions is not as common as biting sensitivity.[35] An explanation for this may be that in teeth with COFs the infractions typically originate internally and propagate peripherally,[27] so a tooth with infraction is not likely to be identified by percussion until the fracture extends to involve the PDL.[29]

Periodontal probing is recognized as an indispensable part of dental examinations in general and that also applies when examining teeth with potential fractures.[26,34] Hiatt[34] described periodontal probing to reveal narrow pockets adjacent to fracture lines, and Ailor[26] has suggested that if the probing extends below the alveolar crest, the tooth is not suitable for restoration (Figure 13-16). Since the pockets adjacent to infractions are extremely narrow, in contrast to those adjacent to VRFs in endodontically treated teeth, it is usually necessary to anesthetize the tissues surrounding the tooth prior to probing.

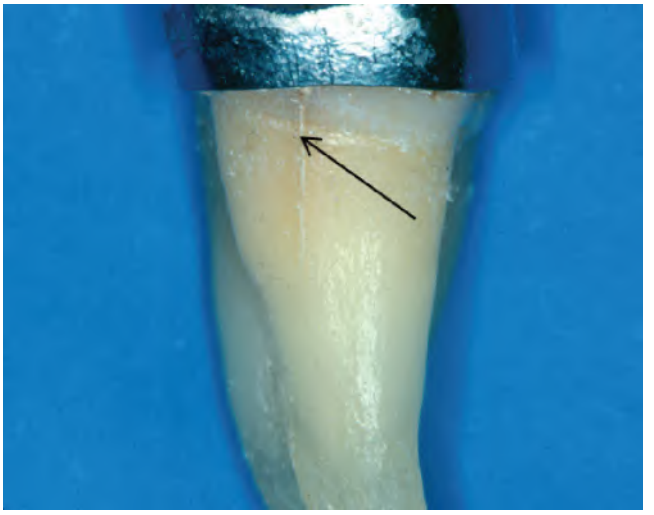

FIGURE 13-15 Infractions located below crown margins can be difficult to detect. Using a sharp explorer to probe around the cervical circumference of teeth suspected of having infractions, they may be identified by the "click" when the explorer encounters the fracture (arrow). Some patients may also feel a sharp, sudden pain at that time.

Radiographic Examination

It is generally recognized that radiographic examinations, while obviously necessary for a number of reasons, do not always provide diagnostic information with respect to COFs.[17,26,27] This is because the infraction lines usually run mesio-distally in the majority of cases[33] and the X-ray beam travels perpendicular to the fracture lines. If, however, the tooth infraction is located in a bucco-lingual direction, the X-ray beam will produce a clear image of the fracture line on the film or sensor (Figure 13-17).

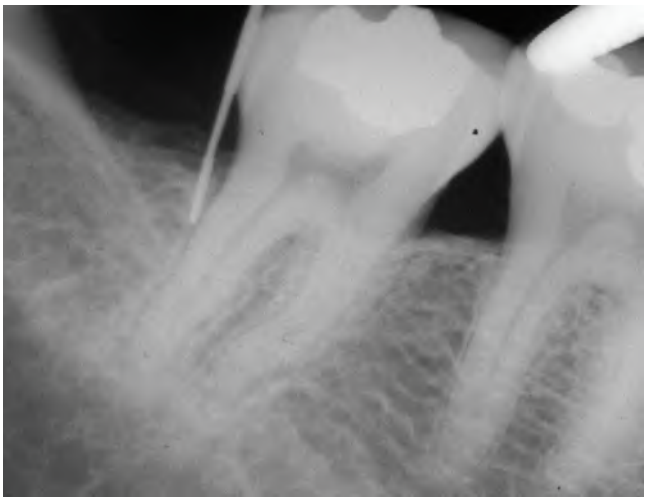

FIGURE 13-16 Illustration of periodontal probing in the narrow pocket adjacent to a crown-originating fracture. Such probing, that must be done using local anesthetic, can reveal approximate extent of the fracture's propagation down the root.

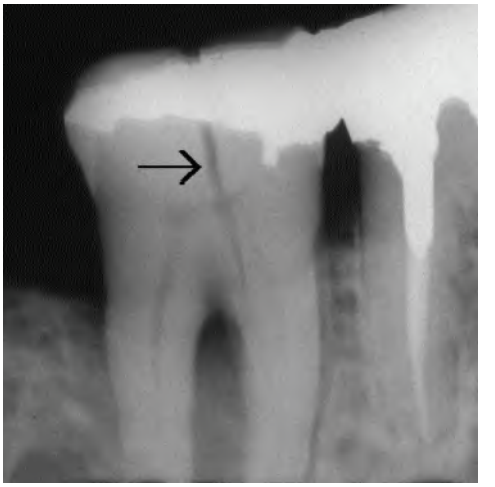

FIGURE 13-17 Radiograph of a mandibular molar showing a fracture (arrow) located in a bucco-lingual orientation. Because the X-ray beam runs parallel to the fracture, it becomes readily visible on the film.

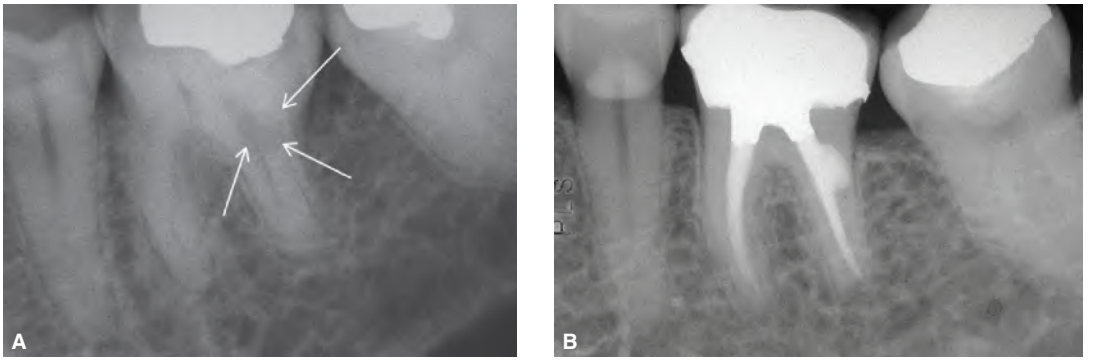

FIGURE 13-18 Illustration of infraction-related internal resorption. ***A.*** The mandibular molar had developed a symptomatic crown-originating fracture located on the distal aspect of the tooth. The radiograph shows the location of the resorptive lesion (arrows). ***B.*** Radiograph taken after endodontic treatment and placement of a full coverage crown. The resorptive lesion was connected to the still vital pulp at the point where the infraction entered the pulp; there was no external communication with the lesion.

Cone beam computed tomography (CBCT) has made radiographic examination of dental conditions of almost every type more easily visualized than ever before. Certainly VRFs in teeth are easily recognized on CBCT images.[7,58–60] This technology is less applicable to diagnosis of teeth with COFs. If an infraction is strongly suspected but the clinical examination lacks enough information for a diagnosis, CBCT may be indicated, selecting a 0.2 voxel system because of the small diameter of the infraction.[60] The use of CBCT for COF diagnosis should be very selective, as CBCT should not be used if other less risky avenues are available.[61]

One pathologic entity connected with COFs that can be detected radiographically, is the rare instance where bacteria in the fracture has stimulated clastic activity in the pulp, resulting in an internal resorptive lesion.[62] (Figure 13-18) This type of internal resorption is unique in many respects in that it appears to occur adjacent to the pulp space and may be mistaken for invasive external resorption. The presence of an infraction, associated with a resorption defect and with no communication of the resorptive defect with the periodontal tissues, confirms the diagnosis.

Future Diagnostic Options

While many tooth fractures can be diagnosed quite readily with just a few examination steps, others are often very difficult to detect and require extensive efforts.[17] There is a need for additional means to detect fractures in teeth; various avenues are being explored including the application of **ultrasound.** This modality may have potential for complementing radiography as an imaging technique by identifying physical discontinuances (infractions) in hard tissues such as enamel and dentin. The application of ultrasound is used in industry for a similar purpose and would, if developed in dentistry, be a great help in the detection of tooth fractures; it has been shown to be capable of imaging cracks in simulated tooth structure.[63]

Another ultrasonic technique is using ultrasonic power to heat the fracture line thus making it detectable by thermography.[64] For more information, see Chapter 9.

Differences Between COFs and VRFs

There are fairly distinct differences between teeth with COFs and those with VRFs. In the former the initial symptoms often are sudden, sharp pains on chewing, and occasionally "rebound" pain during chewing of certain foods. Patients with VRFs are more likely to complain of gradual onset soreness in the fractured teeth.

Another difference is the matter of localization; teeth with COFs are often difficult to identify while those with VRFs can be more readily identified. While chewing may produce responses in both situations, percussion sensitivity is more pronounced in teeth with COFs.

Radiographically, teeth with VRFs are often readily recognized while those with COFs are rarely identified on radiographic films or sensors; this is because the usual direction of fractures is in a mesio-distal direction in teeth with COFs while most often in a facio-lingual direction in teeth with VRFs. Thus in the latter situation, the X-ray beams travel parallel to the fracture lines and perpendicular to the fractures in the former.

TREATMENT

Any discussion of the treatment of teeth with COFs must include an acknowledgement that neither dentin nor enamel can be permanently reunited once a fracture line develops. Treatment efforts therefore are attempts (some better than others) to prevent separation of the hard tissue entities, and perhaps to keep bacteria from colonizing the fracture spaces, however that may be impossible.[49] The goal of treatment is to bind a tooth with infractions together, but one has to expect that in time this discontinuity in the integrity of the tooth structure will progress toward separation such as seen in split teeth and cuspal fractures. Further, root canal therapy, while reducing or eliminating symptoms, will not change the fact that a tooth with a fracture is a weakened tooth that likely will not last as long as a nonfractured tooth.[42,65]

The need for evidence-based treatment guidelines have been pointed out.[4] While these are not yet available,

endodontists appear to be in agreement that many teeth with fractures can be treated,[66] but it is not clear if all teeth with fractures need root canal therapy. The lack of clear understanding in this regard is probably related to the difficulty in determining if an infraction communicates directly with the pulp, in which case endodontic treatment would reasonably be a part of the overall treatment for the tooth, or the fracture is a cuspal fracture that may not communicate with the pulp and the pulp may not be involved.

Using the commonly accepted criteria for pulpal diagnosis, Krell and Rivera[67] reported the outcomes of symptomatic fractured teeth that were initially diagnosed with reversible pulpitis and treated with full coverage restorations. The outcomes suggest that, if an infraction is identified early and the diagnosis is reversible pulpitis and a crown is placed, root canal treatment will be necessary in about 20% of the cases.

One approach that can help in deciding if endodontic therapy for a tooth with COF is necessary is a method followed by some clinicians.[17,26] If the tentative diagnosis is *reversible pulpitis*, the tooth can be stabilized with an orthodontic band for about 2 weeks (Figure 13-19). If the initial pulpal diagnosis is *reversible pulpitis* (based on no lingering pain to cold and no spontaneous, severe pain) a 2-week waiting period will provide useful information. If the symptoms have subsided after placement of the orthodontic band, the patient may be offered the option of only placing a restoration that binds the tooth together, such as a full crown, with the understanding that the tooth may later need root canal therapy.[67] The reason for waiting a while after placement of the orthodontic band is because it takes some time before cold sensitivity subsides. For instance, Davis and Overton[25] found that it took 2 weeks for cold sensitivity to subside after restoring teeth with bonded amalgam restorations.

When fractured teeth have *irreversible pulpitis* or *pulp necrosis*, the need for root canal treatment is self-evident if they are to be retained. Root canal treatment will obviously eliminate pulpal pain and sensitivity to temperature changes and to sweets; one should not, however, expect all fractured teeth to be free of chewing pain after root canal therapy. Pain to mastication is associated with inflammation in the PDL. Such inflammation is generated by bacteria in the infractions that extend from the coronal area apically.[27,29,34,49] The problem of not being able to predict if pain to chewing will cease after root canal treatment and restoration, can be addressed again by the recommendation made previously to first place an orthodontic band to measure the response to binding the crown together, followed by root canal therapy. If the patient is comfortable with the tooth the treatment can be completed; if not, extraction becomes the alternative treatment.

Treatments designed to bind cracked teeth together include the use of adhesives,[20,68–70] amalgam with retention on both sides of the infractions[25] and full coverage crowns,[17,25,26,65,71] and bonded composite overlays.[72] Experimental approaches using lasers (CO_2, Nd:YAG) have not produced any promising results yet.[73]

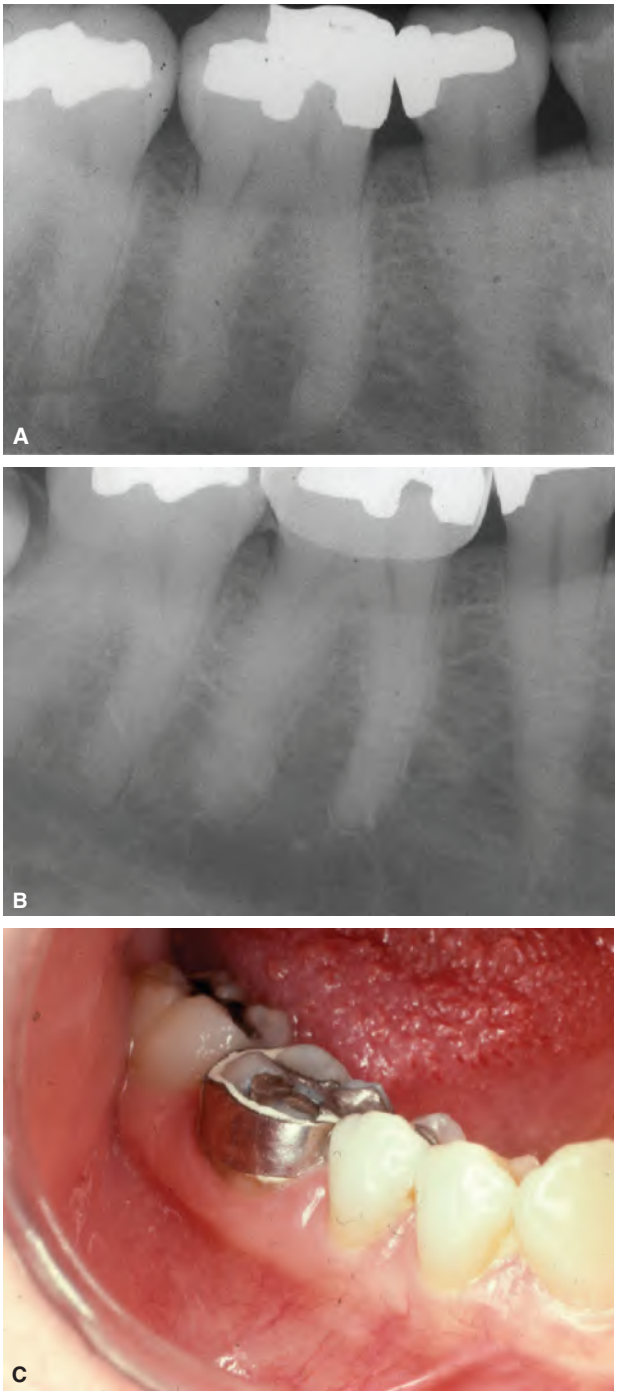

FIGURE 13-19 Diagnostic banding of a tooth with crown-originating fracture (COF). ***A.*** Radiograph of mandibular first molar with a symptomatic COF on the distal aspect of the tooth. Since the fracture line is in a mesio-distal orientation, it is not visible radiographically. ***B.*** With a pulpal diagnosis (based on symptoms and clinical findings) of reversible pulpitis, a band is cemented to bind the tooth together. ***C.*** Photograph taken 2 weeks later when the patient reports complete absence of symptoms. The patient can now be offered the option of a full coverage crown with the information that about 20% such teeth may need endodontic treatment later;[67] some patients may choose to have root canal treatment done prophylactically before crown placement.

Prognosis

The outcome of treatment for teeth with infractions has not been extensively reported. Cameron[33] reported a 75% success after 10 years following placement of crowns, and Guthrie and Di Fiore[71] found that 24 of 25 teeth restored with acrylic crowns were asymptomatic after 1 year. Brynjulfsen et al.[17] achieved pain relief in 90% of their patients after protective restorations were placed on teeth with infraction (endodontic therapy was included when indicated), and Tan et al.[65] had an 85% survival rate 2 years after protective crowns were placed. The available data is insufficient to use as a basis for giving individual patients odds on tooth survival for a specific number of years. Patients must be fully informed of the uncertainty based on lack of data. What one can recognize is that in certain situations the prognosis is poor: terminal teeth, teeth with periodontal involvement related to infractions, and teeth with multiple infractions.[65] Treatment recommendations and informed consent, based on best evidence, suggest that clinicians search what is available in the literature, include the clinician's own experience, and seek the patient's own preferences.

PREVENTION

A number of suggestions have been made toward reducing the incidence of tooth fractures. They include cavity design and size,[25,26,29,38,39,50] proper design of intra-coronal cast restorations,[39,50] avoiding use of self-threading and friction-lock pins, [29,38,50,51] avoiding extensive use of course diamond burs and vibrating high-speed hand pieces,[26,55] and restoring teeth with attention to occlusion to avoid interference from working premature contacts.[26,29] It may also be beneficial to advise patients about potential damage from oral habits (pipes, pencils, finger nails)[26,29] and the stress excessive repetitive heavy mastication can produce in teeth.[6,36,41]

Yeh[36] coined the term "fatigue root fracture" to describe the result of consuming a course diet over a long period of time in a group of Chinese patients. Many of them developed VRFs in often nonrestored teeth with no history of endodontic treatment. The biting force on first molars is up to 90 kg[41] and if there is sudden external trauma, such as biting unexpectedly on something hard in the food, an infraction may result. Kahler et al.[27] described how infraction can occur from "cyclic fatigue" and repeated occlusal loads. Similar to all body parts, teeth are not indestructible.

With respect to care of teeth, a question sometimes raised is, is it damaging to chew on ice? The dental literature is not very informative on this topic. Brown et al.[53] used thermal cycling, alternating exposure to very cold and very hot stimuli on extracted teeth, and reported extensive cracking in the teeth. It is not clear if that information can be applied to the question of ice-chewing, but until more information becomes available, it seems reasonable to advice against chewing on ice.

SUMMARY

The incidence of COF does not present a major problem in dentistry,[30,31] but diagnosis of COF can be difficult when symptoms and clinical findings are not definitive.[15,17,18,19,26,27,33,34] Infractions or cracks in teeth are incomplete fractures, most often originating in the crowns, and will, if untreated, lead to cuspal fractures or split teeth.[6,15,23,25–27,29,35] Cuspal fractures usually do not communicate directly with the pulp and such teeth may not need endodontic treatment. Infractions may, however, propagate from the pulp toward the dentin-enamel and dentin-cementum junctions. In such cases the pulps are usually irreversibly involved requiring endodontic treatment.[3,25,35,65] Restorative treatment involves procedures that provide a binding effect on the tooth infraction; this can be accomplished by using amalgam restorations that incorporates retention on both sides of the infractions,[25] amalgam in combination with bonding agents or full coverage cro wns,[17,25,26,65,74] or composite overlays.[72] Today, however, use of amalgam is less frequent.

Adequate data is not available to determine prognosis for symptomatic teeth with infractions; when such teeth have received treatment, either restorative alone or in combination with root canal therapy, they have been reported to survive for many years.[33,65,71] Patients however, need to be informed that long-term retention is questionable. However, in spite of unfavorable long-term outcome, retaining a tooth with COF may still be an excellent option for many patients.

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Chapter 14

Vertical Root Fractures

Aviad Tamse, Louis H. Berman

Vertical root fracture (VRF) is a fracture originating in the root of a tooth that can present a serious treatment complication.[1,2] It occurs primarily in endodontically treated teeth, but has also been observed in intact nonendodontically treated mesial roots of mandibular molars, mostly in Chinese population.[3] According to the American Association of Endodontists' classification, VRFs are considered a subcategory of longitudinal root fractures since they are chronic in nature and progress longitudinally over time.[2,4,5] VRFs originate in the root at any level and progress either coronally or apically.[5] From a horizontal perspective, the fracture originates in the root canal wall, mostly on the buccal or lingual surface, and extends outwardly to the external root surface.[4] It can involve one root surface (incomplete VRF), or both root surfaces (complete VRF).[4,5] Complete VRF often becomes an eventual buccal-lingual fracture (Figure 14-1 A–D).

Because VRFs originate in the root of the tooth, they are different from other longitudinal fractures that occur subsequent to impact trauma, such as "craze lines," "fractured cusps," "cracked teeth," or "split roots."[4] These fractures originate in the crown of the tooth and progress longitudinally in an apical direction.

The clinician should be aware that when there is no obvious cause for a chronic symptomatic pulpitis or pulp necrosis, especially when there is an intact crown with either no caries or with a shallow restoration, one should suspect a fracture in the crown.[6] Enhanced magnification and illumination are imperative in such cases, and the patient should be informed of a possible decrease in prognosis. In cases whereby these fractures were not recognized prior to root canal therapy, the patient is likely to return with typical signs and/or symptoms of a VRF (Figure 14-2 A–C). Therefore, the clinician should meticulously evaluate the clinical and radiographic appearance of such of a tooth prior to initiating root canal therapy.

VRFs in endodontically treated teeth can present a frustrating diagnostic and treatment-planning dilemma both for the patient and clinician. The clinical signs and symptoms may be difficult to differentiate from nonhealing endodontic treatment or chronic periodontal disease.[4,7–9] Often, the definitive diagnosis is only made years after the endodontic and restorative procedures have been completed or when the tooth is extracted.[4,8,10,11] Prevention of VRFs is difficult since the etiology is multifactorial.[8,12,13]

Subjectively, there is a higher reported incidence of fractured crowns and VRFs in recent years.[14] This may be attributed to the increased awareness of clinicians that endodontically treated teeth may be more prone to fractures. It is also possible that restorative and orthodontic treatment may be directing an increase in occlusal prematurities. Alternatively, changes in dietary habits and ingredients may be altering the way teeth are developing, possibly making them more brittle or prone to fracture. However, presently there is no objective explanation for the possible increased incidence of VRFs. Seo et al.[14] evaluated 107 extracted endodontically treated teeth and found 13% incidence of VRFs. Fuss et al.[15] and Coppens et al.[16] showed an incidence of 11% and 20% VRFs, respectively. In teeth referred for extraction, the incidence was higher.[17,18]

CLINICAL DIAGNOSIS AND PATHOGENESIS

When present, it is imperative that a diagnosis of a VRF be made as soon as possible. A direct correlation was found between bone resorption around a VRF and time without treatment.[19] As bone resorption increases over time, it may compromise other roots of the same tooth or adjacent teeth. This bone loss may also compromise the subsequent placement of an endosseous implant in the area of the extracted tooth. When the diagnosis of a VRF is made, the tooth or offending root(s) should be extracted as soon as possible, assuming patient compliance.

Achieving an accurate and timely diagnosis of a VRF in an endodontically treated tooth can sometimes be challenging. Fractures may be obscured by obturation material or there may be no significant periodontal or radiographic findings to suggest a VRF.[20] In addition, typical signs and symptoms of a VRF, such as spontaneous dull pain, pain upon mastication, tooth mobility, presence of a sinus tract, deep periodontal probing defects, periodontal-type abscesses, and bony radiolucencies, are often similar to those observed in nonhealed endodontic treatment, reinfected root canals, and primary periodontal disease.[1,4,7–9,21]

According to the 2008 AAE Communiqué,[5] there is a specific combination of signs and symptoms that, when present in the endodontically treated tooth, are considered "pathognomonic" for the presence of a VRF. These parameters are:

1. Presence of a sinus tract.
2. Existence of a narrow and deep isolated periodontal defect.

However, this "pathognomonic combination" is not necessarily present in all VRF cases. According to Tamse et al.[7] this combination of clinical finding was only observed in 23.9% of diagnosed VRFs. In a recent study,[21] this "pathognomonic

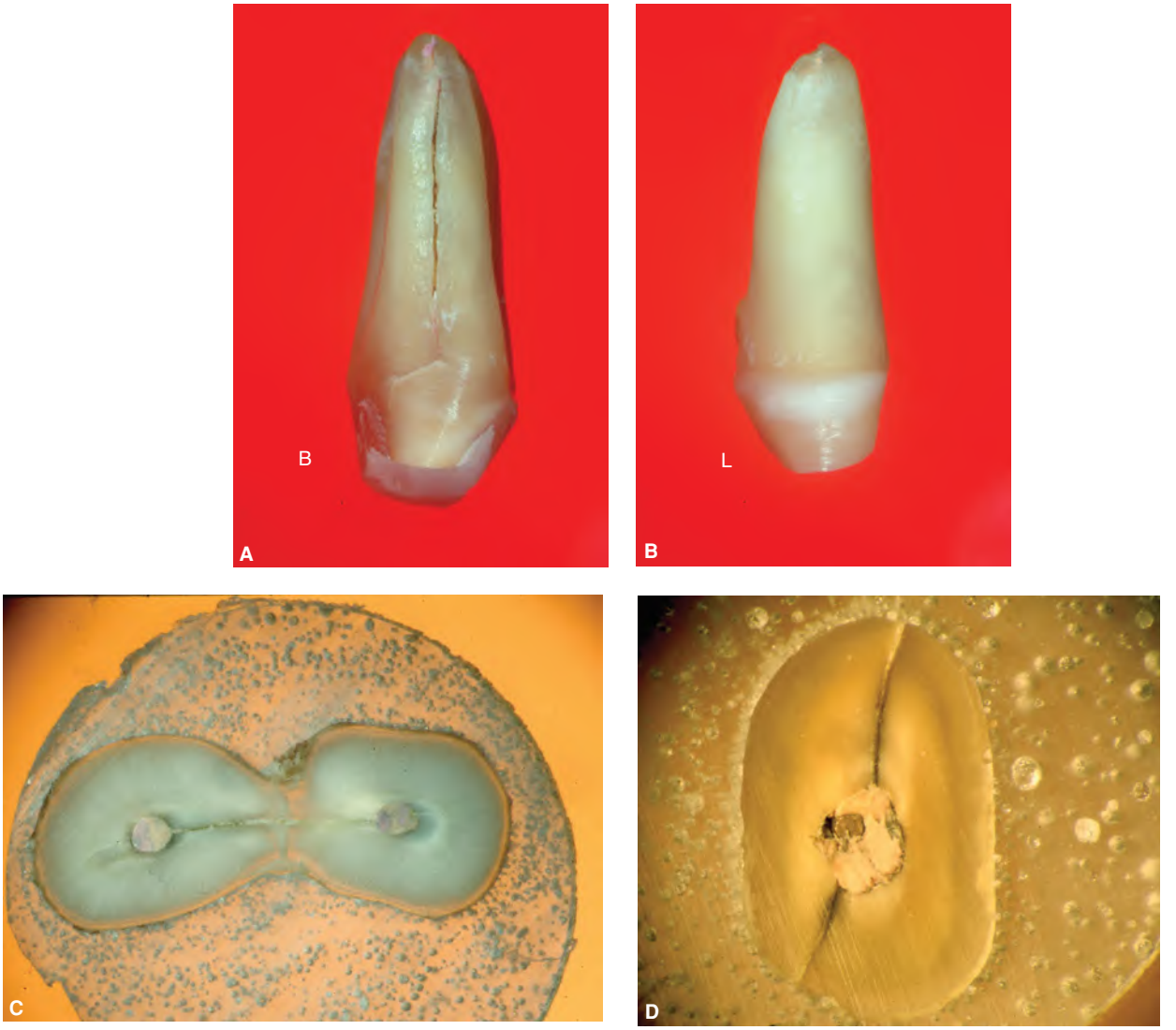

FIGURE 14-1 ***A***. Buccal aspect of incomplete VRF from apical to coronal in an extracted maxillary premolar. ***B***. Incomplete fracture does not involve the lingual aspect. ***C***. Incomplete fracture in a maxillary premolar. ***D***. Complete fracture in a mandibular premolar.

combination" was only observed in 43.8% of vertical root fractured cases. (Figure 14-3 A,B)

Although there are several radiographic features associated with VRF's,[22,23] they are not part of the AAE's "pathognomonic combination" but rather described by the AAE as "radiographic evidence varies."[5] The typical pattern of bone loss associated with a VRF is a dehiscence of the buccal plate adjacent to the fracture site, followed by loss of the interproximal bone of the affected root.[19] (Figure 14-4 A,B) This process continues unless the tooth, or root, is extracted. These radiographic changes are often observed on 2-D radiographs; however, 3-D radiography, that is, cone beam computed tomography (CBCT), is often necessary to better observe these bony changes.[24] For more details see Chapters 9C and 10.

Two typical clinical signs that can help the clinician make an accurate diagnosis of a VRF are

1. Deep and often narrow periodontal probing defect (Figure 14-5 A,B)
2. Sinus tract positioned more coronally in the attached gingiva (Figure 14-6 A,B)

In four retrospective clinical studies of teeth with VRFs, deep probing defects were found in 64% to 93% of cases; a more coronally positioned sinus tract was present in 13% to 35% of cases.[7,21,25–27]

This periodontal presentation and position of the sinus tract are expected considering the dynamics of the host response to the fracture. Once the fracture progresses to the outer surface of the root, the periodontal ligament (PDL) space becomes involved and local inflammation develops. This is due to bacterial invasion of the fracture from the oral cavity that extends into the PDL space.[10] The local inflammatory process results in development of vertical bone loss and formation of a narrow periodontal defect. Over time, an abscess develops that is similar to any other form of a chronic periodontitis.[7,25–27] An exploratory surgical procedure is highly recommended

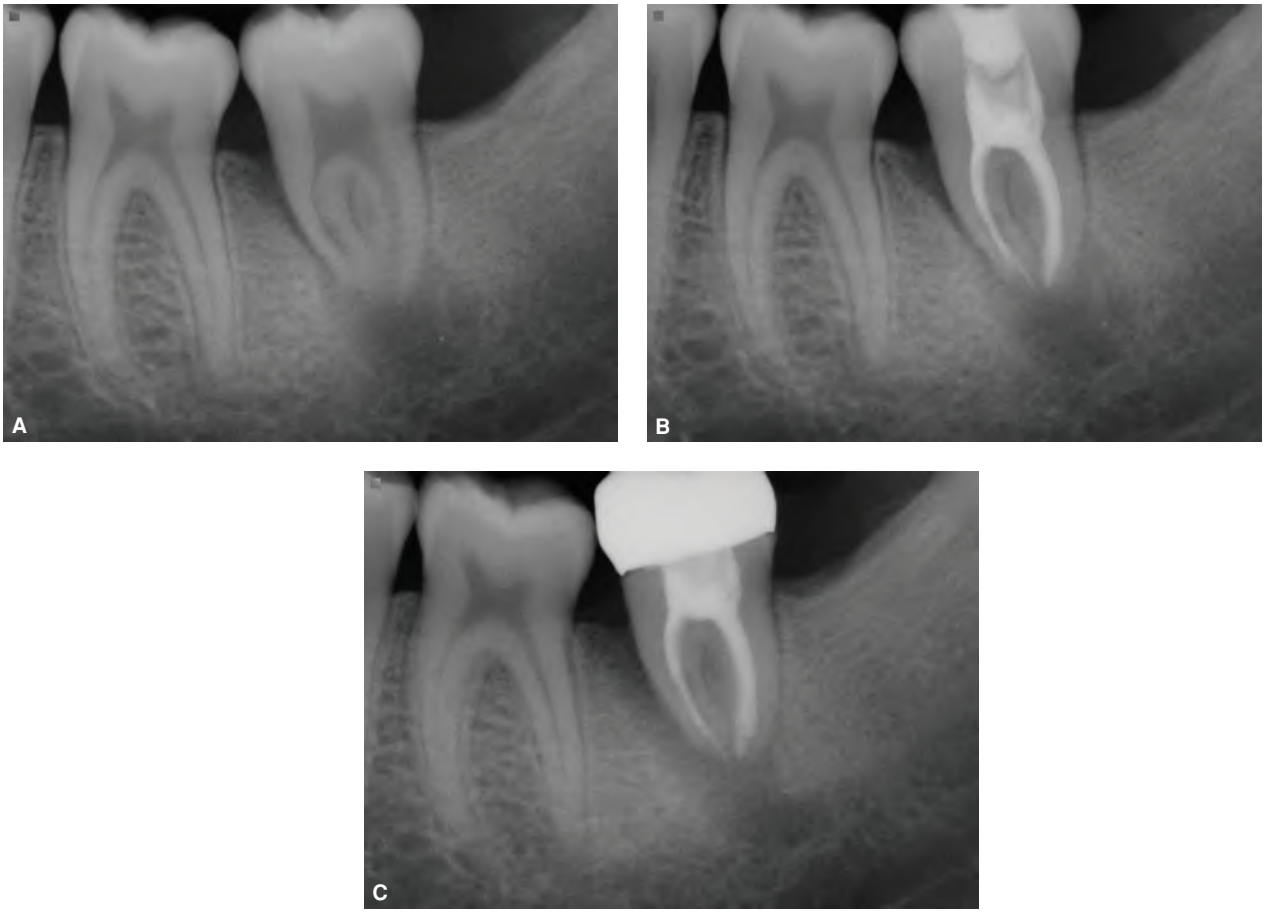

FIGURE 14-2 ***A***. Lower left second molar with nonvital pulp and no apparent reason for necrosis other than possible root fracture. Note extensive periapical bone loss with some vertical bone loss on the mesial aspect of the root. ***B***. One year after endodontic treatment. Note increase in periapical and vertical bone loss. ***C***. Two years after endodontic treatment a substantial increase in periapical and vertical bone loss is observed. The tooth was extracted and the presence of a vertical root fracture was confirmed.

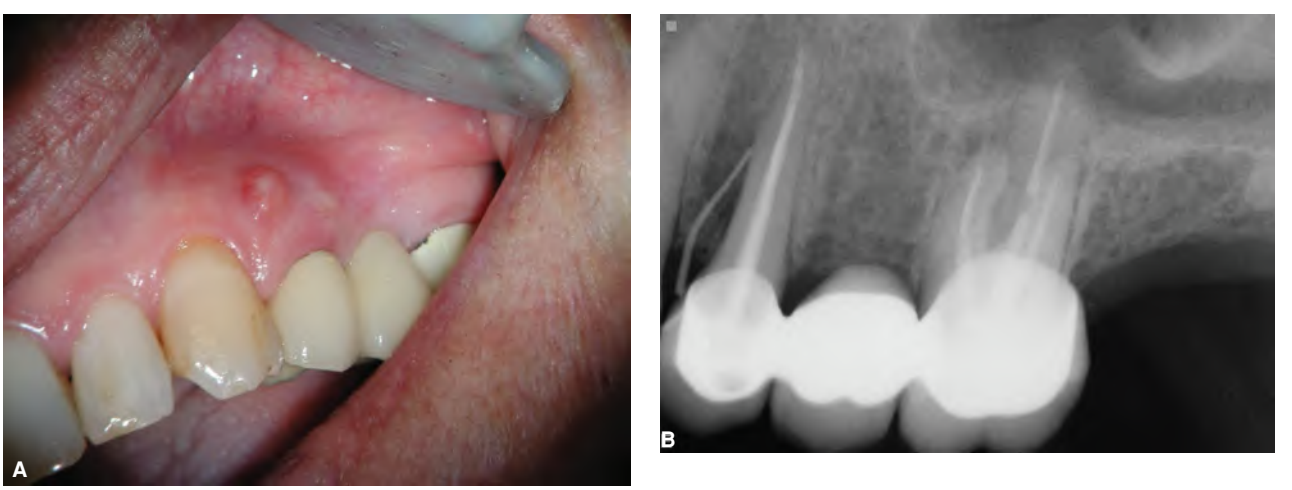

FIGURE 14-3 Typical "pathognomonic combination" of signs and symptoms for VRF diagnosis in a maxillary premolar serving as mesial abutment for a three-unit bridge. ***A***. Highly located sinus tract in the attached gingiva. ***B***. Gutta-percha tracing to the lateral bony radiolucency at the mesial aspect of the maxillary premolar. (Courtesy of Dr. Russell Paul, Zichron Ya'acov, Israel.)

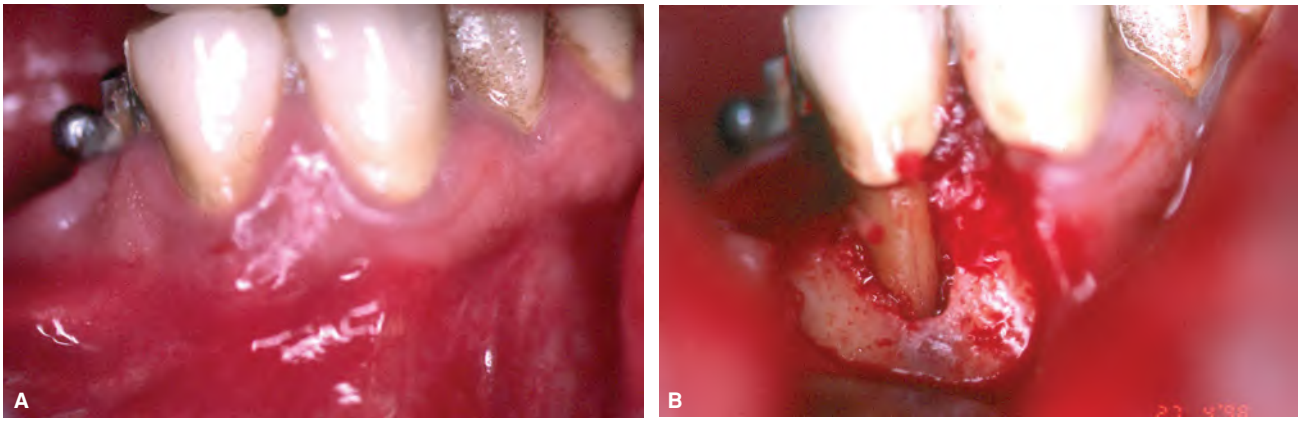

FIGURE 14-4 ***A***. Mandibular premolar suspected of VRF. ***B***. Buccal exploratory flap reflection reveals the nearly complete loss of buccal bone-dehiscence type.

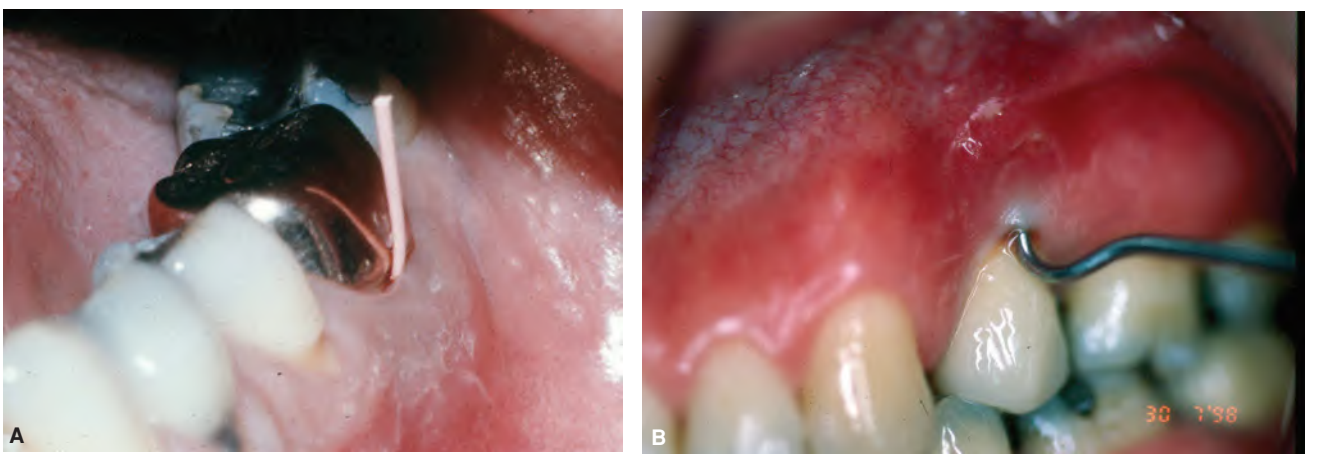

FIGURE 14-5 ***A***. Gutta-percha cone demonstrating deep probing defect on the buccal aspect of mesial root in a mandibular molar. ***B***. Periodontal probe in a buccal probing defect of a maxillary premolar.

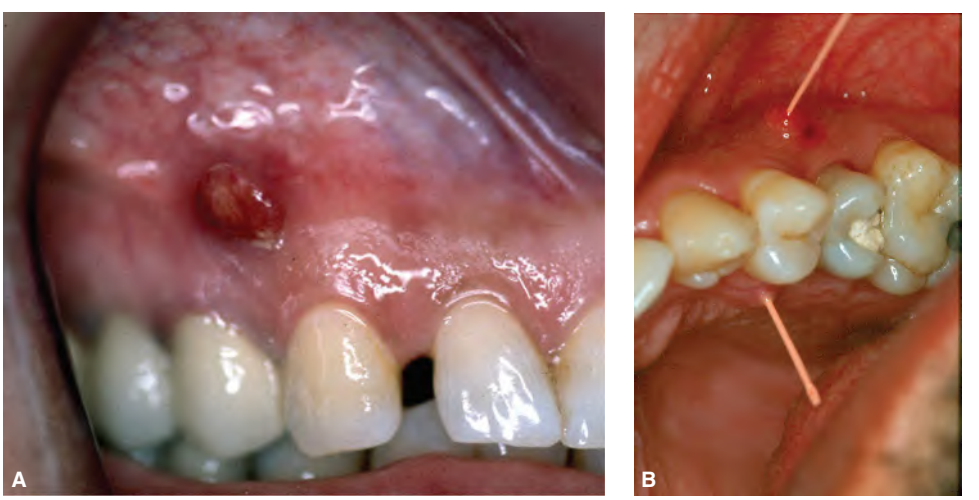

FIGURE 14-6 ***A***. Coronally located sinus tract near a maxillary premolar. ***B***. Buccal and lingual sinus tracts from a complete VRF of a second maxillary premolar. The lingual sinus tract is located mesially to the fractured tooth.

for an immediate and definitive diagnosis and treatment plan[9,19,21] (Figure 14-7 A–D).

Lustig et al.[19] described the typical pattern of bone resorption associated with a tooth having a VRF. In 90% of cases wherein teeth were extracted due to a VRF, buccal bone dehiscence was observed along the entire length of the fractured root. In addition, a direct correlation was found between the amount of bone resorbed and time without treatment (Figure 14-8). A fenestration type of bone loss was found in only 10% of maxillary premolars and mesial roots of mandibular molars (Figure 14-7 C,D). Walton and Rivera et al[28] described this progression of bone loss when the fracture propagates in an apical-to-coronal direction. They observed that when the fracture extends to the gingival margin, bone resorption progresses rapidly in apical and lateral directions, into the interproximal area. However, even if the buccal plate is completely resorbed, with no interproximal involvement, a single periapical radiograph may often not reveal the entire extent of bone loss (Figure 14-9 A,B).

Typically, when bone resorption is present on the lingual aspect of a VRF, it appears as a shallow rounded U-shaped defect. This is due to the cancellous bone in this area being much thicker than on the buccal side; over time, the resorption continues to extend laterally. Surprisingly, only 10% of bony fenestrations, secondary to a VRF, appear as an abscess, similar to a typical abscess of endodontic origin without significant periodontal defects on the buccal aspect.[19]

When a bony dehiscence is present and associated with a visible fracture, as seen during an exploratory surgery,

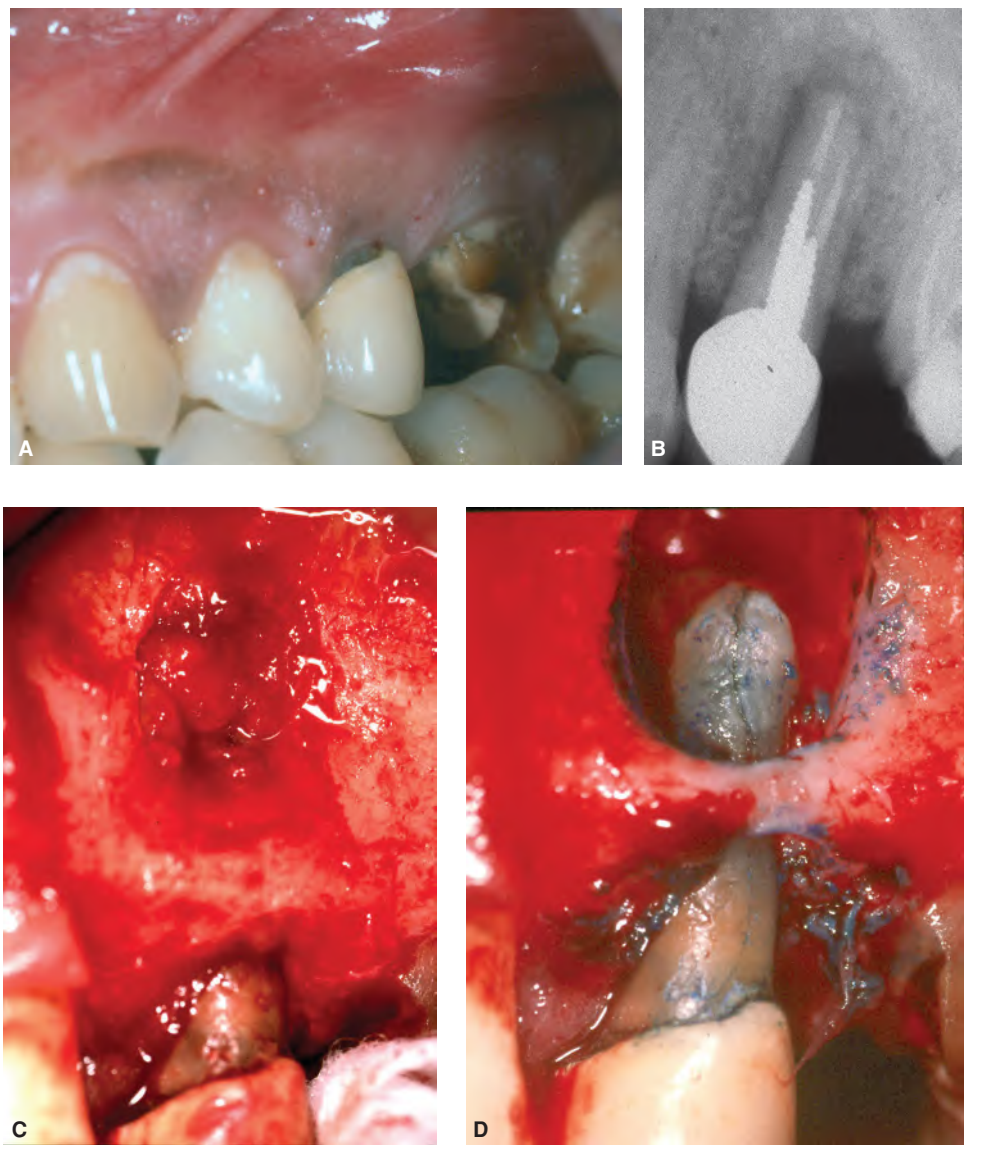

FIGURE 14-7 ***A***. VRF in a maxillary premolar. New restorative treatment was required for the posterior segment. ***B***. Periapical radiograph shows the "halo" radiolucency around the root. ***C***. Exploratory flap procedure reveals buccal fenestration filled with granulation tissue. ***D***. Removal of the soft tissue reveals fracture line from apical to cervical root area. Methylene blue dye helps to demonstrate the fracture.

(Figure 14-7) extraction of the root, or tooth, will prevent further bone loss.[19]

Sinus tracts associated with VRFs, unlike those that result from a typical chronic periapical abscess, are not usually located in the apical area, but rather several millimeters below the gingival margin[7,29] (Figure 14-6). This type of sinus tract was found in 14% to 42% of VRF cases.[7,8,21,27] Therefore, when a deep narrow periodontal probing defect is present with a coronally located sinus tract, and when a bony dehiscence is observed during exploratory surgery, the tooth with a VRF can be better diagnosed in a timely manner.

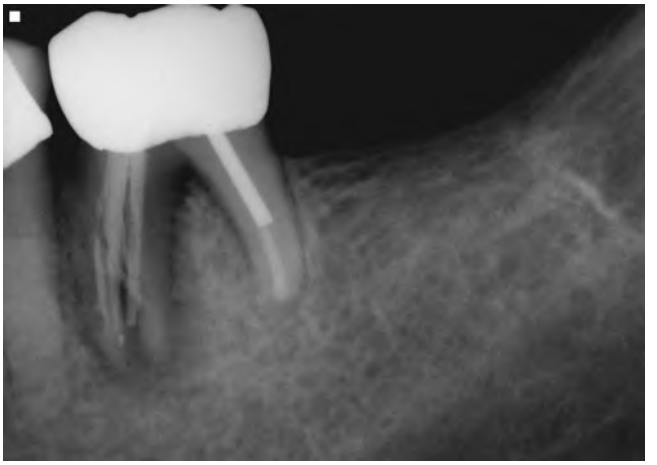

FIGURE 14-8 A late diagnosis of a VRF showing separated root segments of a mesial root of a mandibular molar. Large bony lesion can be seen surrounding the tooth from all aspects.

Radiographic Findings

Occasionally, direct radiographic detection of a VRF is possible. If there is separation of the root segments (Figure 14-10), especially when accompanied by a large radiolucent area between the roots, radiographic visualization is more likely. Note that the inflammatory tissue between the root segments may actually aid in facilitating the separation of the segments over time.[29,30] Also, sometimes a "hairline" fracture can create a radiolucency that can occasionally be detected on a periapical radiograph[9] (Figure 14-11). However, in clinical practice, radiographic detection of fractures does not seem to occur that often.[9,31] Hairline fractures are quite difficult to detect radiographically. When a root fracture is suspected, it is recommended that at least two periapical radiographs be taken, from different angulations (Figure 14-9).

The appearance of a radiolucency associated with a VRF is influenced by the extent of bone destruction, the direction and location of the fracture, and the time elapsed since the fracture developed.[29] To help distinguish between a radiolucency associated from a VRF and bone loss associated with periodontal disease, or nonhealing root canal treatment, certain characteristics of bone loss were suggested.[4,20,23,22,32] The most frequent radiographic presentation observed with VRFs, especially with maxillary premolars and the mesial roots of mandibular molars (that are among the

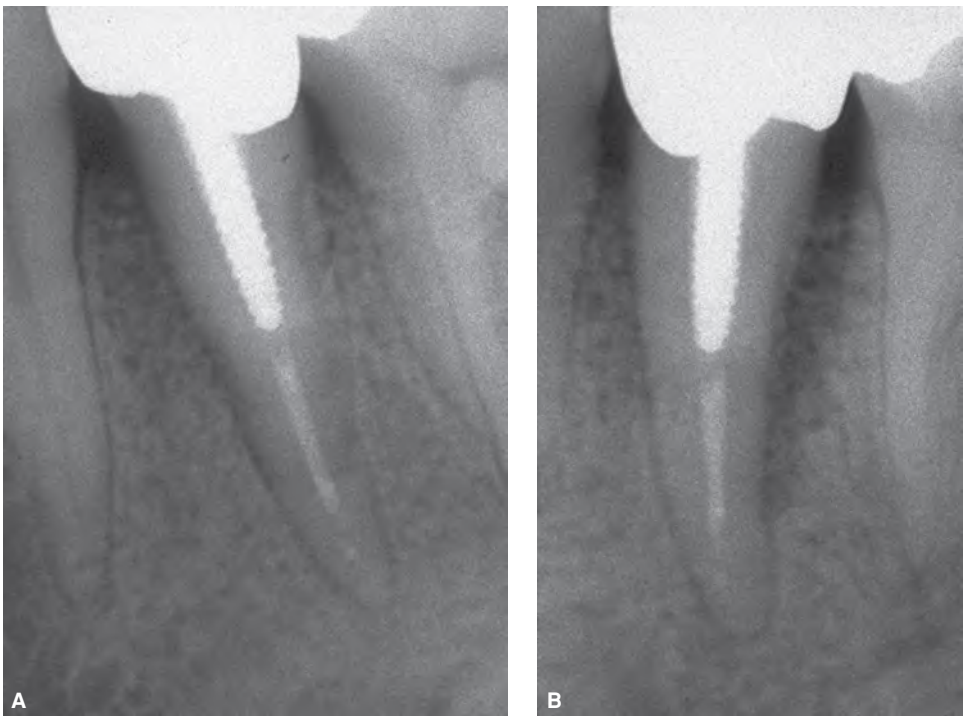

FIGURE 14-9 ***A***. Periapical radiograph of a mandibular premolar reveals widening of the periodontal ligament space on the mesial tooth aspect. ***B***. Change of horizontal angulation reveals radiolucency along most of the distal part of the root from the coronal aspect, not involving the periapical area (periodontal radiolucency).

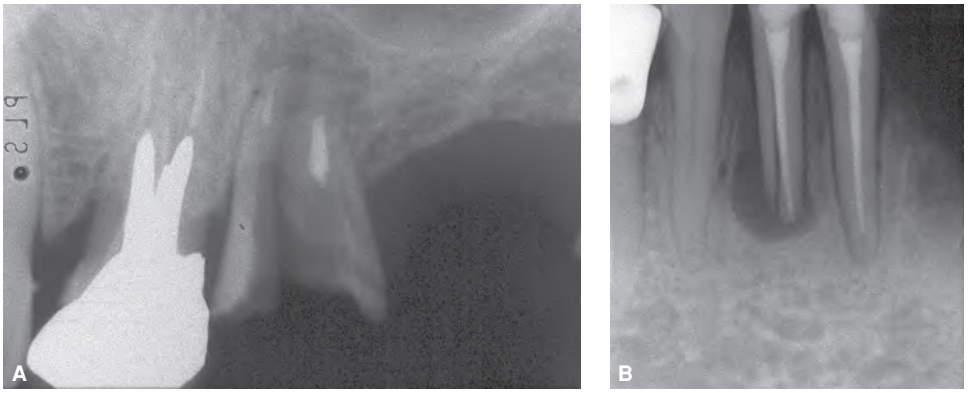

FIGURE 14-10 Separation of root segments. ***A***. In a maxillary premolar. ***B***. In a mandibular incisor.

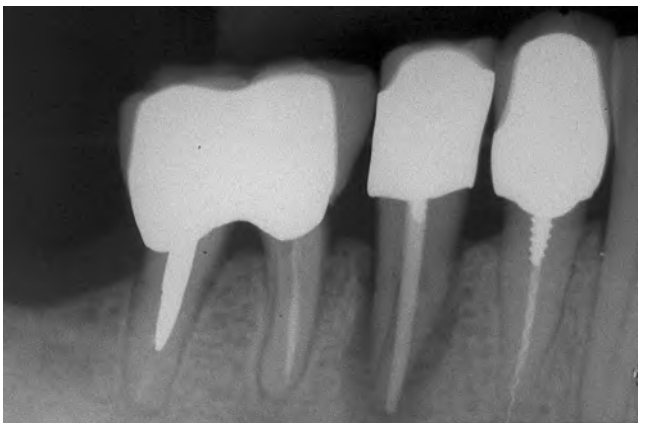

FIGURE 14-11 A "hairlike" radiolucent line can be seen at the distal aspect of a fractured mandibular incisor.

most susceptible teeth and roots to fracture), is a "halo" or "J-shaped" appearance of bone loss. These bony defects are a combined periapical and lateral root radiolucency, present on one or both sides of the involved root[1,22,23] (Figure 14-12 A–C). Also, in 63.3% of VRF cases involving the mesial roots of mandibular molars, the "halo" pattern of bone loss involves the furcation area between the mesial and distal roots[22] (Figure 14-13 A–D). Although this pattern of bone loss is not necessarily pathognomonic for a VRF, it is highly suggestive, especially when associated with other signs and symptoms as previously described.

In recent years, CBCT) has become popular due to its ability to accurately diagnose VRFs, especially as compared to periapical radiography.

Several publications in recent years claim that the CBCT is superior to periapical radiographs in the sensitivity, specificity, and accuracy, over periapical radiographs in detecting VRFs in endodontically treated teeth.[33–35] However, this has been disputed by a recent systematic review and meta-analysis study,[36] as well as two other clinical investigations.[37,38]

The primary challenge in diagnosing VRFs from CT scans is the difficulty observing incipient fractures. Additional issues include the presence of streak (radiopaque) artifacts secondary to the presence of gutta-percha or metal dowels, presence of beam hardening artifacts, increase in radiation dosage, and cost (Figure 14-14 A–F). According to the American Dental Association (ADA) statement,[39] CBCT should be used only when the clinician expects the diagnosis will yield a benefit to the patient. In a publication from the AAE,[40] it is stated that "Fine vertical cracks appear not to be revealed unless the radiolucency width is higher than 0.15 mm. What may be observed is the Specific patter of bone loss in one or more CBCT

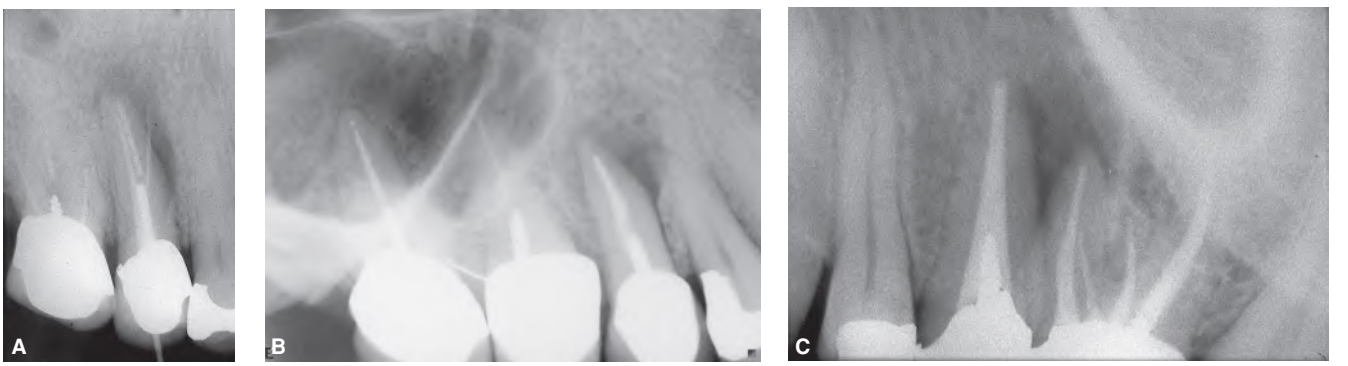

FIGURE 14-12 ***A***. "Halo" appearance radiolucency around the apical and mesial aspects of the root of a maxillary premolar. ***B***. "Halo" type radiolucency including the mesial and distal aspects of the apical third of the root. ***C***. Isolated lateral radiolucency in a maxillary premolar. The apical and coronal areas are not involved.

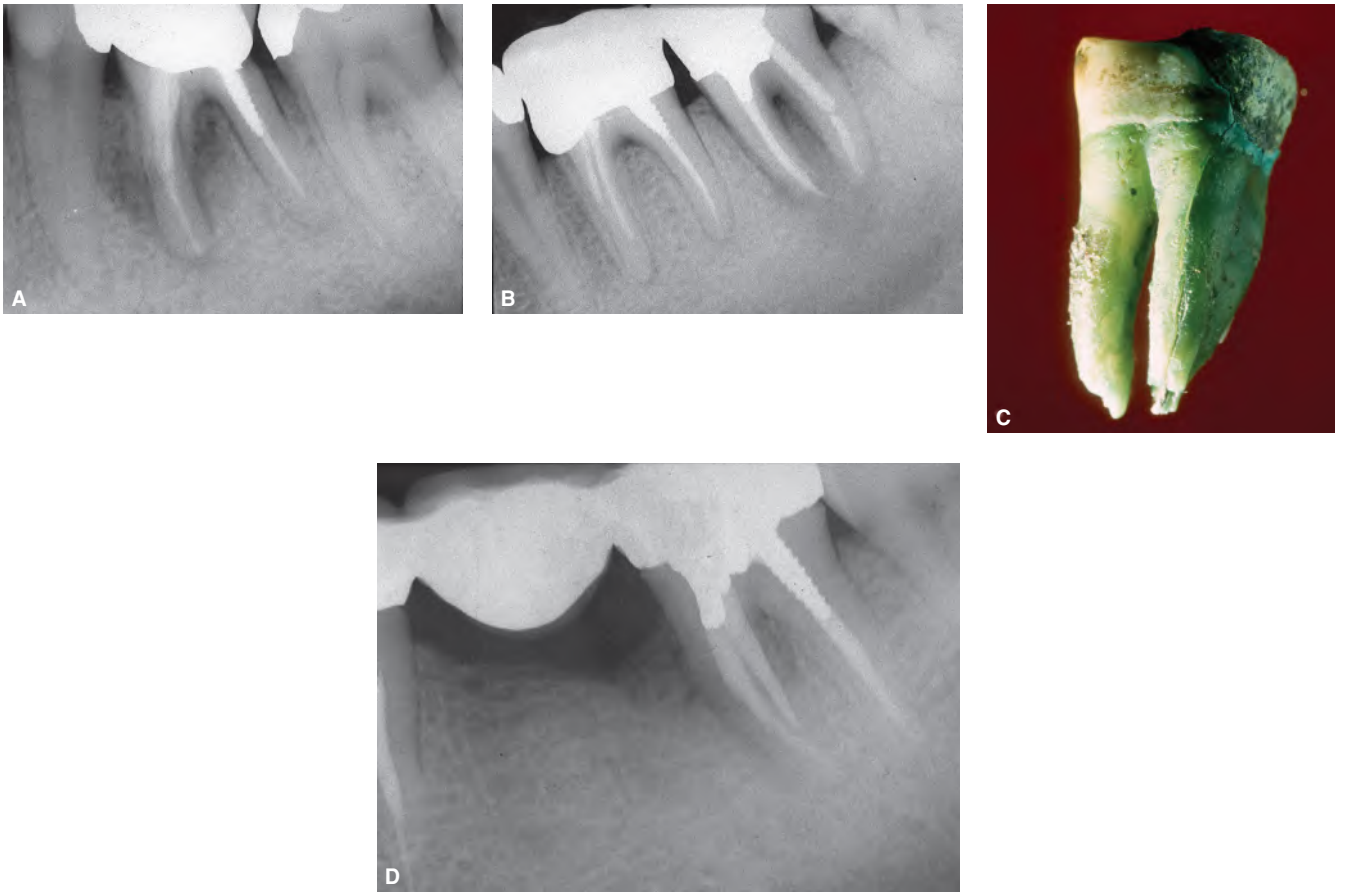

FIGURE 14-13 Typical bony periradicular radiolucencies around mesial roots of mandibular molars. ***A***. Lateral radiolucency on the mesial aspect of the root and at the bifurcation area of a mandibular first molar. ***B***. Periapical and bifurcation radiolucency around a second mandibular molar. ***C***. Extracted tooth (B) shows the fracture line at the buccal aspect of the mesial root. ***D***. Angular and bifurcation radiolucency.

scans." However, future generations of CBCT machines may yield better detection capabilities. For more details, see Chapter 9.

Unless an actual fracture of the root is seen on the radiograph, the pattern of bone loss can only be considered as highly suggestive of a VRF. It should be noted that the pattern of bone loss associated with VRFs is also similar to those seen in primary endodontic or periodontal lesions.[7,9,22,23,32] Therefore, with radiographic evidence alone, a final diagnosis cannot and should not be made. Unfortunately, with the lack of other clinical findings, there is typically a delay in making a diagnosis, resulting in subsequent increase of bone loss surrounding the tooth. When VRF is suspected, the clinician must inform the patient of a potentially reduced prognosis prior to considering any endodontic treatment.

ETIOLOGY

Although VRFs are typically associated more with endodontically treated teeth, they can, on rare occasions, also occur in nonendodontically treated molars. As stated before, this finding seems to be more prevalent in Chinese population[3]; the reason is not well understood.

The causes of VRFs in endodontically treated teeth are multifactorial. They can be divided into predisposing factors and contributing factors.[13,41]

Predisposing Factors

Changes in the quality and mechanical properties of root dentin of an endodontically treated tooth may predispose the root to fracture. In addition, the specific anatomy of a susceptible tooth may also be involved.[13,41] The amount of remaining tooth structure, as a result of caries and trauma, can also be a factor that increases the risk of future crack formation in dentin.[26]

The roots most susceptible to fracture are those in which the mesio-distal dimension is narrower than the bucco-lingual dimension (i.e., the root cross section is oval, triangular, kidney-shaped, ribbon-shaped or hourglass-shaped).[42] Examples of such teeth are maxillary and mandibular premolars, mesial

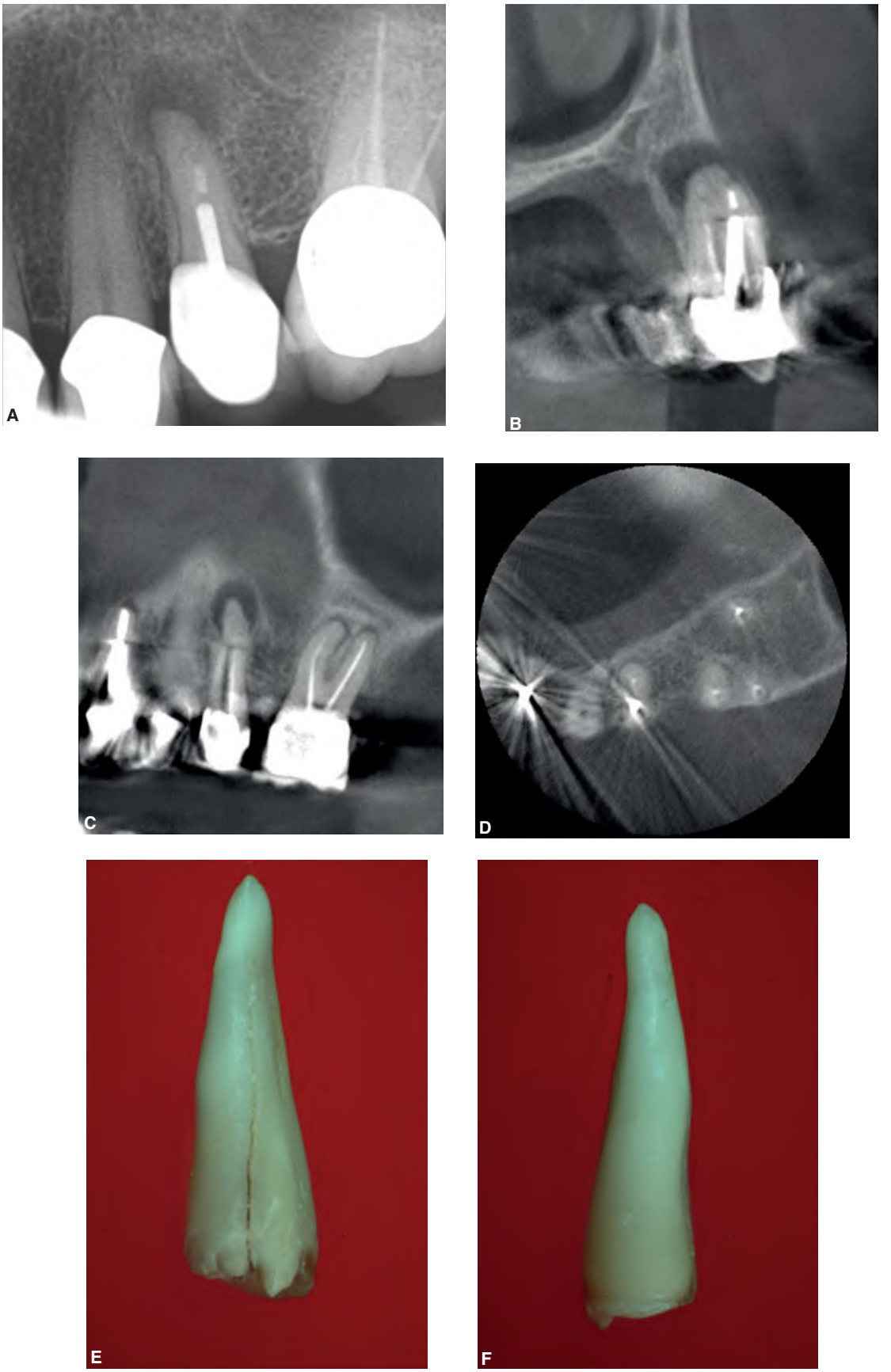

FIGURE 14-14 Restored, endodontically treated maxillary premolar. "Halo" radiolucency can be seen around the tip of the root (***A***). Despite deep probing defect in the midbuccal area, no evidence of fracture can be seen in CBCT coronal (***B***), sagittal (***C***) or in axial (***D***) views. After the tooth was extracted, the VRF can be seen clearly in the buccal aspect of the root (***E***) but not in the lingual one (***F***). (Courtesy of Dr. Ron Ganik, New York, NY.)

roots of mandibular molars, mandibular incisors, and mesiobuccal roots of maxillary molars.[43] In a retrospective study of fractured roots, Tamse et al.[7] found these root shapes to be the most frequently fractured (79%). To minimize the risk of VRFs and to increase the success rates of endodontic and restorative treatments, familiarity with root anatomy and morphology is essential.[42] For more details, see Chapter 1.

Root curvatures and depressions, like those present in the mesial root of mandibular molars and the buccal root of a bifurcated maxillary premolar, are anatomic entities that can predispose a tooth to fractures (also root perforations)[42,44,45] (Figure 14-15).

Canal and root shape, as well as dentin thickness, can affect the tensile stress distribution during intra-canal procedures.[46] Recognition of canal shape is paramount since the area of reduced curvature radius (the buccal and lingual areas) is strongly influenced by stress concentrations.[47] This is probably the reason why these roots have a predilection to fracture in a bucco-lingual direction as opposed to mesio-distally, even though more tooth structure is removed from the mesial and distal aspects during canal instrumentation and post space preparation.[48]

Another important predisposing factor for VRFs is the amount of pretreatment tooth structure missing; mainly due to caries or trauma. Coupled with reduced amount of radicular dentin resulting from various intra-canal procedures (i.e., initial root canal therapy, endodontic retreatment,[21] and post space preparation), sound tooth structure is directly correlated with the ability of the endodontically treated tooth to resist fractures[42,48,49] (Figure 14-16 AB).

Reduced moisture content of dentin has been reported in endodontically treated teeth.[50] The dehydration of dentin can induce cracks in the root canal wall, which can subsequently develop into a fracture. Dentin "age" is also considered a predisposing factor for tooth fracture in endodontically treated teeth.[51,52] Crack extension in older dentin has been shown to propagate at a faster rate than in younger dentin.[53] Also, aged dentin has an increased amount of translucent dentin, making it more susceptible to fracture.[54]

Small cracks are often present naturally in the dentin of intact teeth, appearing both parallel and/or perpendicular to the root canal space.[55] During intra-canal procedures when dentin is removed, especially in the mesio-distal areas, these cracks may be exposed and progress over time in a buccal and/or lingual direction, potentially creating incomplete or complete fractures.[56]

There are several factors that may directly or indirectly contribute to the formation of a VRF. These include endodontic access preparation, root canal preparation, various solutions and medicaments used during canal preparation, root canal obturation, post space preparation and placement, and appropriateness of the coronal restoration.[20,41,57,58]

Excessive removal of dentin during access preparation and enlargement of the coronal third of the root may result in unintentional thin dentin walls and a weakened root, predisposing it to a future fracture[41] (Figure 14-16 AB).

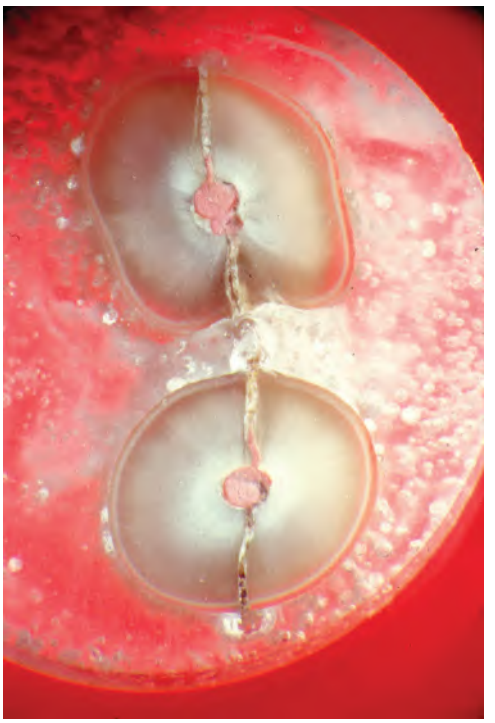

FIGURE 14-15 Complete VRF in buccal and lingual roots of a maxillary bifurcated premolar. Note the concavity on the bifurcation aspect of the buccal root.

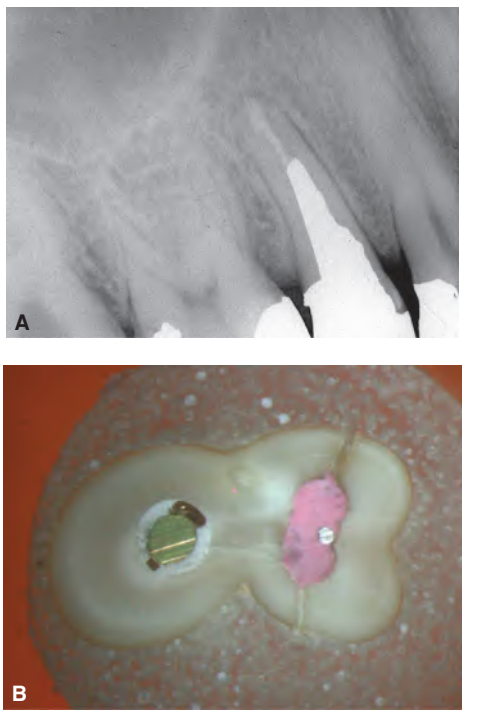

FIGURE 14-16 Thin dentin walls following excessive preparation for a post in a maxillary premolar.

Studies suggest a correlation between the type of canal preparation and susceptibility to fracture.[49,59] Therefore, the design of the NiTi instruments is an important factor when evaluating the ability of roots to resist fractures. Roots may be significantly weakened when instruments with larger tapers are used. In recent years, in vitro studies have shown that rotary NiTi endodontic instruments may create micro cracks in the dentin.[60,61,62] These small cracks produced in the canal wall, or at the root surface, coupled with preexisting cracks in the dentin (even in untreated teeth),[56,63] may be precursors to the development of incomplete and complete fractures at a later time. Dentinal cracks have also been associated with root-end preparation using ultrasonic root-end tips.[64] It is possible, these cracks could be an important cause for some failures after surgical endodontic treatment. When evaluating the stress–strain response of human dentin, Kishen,[41] found that dentinal cracks that originate from the inner walls of the root canal and placed under load, tend to progress to the outer surfaces in the buccal and lingual directions.

There are also some recent in vitro studies suggesting that excessive use of various root canal irrigation solutions and medicaments, such as sodium hypochlorite, chelating agents and calcium hydroxide, may change the quality of dentin, possibly causing an increase in dentinal wall erosion that makes the dentin more prone to cracks and fractures.[65–67]

During lateral compaction of gutta-percha, stress is generated through the dentin walls. This is due to the wedging effect of the spreader, either directly on the canal walls or indirectly through lateral compaction of the gutta-percha.[57] The design of the spreader and the metal used may also be a contributing factor to the initiation and propagation of a VRF.[68] Since the introduction of NiTi instruments for canal preparation, specially designed NiTi spreaders have been designed that are more elastic than earlier stainless steel spreaders, exerting less strain on the canal walls during lateral compaction.[69] On the other hand, any decrease in the initiation or exacerbation of a fracture is not known. Chai and Tamse[47] designed a model to analyze the fracture mechanics that occurs during the compaction of gutta-percha. After creating a dentinal crack in the root canal wall of oval and round root canals, it was shown that less load was required to propagate the preexisting crack to the outer surface of oval canals compared to round ones, confirming previous studies.[70,71]

Sathorn et al.[70,71] showed a significant correlation between root fractures and root canal anatomy related to other factors, such as stress distribution, residual dentin thickness, and types of canal preparation. Canal diameter, shape, and proximal concavity all have a significant influence on fracture susceptibility. Surprisingly, residual dentin thickness in itself does not appear to increase the susceptibility for fracture.

Restorative considerations after root canal therapy, such as post space preparation, post type and shape, hydrostatic pressure from traumatic cementation of a post, and the expansion of a post due to corrosion, are all possible additional iatrogenic factors contributing to the initiation of a VRF in endodontically treated teeth.[72] Post space preparation and post selection are of utmost importance, especially in teeth susceptible to VRFs because of specific cross-sectional contours and curvatures associated with certain roots.[42]

Recently, the use of fiber posts has been recommended because their modulus of elasticity is similar to that of dentin, allowing them to flex with the root once under stress.[73] It has been shown[74] that placement of fiber posts improved the survival rate of endodontically treated premolars. Other studies have shown a preference of fiber posts depending on the type of coronal coverage.[75,76] Fiber reinforced resin posts have also been shown to be superior to other types of posts.[77–79] Ferrari et al.[79] suggested to preserve at least one coronal wall during restoration of an endodontically treated tooth as a significant preventive measure in reducing fractures in maxillary premolars.

Endodontically treated teeth should have suitable coronal coverage after completion of treatment[13] (Figures 14-17 A,B). Proper type and design of coronal coverage with adequate ferrule may be more important than the post characteristics.[80]

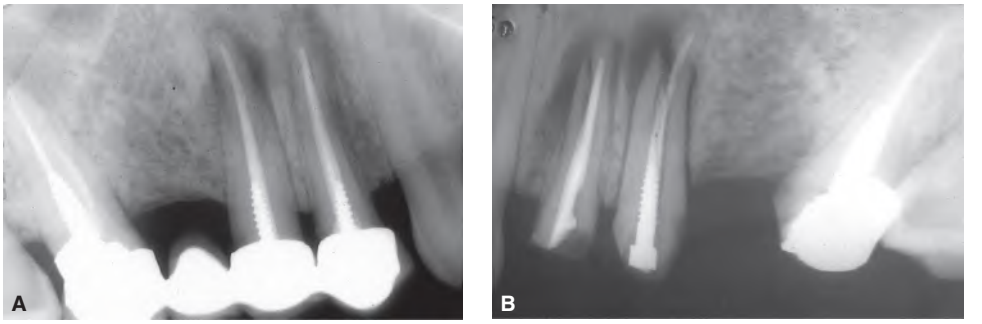

FIGURE 14-17 Endodontically treated maxillary premolars serving as abutments. ***A***. Typical periradicular radiolucencies around the two premolars. ***B***. Six months after diagnosis of VRF. Separation of root segments can be seen in both teeth.

CLINICAL MANAGEMENT

In the presence of a VRF, there is a constant ingress of bacteria from the oral cavity into the subjacent periodontal tissues and alveolar bone. Therefore, in order to prevent further breakdown of these supporting structures, extraction (or the removal of the affected root(s)) is the only option. If selective root amputation is planned, a new coronal restoration is often advised. Occasionally, the partial removal of a root can be accomplished if the VRF is limited to the apical part of the root[81,82] (Figures 14-18 and 14-19).

PREVENTION

Prevention of VRFs in endodontically treated teeth is essential; however, because of the multifactorial etiologies, this can be a difficult task. The following can reduce potential risks of VRFs:

1. Recognize the roots and teeth most susceptible to fracture.
2. During endodontic and restorative procedures preserve as much tooth structure as reasonably possible. It

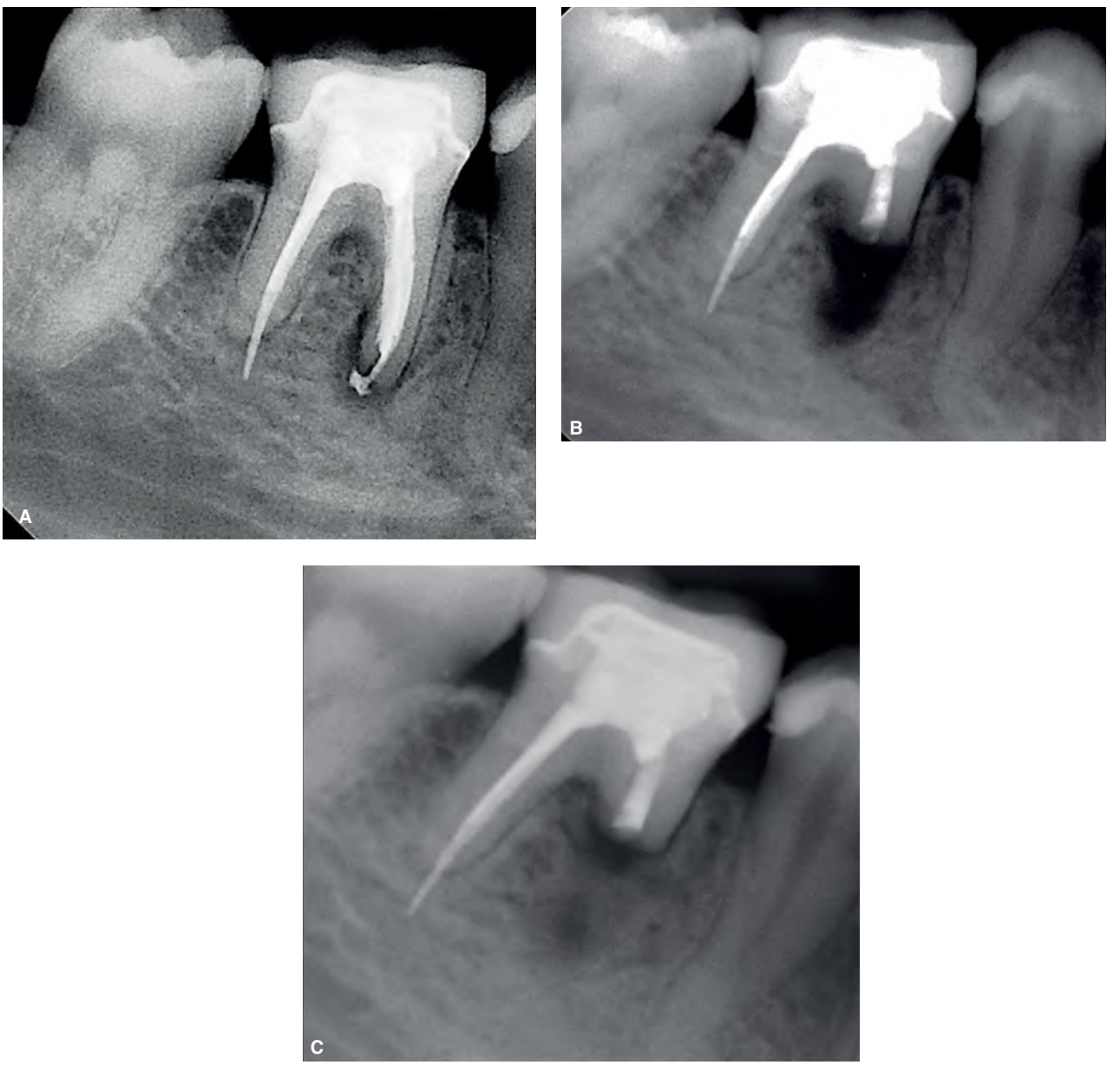

FIGURE 14-18 ***A***. Poor obturation in apical third of the mesial root in an endodontically treated mandibular molar. ***B***. During the surgery, a root fracture was noted in this region. The root was shaved until elimination of the fracture, leaving part of the root in the bone. ***C***. Twelve-month postoperative radiograph showing evidence of periapical healing. (Courtesy of Dr. Mario E. Abdennour, Boston, MA.)

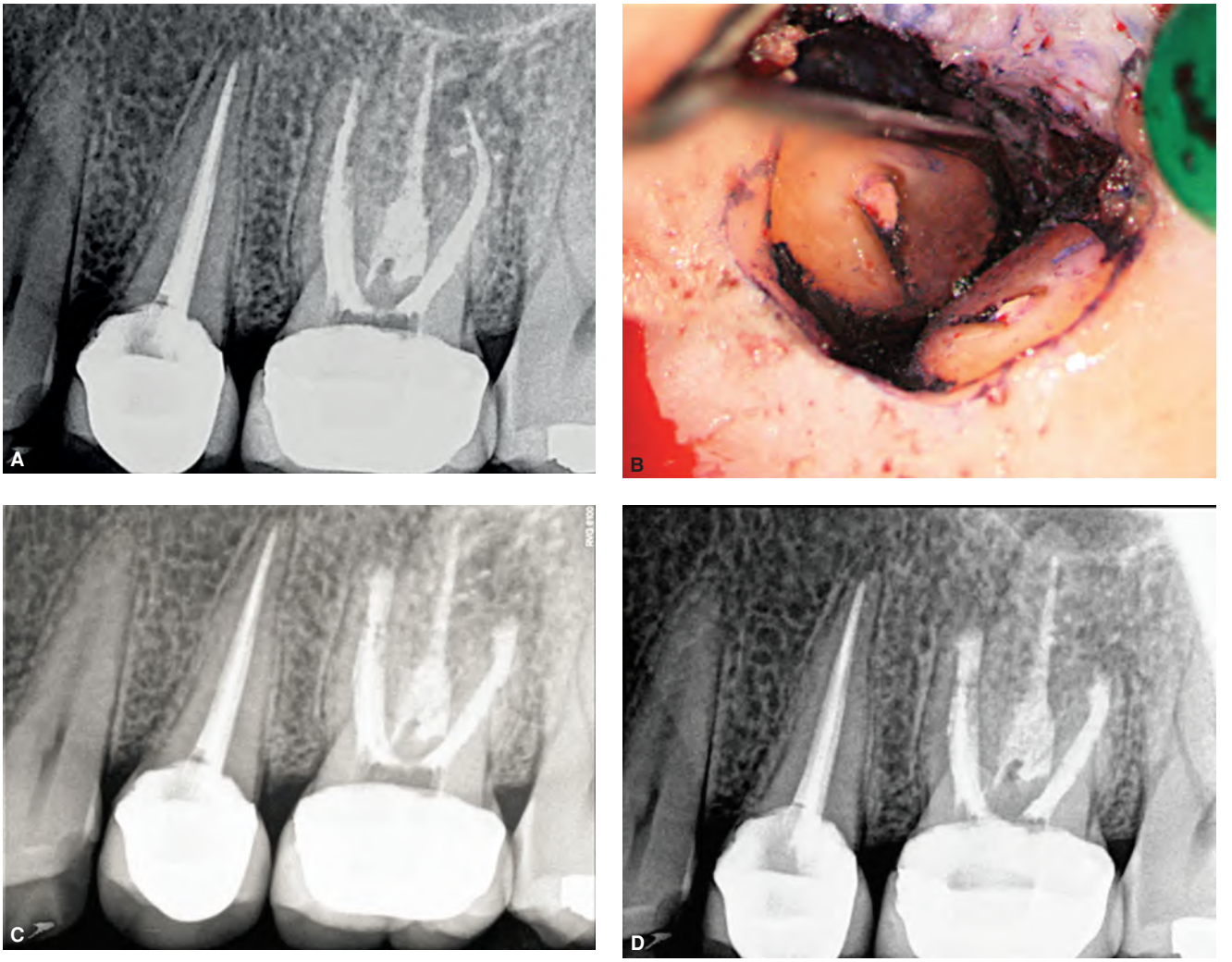

FIGURE 14-19 ***A***. Periapical radiolucency around distobuccal root of a restored endodontically treated maxillary molar. ***B***. During endodontic surgery, an incomplete fracture was seen. The two roots were root-end sealed with MTA. ***C***. Immediate postoperative radiograph showing the two root-end fillings. ***D***. Six-month postoperative radiograph showing evidence of progressive periapical healing around the DB root. (Courtesy of Dr. Spyros G. Floratos, Athens, Greece.)

is recognized that there may be a challenging balance between the need to achieve a clean root canal system and removing only the amount of dentin necessary for the specific intra-canal procedure.

3. Compaction forces during canal obturation should be minimized. Future developments of root canal obturating methods and materials should focus on preventing excessive stresses on root dentin.
4. Posts should only be used when necessary for additional core support.
5. Metal posts should have parallel walls with passive fit and round edges.
6. Fiber reinforced resin based composite posts should be used when indicated.
7. Full coverage coronal restoration should have a ferrule margin with a vertical length of 1.5 to 2 mm on sound dentin.

SUMMARY

1. VRFs originate from the root and may progress coronally or apically. They differ from fractures in nonendodontically treated teeth tending to originate in the crown and remain either in the crown or progress apically.
2. When a combination of highly located sinus tract and a narrow deep periodontal defect is present, it is highly suggestive of a VRF.
3. There is no correlation between the location and the extent of a root fracture and their signs, symptoms, and radiographic features.
4. Evidence-based studies on the clinical and radiographic features of VRFs are lacking and difficult to perform.
5. VRFs are usually diagnosed after all endodontic and restorative procedures have been completed, and often not recognized until years later.

6. Most VRFs occur in cases where the endodontic and restorative procedures seem to have been done correctly.
7. When the diagnosis is made of a VRF, extraction should be done in a timely manner in order to minimize complications like periradicular bone loss. This bone loss may compromise subsequent implant placement. It is recognized that patient compliance can often be an issue, especially if the patient is asymptomatic.
8. Prevention of VRF is often difficult due to the multifactorial nature of the causes.

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Chapter 15

Pathologic Tooth Resorption

Jens O. Andreasen, Geoffrey S. Heithersay, Leif K. Bakland

Resorption of permanent teeth has been considered an unfortunate and unpredictable phenomenon. In the past 30 years, new information about the nature of osteoclasts, and more specifically how they behave in the oral environment, has provided valuable insight into the previously mysterious osteoclastic attacks on the roots of teeth.[1] The sequence of traumatic and pathologic events that may lead to root resorption will be described; however, it may be of interest first to consider why permanent teeth normally survive without succumbing to root resorption.

THE HOMEOSTASIS PHENOMENON OF PULP AND PERIODONTAL LIGAMENT PREVENTING OSTEOCLASTIC ATTACKS

It is a surprising fact that a permanent tooth throughout life survives in an environment surrounded by alveolar bone with very active osteoblasts and osteoclasts, without being approached by any of these two cell lines, under normal conditions (Figure 15-1).[2] Several studies indicate that this immunity possibly relates to the presence of intact cementoblast and odontoblast cell layers, and perhaps also to adjacent cell layers.[2] In various animal experiments, it has been shown that physical and chemical insults to these cell layers can result in loss of protection, leading to active root resorption, both on the external root surface and in the root canal.[3,4]

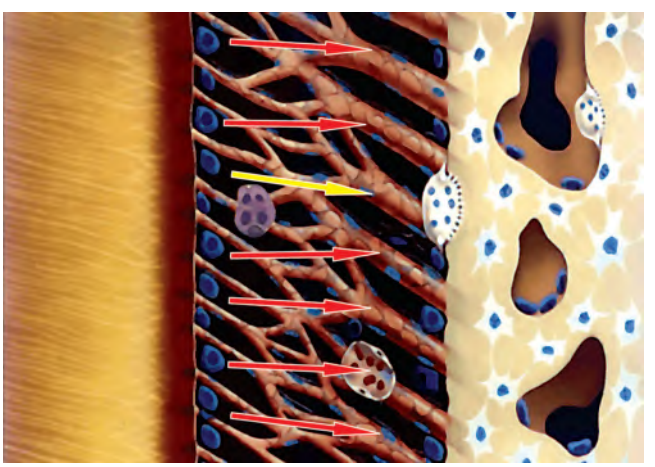

FIGURE 15-1 Antisorptive and antiosteogenesis signals released from cementoblasts, periodontal ligament cells, and Mallassez epithelial islands creating a periodontal ligament space homeostasis. (From Lindskog et al. 2007[1]; used with permission.)

To understand the full scope of tooth resorption it is necessary to understand the nature of osteoclasts, a cell with multipotential behavior that plays at least five significant roles during adolescence and adult life.

PHYSIOLOGY OF OSTEOCLASTS

Osteoclasts represent a syncytium of stimulated macrophage progenitor cells. This stimulation is under control of the Rank-Rankl/osteoprotegerin (OPG) system.[1] It serves as an on-off system for osteoclastic activity, where activation of the OPG system, and down-regulating of the RANKL system, may favor differentiation of new osteoclasts. Osteoclastic activity of the opposite OPG system may slow down or arrest the resorption (Figures 15-2 and 15-3).

MECHANISM OF HARD TISSUE DECONSTRUCTION

After an osteoclast has been formed, its role is to resorb hard tissue (i.e., cementum, dentin, enamel, or bone). The mechanism of hard tissue deconstruction is dissolution of inorganic materiel by acids associated with a breakdown of the organic structures (Figure 15-4).[1]

THE ROLE OF OSTEOCLASTS IN TOOTH ERUPTION

The osteoclast plays a key role in the shedding of primary teeth (**physiologic resorption**). At a certain stage in the development of a permanent successor and the eruption process, the coronal part of the follicle induces a rim of osteoclasts that, over a couple of years, resorb the roots of the primary predecessor and the adjacent bone.[5–7] In the case of ectopic position of a permanent tooth germ, **nonphysiologic root resorption** may affect adjacent permanent teeth.

Endodontic Implications

As to the physiologic resorption of primary teeth, it should be noted that chronic periapical inflammation from pulp necrosis in primary teeth accelerates shedding.[6,7] For example, with the ectopic eruption of a canine, resorption of the permanent lateral incisor may take place (Figure 15-5). If the lateral incisor has an intact pulp and if the ectopic eruption path changes to a normal path, or if the canine is removed,

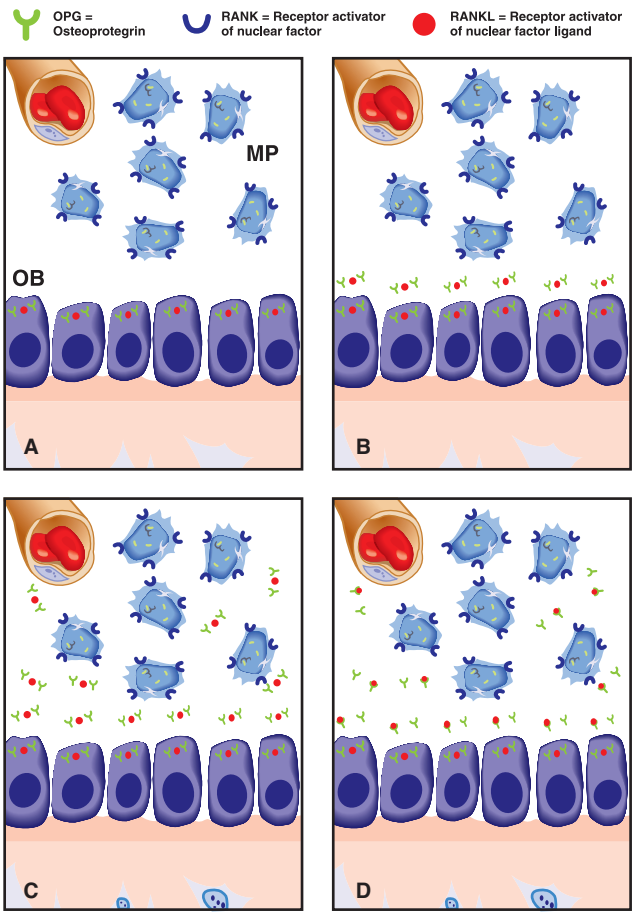

FIGURE 15-2 ***A & B.*** Activation of the OPG/RANK/RANKL system. Osteoclastogenesis is the result of differentiation of mononuclear/macrophage progenitor cells and fusion of these cells to become osteoclasts. The commitment of these cells to become osteoclasts depends on the activation of the RANK receptors on the surface of RANKL, that is produced by stroma cells and osteoblast. ***C.*** RANKL is liberated into the tissue and attaches to the receptors of the mononuclear/macrophage progenitor cells. ***D.*** Mononuclear/macrophage cells aggregate, fuse, and form osteoclasts. Proresorptive factors are hormones and cytokines such as PTH, $1,25(OH)_2D_3$, IL-1, IL-6, TNF, LIF and corticosteroids, that activate osteoblasts and stroma cells to produce RANKL and depress OPG. (From Lindskog et al. 2007[1]; used with permission.)

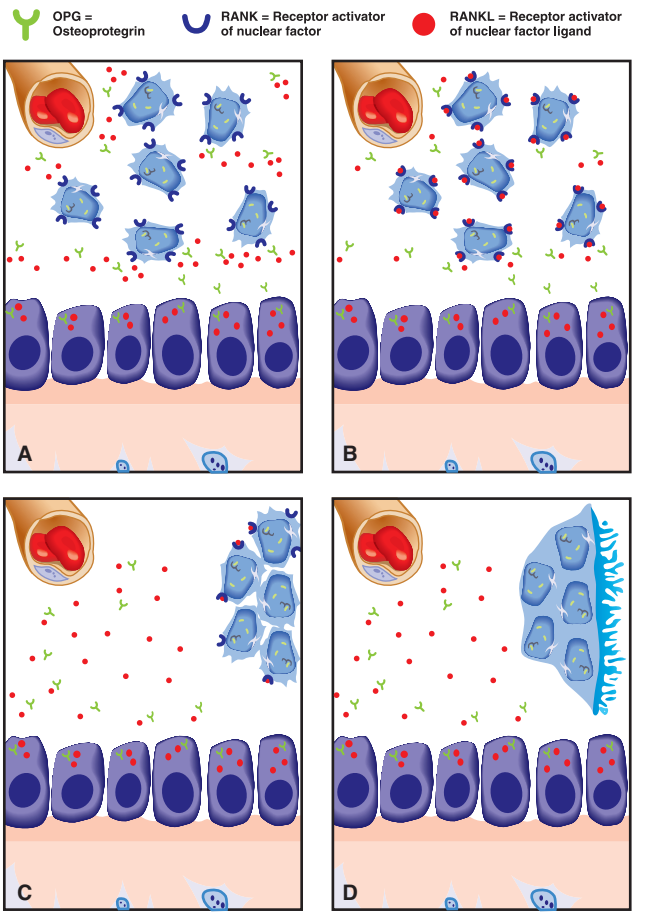

FIGURE 15-3 ***A–C.*** Inactivation of the OPG/RANK/RANKL system. Antiresorptive factors such as estrogens, calcitonin, BMP, TGF-β, IL-17, PDGF and calcium depress RANKL production by osteoblasts and stroma cells, and activate their OPG production. ***D.*** OPG binds and neutralizes RANKL, leading to a block in osteoclastogenesis and decreased survival of osteoclast. (From Lindskog et al. 2007[1]; used with permission.)

the resorption process of the lateral incisor will be arrested and the pulp can survive. Consequently, root canal treatment of such teeth is not necessary.

THE ROLE OF OSTEOCLASTS IN ALVEOLAR BONE GROWTH AND MAINTENANCE

The alveolar bone structure is entirely dependent on a controlled symbiosis between osteoblastic and osteoclastic activity, whereby a continuous turnover of bone takes place throughout life. Table 15-1 shows the stimulatory and inhibitory factors that are associated with osteoclastic activity. If there is a lack of equilibrium in the activities of these two cell types, either osteoporosis or osteosclerosis will occur.[1,6]

Endodontic Implications

In case of ankylosis-related (replacement) resorption of a tooth (external root surface or internal root canal), the tooth becomes a part of the general bone remodelling process and continuous replacement of the root with bone takes place. Internal (root canal) ankylosis-related resorption, leading to infraocclusion, can be arrested by pulp extirpation and root canal filling. In case of external (root surface) ankylosis-related resorption, the condition can, as a rule, not be successfully treated today, at least if the ankylosis has reached a nonreversible stage. Endodontic interventions and placement of calcium hydroxide may slow, but not arrest, the ankylosis process. Intervention also carries the risk of accelerating the process.[8]

OSTEOCLASTS AS MEMBERS OF THE REPAIR TEAM AFTER INJURY

In revascularization situations in bone and teeth, the osteoclast and ingrowing vessels work jointly together as part of the wound healing module; the osteoclast having the role

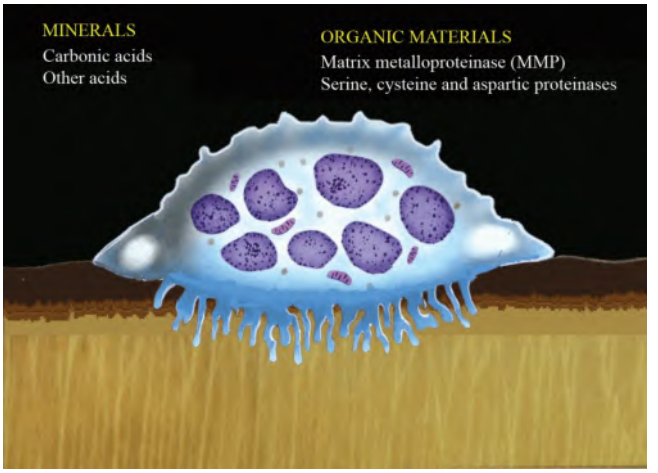

FIGURE 15-4 Physiology of an osteoclast: Removal of inorganic material, such as minerals, by acids (proton pump) and organic material by matrix metalloproteinases, such as MMPs, serine, cysteine, and aspartic proteases.

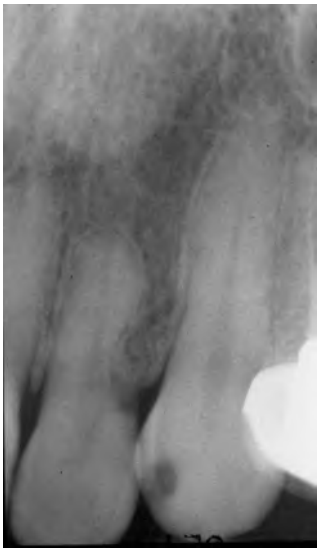

FIGURE 15-5 An example of root resorption on a maxillary lateral incisor from the ectopic eruption of the adjacent canine.

of creating space for the new vascular tissue to grow into narrow spaces (and the root canal).[2] This is later reversed by apposition of new hard tissue (cementum or bone). This phenomenon has been **termed transient apical breakdown, transient marginal breakdown, transient ankylosis, and transient internal surface resorption** according to the location of the process[2,9] (Figure 15-6). These events are frequently seen after luxation injuries and root fractures with coronal displacement, where avascular, but sterile, pulp tissue is replaced in conjunction with the revascularization process.

Endodontic Implications

It is very important to consider that the radiographic signs of osteoclastic activity are not always signs of ongoing infection, but may represent an actual healing process and therefore only requires monitoring.

TABLE 15-1 Stimulatory and Inhibitory Factors Associated with Osteoclast Activity

Factors	Stimulatory	Inhibitory
Hormones		
Androgens		+
Calcitonin		+
Calcitonin gene-related peptide		+
Glucocorticoids	+	
Estrogen	+	
Parathyroid hormone	+	
PTHrP	+	
Thyroid hormone	+	
$1,25(OH)_2$vitamin D3	+	
Cytokines and growth factors		
Bone morphogenic proteins, BMP	+	
Colony stimulating factors, CSF-1	+	
Endothelln-1	+	
Epidermal growth factor, EGF	+	
Fibroblast growth factor, FGF	+	+
Granulocyte macrophage colony stimulating factor, GM-CSF	+	
Insulin-like growth factor, IGF-1	+	
Interferon		+
Interleukin	+	
Kinins	+	
Macrophage inflammatory protein 1-α, MIP-1α	+	
Nitric oxide		+
Platelet-derived growth factor, PDGF	+	
Prostaglandins	+	
Transforming growth factor alpha, TGF-α	+	
Tissue inhibitors of metalloproteinases, TIMP	+	
Transforming growth factor β, TGF-β	+	
Tumor necrosis factor, TNF-α	+	
Tumor necrosis factor β, TNF-β	+	
Substance P	+	
Vasoactive intestinal peptide, VIP	+	
Pharmaceuticals		
Bisphosphonates		+
Corticosteroids		+

After Lindskog et al. 2007[1].

OSTEOCLASTS AS DEFENSE CELLS AGAINST MICROBIAL INVASION

In the periodontal ligament (PDL) area, a constant battle takes place between microbial invasion in the cervical area, due to plaque accumulation and periodontal pocket formation, and in the apical area, due to microbial accumulation in the root canal. In both situations, a series of osteoclast-promoting signals are released by microorganisms and inflammatory cells, stimulating osteoclast generation and accelerating osteoclast activities[1,10] (Figure 15-7). This typically results, after some time, in marginal or periapical bone loss. Likewise, if a combined injury has happened to the root surface and the pulp space has become infected, the osteoclast may get a dual stimulation to resorb the root surface. In fact, the first tissue damage to the root surface initiates resorption, and after penetration of the

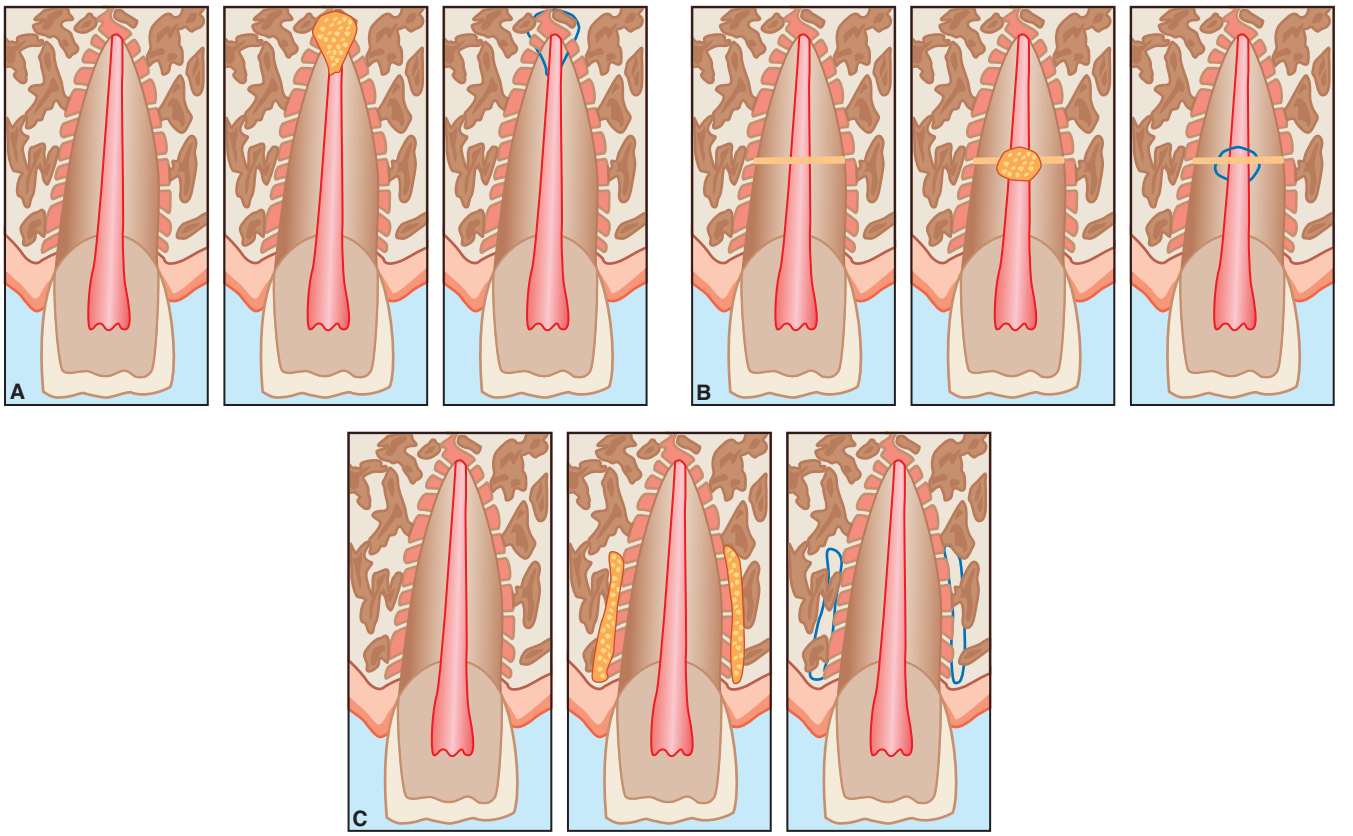

FIGURE 15-6 Examples of various transient healing events. ***A.*** Transient apical breakdown. ***B.*** Transient internal surface resorption. ***C.*** Transient marginal breakdown.

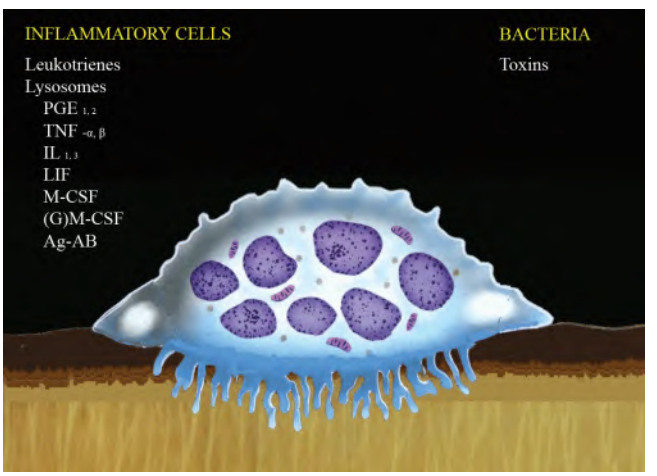

FIGURE 15-7 Osteoclast activators related to bacteria in the root canal and dentinal tubules.

cementum, the osteoclast can be triggered by microbial toxins from the root canal (if present in the canal), through the dentinal tubules, to continue to resorb dentin (see later).

Endodontic Implications

When radiographic evidence of apical or marginal breakdown, or external root resorption, is seen, infection-related root resorption caused by necrotic and infected pulp tissue should be suspected. Endodontic intervention in such cases is indicated and most beneficial.

METHODS FOR CONTROLLING OSTEOCLAST ACTIVITY

A series of osteoclast activators have been identified and all are capable of initiating and activating osteoclast activity.[10] In Figure 15-8, some of the agents that have been used to try to interfere with, or inactivate these triggers of, root resorption are shown.[2,11] These agents, whether administered systemically or topically (in the root canal or on the root surface), have had either no, or a very limited effect on the osteoclastic activity on resorption-prone replanted teeth.[11] The most promising medication appears to be tetracycline and cortisone combinations (e.g., Ledermix®, Lederle Pharmaceuticals, Wolfratshausen, Germany) topically applied in the root canal. In a detailed experimental study in dogs, it was found that the cortisone part of this combination (Ledermix®) had the most significant influence on reducing the extent of the osteoclast attack on the root[11] (Figure 15-9).

An alternative chemical way of arresting or preventing osteoclastic attack on the root surface is to change the dentin environment from a neutral pH to a basic pH. This may

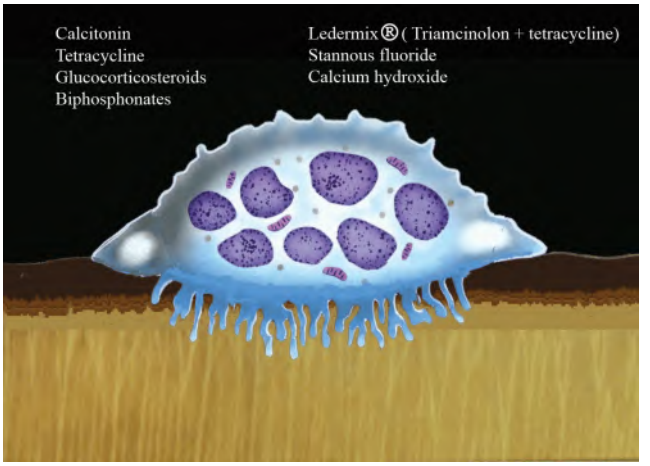

FIGURE 15-8 Example of various medicaments used to influence osteoclast activity.

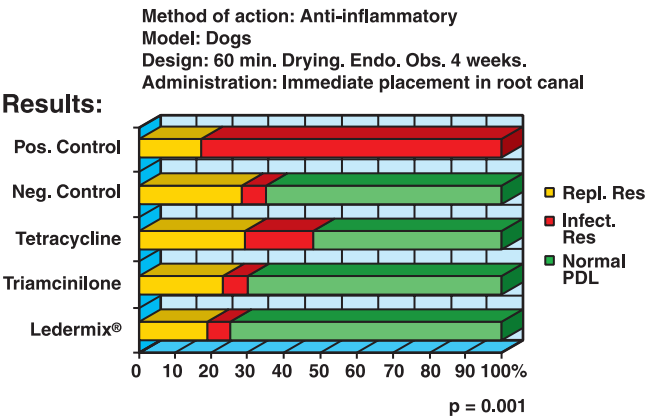

FIGURE 15-9 Effect of Ledermix® (triamcinilone and tetracycline) on PDL healing after replantation. (From Chen et al. 2008[11]; used with permission.)

interfere with the osteoclast's mineral dissolution.[1] Such a change has been shown in several in vitro and in vivo experiments to be possible by an intracanal dressing of calcium hydroxide.[8] In these cases, the pH of the external surface of the dentin reached a level of 9–10.[12,13] In one clinical study, there was a tendency for an elevated pH to result in a slightly higher likelihood of arresting external root resorption.[8]

Elimination of Microorganisms Responsible for Infection Related Resorption

Only one clinical study has investigated the use of antibiotics for infection-related resorption and it appears that short term use of antibiotics (a mixture of neomycin and bacitracin) has almost the same capacity as short term use of calcium hydroxide in reducing the extent of infection-related resorption in replanted human teeth[8] (Figure 15-10). Due to possible side-effects of antibiotics (e.g., allergy) it seems inappropriate today to use antibiotics for these purposes. There might be an indication for disinfection of the root

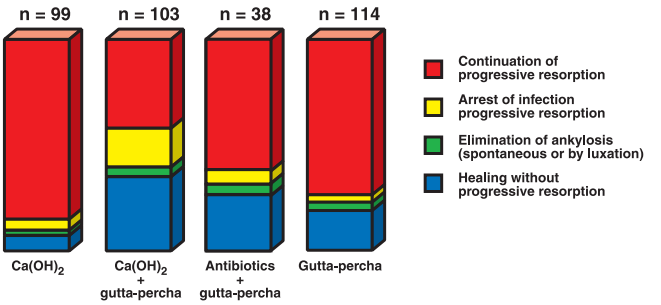

FIGURE 15-10 Periodontal ligament healing related to four types of endodontic treatment of 354 avulsed and replanted teeth. Efficacy of various endodontic procedures to reduce progressive root resorption after replantation in a clinical study of 354 replanted human teeth. (From Andreasen et al. 2015[8]; used with permission.)

canal and dentin with antibiotics in cases where pulp revascularization is intended. Calcium hydroxide, however, is not able in all cases to eliminate all microorganisms in the root canal that seems to be necessary for successful revascularization.

TYPES OF ROOT RESORPTION

The following short systematic synopsis will present the various root resorption types and how they relate to endodontic practice. The reader is also referred to a series of survey articles dealing with diagnosis and treatment of root resorption.[4,14–21]

REPAIR-RELATED RESORPTION (EXTERNAL SURFACE RESORPTION)

Etiology

Repair-related resorption is a part of the healing response to chronic and/or acute tissue injury in the PDL, and it affects the cells adjacent to the root surface. The typical situation where **acute transient** repair-related resorption occurs appears to be following luxation injuries such as concussion, subluxation, and lateral luxation. It can also occur following intrusion and replantation of avulsed teeth. In such cases, it may affect all parts of a root; in root fractures it is observed adjacent to the fracture lines. This type of resorption also occurs frequently after **chronic** injury affecting the PDL such as that associated with orthodontic treatment, traumatic occlusion and pressure from developing cysts/apical granulomas and ectopically erupting teeth. When the trauma and/or pressure is discontinued, spontaneous healing takes place, that is a typical feature of repair-related resorption.[2]

Pathogenesis

Injured tissues, adjacent to the root and surface cementum, are removed by macrophages and osteoclasts. These events

take 2–4 weeks. Repair is accomplished by progenitor cells from the adjacent PDL, that form new cementum and insertion of PDL fibers[2] (Figure 15-11A–C).

Radiographic Findings

After 2–4 weeks, a localized widening of the PDL space can be seen, due to loss of surface layers of the cementum and the bony alveolar socket. At this time, the radiographic images are similar to infection-related root surface resorption. Subsequently though, healing takes place with reformation of the PDL and deposition of hard tissues. Radiographically, slight cavitations may be seen on the root surface.[16,22,23] However, most repair-related root surface resorptions have such minimal size that they cannot be recognized radiographically.[24,25]

Endodontic Implications

Due to their nature (being primarily related to a periodontal injury), endodontic intervention is not indicated for teeth with this type of root resorption. However, a diagnostic problem may exist since the initial stage of infection-related root resorption has an identical beginning.

Treatment

If traumatic and/or pressure factors are eliminated, there is almost 100% repair. If the root apex has been resorbed, increased mobility may become a problem if the root is shorter than 9–10 mm.[26,27]

EXTERNAL INFECTION-RELATED RESORPTION (INFLAMMATORY ROOT RESORPTION)

Etiology

Infection-related resorption was described in 1965 in a clinical and histological study of avulsed, replanted teeth.[22,23] Such trauma represents a combined injury to the pulp and PDL, in which microorganisms, primarily located in the pulp spaces and in the dentinal tubules, trigger osteoclastic activity on the root surface and adjacent alveolar bone. It can affect all parts of the root and is typically diagnosed 2–4 weeks after injury. The resorption is a rapidly progressing process that may result in total resorption of the root within a few months. It is almost exclusively related to acute trauma and is especially common after intrusion injuries and replantation of avulsed teeth.[16]

Pathogenesis

In the event the initial resorption has penetrated cementum and exposed dentinal tubules, toxins from microorganisms present in the infected root canal and/or the dentinal tubules can diffuse via the exposed tubules to the PDL[2] (Figure 15-12A–C). This results in continuation of the osteoclastic process and associated inflammation in the PDL leading to resorption of adjacent alveolar bone. The process is progressive and root dentin is resorbed until the root canal is exposed. If however, microorganisms are eliminated from the root canal and dentinal tubules by endodontic therapy, the resorptive process will be arrested.[27–30] The resorption cavity will then be filled in with cementum or bone, according to the type of vital tissue found next to the resorption site (PDL or bone marrow-derived tissue).

Clinical Findings

A tooth undergoing infection-related root resorption will have increased mobility and a dull percussion tone. Sometimes the tooth may be extruded. Sensibility testing gives no response and sometimes a sinus tract develops.

Radiographic Findings

Infection-related resorption is typically diagnosed 2–4 weeks after injury and appears as progressive cavitations involving the root and adjacent alveolar bone. The resorption is a rapidly progressing event that may result in total loss of root

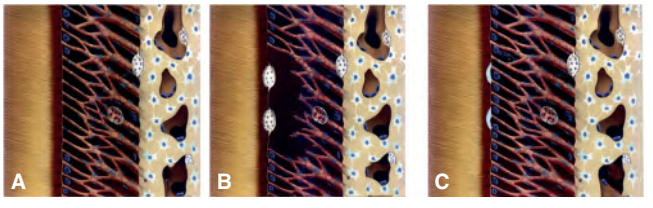

FIGURE 15-11 Pathogenesis of repair-related resorption (surface resorption): healing following minor injury to the periodontal ligament. The injury site is resorbed by macrophages and osteoclasts. The osteoclasts have exposed factors and other soluble molecules in dentin such as IGF-1, TGF-β, PDGF, BMPs, FGF-2, and A4 (amelogenin gene splice products). These molecules may serve as stimulators for cementoblasts. Subsequent repair takes place by the formation of new cementum and insertion of Sharpey's fibers. (From Andreasen and Lövschall, 2007[2]; used with permission.)

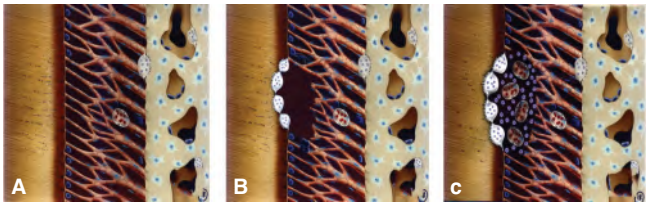

FIGURE 15-12 Pathogenesis of infection-related resorption (inflammatory resorption): Response to moderate or extensive injury to the periodontal ligament and associated infection in the pulp and/or dentinal tubules. The initial injury to the root surface triggers a macrophage and osteoclast attack on the root surface. Osteoclasts are exposed to toxins from microorganisms located in the root canal and dentinal tubules, such as LPS (lipopolysaccharide) MDP (muramyl dipeptide) and LTA (lipoteichoic acid). These toxins serve as direct activators of osteoclastic activity. The resorption process is accelerated and granulation tissue ultimately invades the root canal. (From Andreasen & Lövschall, 2007[2]; used with permission.)

structure after only a few months, particularly in young children (Figure 15-13A–D).

Endodontic Implications

This type of resorption represents a combined periodontal and pulpal injury and requires immediate endodontic treatment to control or remove the osteoclast promoting factors (microbial toxins from the root canal system).

Treatment

The treatment goal is to remove or destroy microorganisms in the root canal and dentinal tubules. This will allow healing to take place in the entire periradicular space (root surface, PDL, and adjacent alveolar bone). Microorganisms are effectively destroyed by using $Ca(OH)_2$ as an intracanal medicament. A side effect, however, of using $Ca(OH)_2$ for a long term (>30 days) is a weakening of the root structure in **immature** teeth that can lead to cervical root fracture. This is a risk that has been found to be as high as 66%–72% in two clinical studies.[30,31] In **mature** teeth, the problem apparently does not exist.[30] The endodontic technique, therefore, should be related to the maturity of the tooth.

Mature Teeth

Replantation of avulsed, fully developed, teeth needs to include prophylactic extirpation of the pulp to prevent infection-related resorption. Biomechanical canal preparation should include the use of sodium hypochlorite and $Ca(OH)_2$. The latter medication can be expected to accomplish its task of disinfection of the canal in 2–3 weeks.

Immature Teeth

In a tooth where the root is not fully developed, and where the pulp becomes infected, the apical opening is often large and thus, placing a root filling can be difficult. In such cases, apexification procedures, using calcium hydroxide, have been performed with good success. The disadvantage of using calcium hydroxide for apexification is that it takes many months to obtain enough of an apical barrier to allow placement of a root canal filling. Additionally, and as stated earlier, long-term use of $Ca(OH)_2$ may weaken dentin, possibly by dissolving its organic component, and thereby resulting in cervical root fracture from slight impacts or even normal use.[31,32] By using mineral trioxide aggregate (MTA) and similar materials as a physical barrier apically, a root canal filling can be placed immediately without waiting for a biological response (Figure 15-14A–H). Minimizing exposure of immature root dentin to calcium hydroxide, may result in less damage to the dentin.[33,34]

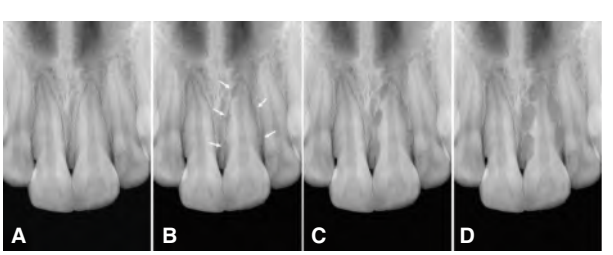

FIGURE 15-13 Progression of infection-related root resorption. If left untreated, the root will resorb rapidly, particularly in children. (From Andreasen et al. 2011[58]; used with permission.)

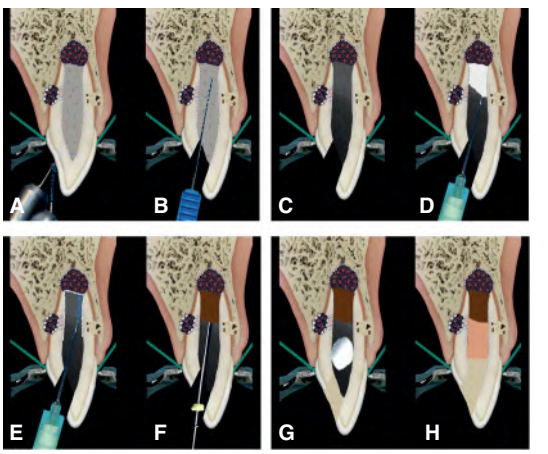

FIGURE 15-14 Combined calcium hydroxide and MTA treatment of a traumatized permanent tooth with immature root formation. ***A.*** The tooth is isolated with dental dam, the crown disinfected, and an access cavity to the root canal prepared. ***B.*** Extirpation of necrotic pulp tissue is done to a level apically, where fresh bleeding from healthy tissue is encountered. This can be anywhere from several millimeters from the apex to flush with the apical foramen. ***C.*** Root canal preparation in developing teeth requires a conservative approach, so as to preserve as much root dentin as possible. Hence minimal canal shaping is appropriate. ***D.*** Disinfection of the root canal with sodium hypochlorite is followed by short-term (approx. 2–4 weeks) interim dressing with calcium hydroxide. The use of calcium hydroxide allows acceptable disinfection of the root canal system and provides a dry root canal, free of seepage of apical exudates. The coronal access opening must be sealed with a dependable temporary restoration. ***E.*** At the next visit, the calcium hydroxide is removed and the canal is thoroughly irrigated with saline or sodium hypochlorite to obtain a debris-free canal. Small increments of the MTA-water mixture are introduced into the canal and gently condensed. Length can be controlled using a rubber stopper on a plugger. No harm is done, however, with slight overfilling. ***F.*** When properly condensed, the apical MTA plug should be at least 4 mm thick. The entire canal can be filled with MTA (to the cervical level), or the coronal part of the canal can later be filled with gutta-percha and sealer. ***G.*** To allow setting, a moistened (water) cotton pellet is placed in the access cavity that is then sealed with a temporary filling material. Once set (usually within 4–6 hours), the canal can be conventionally filled. ***H.*** The temporary material and cotton pellet are removed, the apical plug is checked for setting hardness; it should not be vigorously probed, as the material can break. The canal is then irrigated, dried, and filled, followed by a bonded coronal composite restoration. (From Andreasen et al. 2011[58]; used with permission.)

Prognosis

Dentin lost through resorption cannot be replaced with new dentin, at least with today's treatment approaches. Healing occurs after arresting the resorption process and with either a replacement layer of new cementum or bone and establishment of new PDL.[35–37]

EXTERNAL ANKYLOSIS-RELATED RESORPTION (REPLACEMENT RESORPTION)

Ankylosis-related PDL complications represent a sequel to a defect in, or injury to, the PDL cells, including the cell layer next to the cementum.[2] The most frequent cause appears to be acute trauma[2] (severe luxations such as lateral luxations, intrusions, and replantation of avulsed teeth). In those situations, the homeostasis of the PDL has been reduced. This leads to healing events taking place from the bony alveolus resulting in creation of a bony bridge between the socket wall and the root surface (Figure 15-15A–C).

In situations of moderately sized injuries (1–4 mm^2) an initial ankylosis forms that can be reversible. If the tooth is allowed functional mobility such as by the use of a nonrigid splint, or no splinting, small areas of resorption can later be replaced with new cementum and PDL attachment (transient ankylosis).[38–42] In more extensive injuries (>4 mm^2), a progressive ankylosis takes place. This implies that the tooth becomes an integral part of the bone remodeling system. The entire process includes osteoclastic resorption dependent upon bone remodeling processes, parathyroid hormone induced resorption, remodeling due to function, and resorption due to microorganisms present in the gingival area and/or the root canal.[2] All of these processes are very active in children and lead to gradual infraocclusion and arrested development of the alveolar process.[43] As a result, this combination of resorption processes leads to loss of ankylosed teeth within 1–5 years. In older individuals, ankylosis-related resorption is significantly slower and often allows a tooth to function for much longer periods of time (i.e., 5–20 years), and the position of the tooth in the arch remains the same (similar to a dental implant).[44]

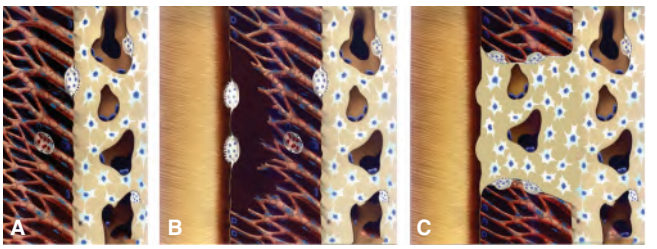

FIGURE 15-15 Pathogenesis of ankylosis-related resorption (replacement resorption): Response to extensive injury to the periodontal ligament. The osteoclasts have been exposed to stimulating factors and other soluble molecules in dentin such as IGF-1, TGF-β, PDGF, BMPs, FGF-2, and A4 (amelogenin gene splice products). These molecules may serve as stimulators for cementoblasts and/or osteoblasts. Ankylosis is formed because healing occurs almost exclusively by cells from the alveolar wall. (From Andreasen & Lövschall, 2007[2]; used with permission.)

Clinical Findings

A tooth undergoing ankylosis-related root resorption appears very firm in its socket, eliciting a high metallic sound on percussion. This can be demonstrated after 4–6 weeks post-trauma.[42,45]

Radiographic Findings

The ankylosis-related resorption process is in response to extensive damage to the layer of the PDL closest to the root surface. Because of the normal remodeling characteristic of bone, the root structure is gradually replaced by bone (Figure 15-16A–D). Ankylosis is a frequent occurrence after intrusion and replantation of avulsed teeth. It can usually be diagnosed radiographically within 2 months after injury and clinically, within 1 month by a high percussion sound.[42,45]

Endodontic Implications

Endodontic therapy cannot arrest progressive ankylosis-related resorption. If the pulp is vital, endodontic treatment is not indicated. In the case of an associated pulp necrosis, the treatment described for infection-related root resorption is applicable, but it will only address the related infection-related situation.[8]

Treatment

In children and adolescents, an ankylosed tooth will fail to erupt and will gradually be in infraposition with respect to the adjacent teeth. The younger the age, the more pronounced would be the infraposition (Figure 15-17A, B). Presently, the following are possible treatment options: (1) Decoronation (in order to maintain and augment the alveolar process); (2) luxation of the tooth (breaking of ankylosis sites if they are minimal); (3) extraction with ridge augmentation in preparation for later implant placement; (4) extraction and prosthetic bridge; (5) distraction osteogenesis; (6) prosthetic elongation; and (7) extraction and transplantation of a premolar tooth.

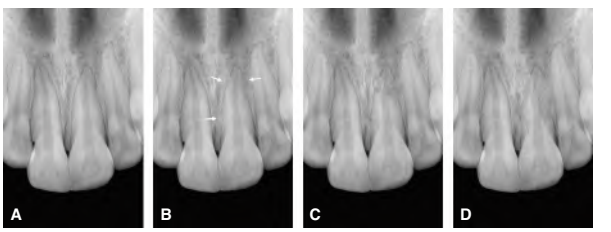

FIGURE 15-16 Progression of ankylosis-related resorption (replacement resorption). (From Andreasen et al. 2011[58]; used with permission.)

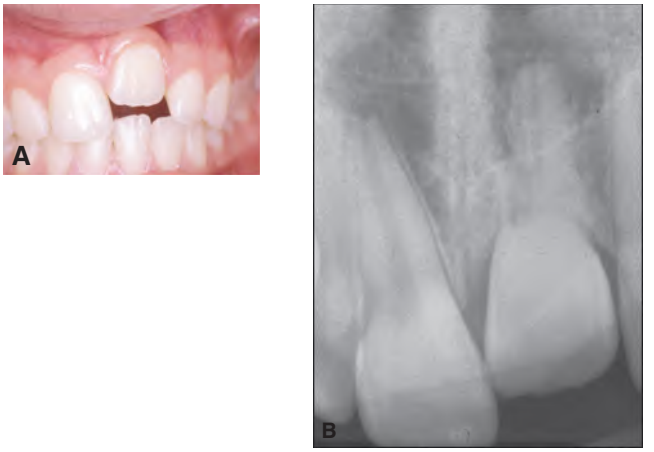

FIGURE 15-17 Infrapositioned maxillary central incisor initially intruded at the age of 9. ***A.*** Clinical photograph shows position of tooth at age 13. ***B.*** Radiograph shows extent of ankylosis-related resorption. The pulp retained vitality, that is not unusual in teeth with ankylosis-related resorption.

Decoronation treatment is very suitable in children and adolescence where significant remaining alveolar growth is expected. The procedure involves removal of the crown of the tooth (leaving what remains of the root) allowing continued vertical growth of the alveolus.[46–49] If the crown is surgically removed slightly below the cervical bone level and any root filling or pulp tissue removed, the reformation of the interdental fibers, dento-periosteal fibers and the periosteum will lead to a normalization of the bone level.[48] Due to the ankylosis-related resorption of the remaining root with bone replacement, the alveolar structure will be optimal for implants placed later when growth has been completed.[46–49]

EXTERNAL SPONTANEOUS ANKYLOSIS-RELATED RESORPTION

Etiology

This type of ankylosis may affect one or several primary or permanent teeth. The etiology is presently not known. It is suspected to be related to instability of the RANK-RANKL-OPG system[2] (Figure 15-18A, B).

Pathogenesis

This ankylosis-related resorption process leads to infraposition of the involved teeth in young individuals and in all cases leads to gradual substitution of the root substance with bone.

Clinical Findings

Primary dentition: This type of resorption especially affects primary molars and the mandibular second primary molar is most prone to be involved (20%). The ankylosis leads to gradual infraposition and tilting of neighboring teeth.[50]

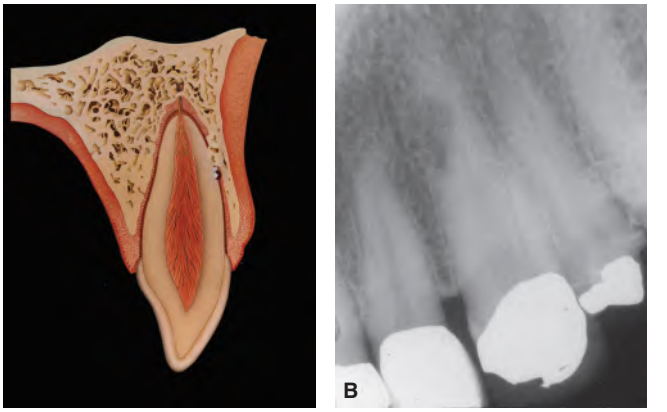

FIGURE 15-18 External spontaneous ankylosis resorption. ***A.*** Graphic illustration. ***B.*** Clinical example.

Permanent dentition: The first and second permanent molars are most prone to show this type of resorption. These molars will, in young individuals, show a gradual infraposition and the percussion tone is high and metallic.[6,7]

Radiographic Findings

Primary molars: The ankylosis process starts in the interdental area and gradually spreads to the remaining part of the root.

Permanent molars: The first molars will, in most cases, show replacement resorption starting in the intraradicular area. From this location it will gradually spread to the remaining part of the root (Figure 15-19). Semi-impacted or impacted third molars will, only in rare cases, show ankylosis.

Endodontic Implications

This is primarily a PDL problem and endodontic intervention may actually aggravate the aggressive nature of the ankylosis process.

Treatment

Primary dentition: If diagnosed early, decoronation is a reasonable option. If diagnosed late in adolescence, the crown may be rebuilt to prevent over-eruption of the antagonist.

Permanent dentition: If diagnosed in early adolescence, the ankylosed tooth should be extracted. At later stages, the crown may be rebuilt with a restoration to achieve a functional occlusal level.

Pathogenesis

Any permanent tooth may become involved and the process takes place over 10–20 years. It usually affects a single group of teeth (premolars, molars) and gradually affects other groups of teeth.

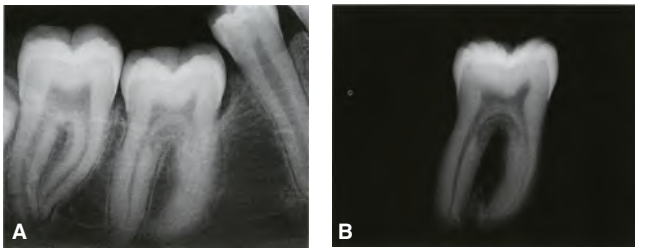

FIGURE 15-19 Spontaneous ankylosis affecting the interdental area of a first molar. (From Andreasen et al. 1996[6]; used with permission.)

INVASIVE CERVICAL RESORPTION

Etiology

While the etiology of invasive cervical resorption remains obscure, a defect in the cementoblast layer seems to be a prerequisite, coupled with a breakdown of the RANK-RANKL-OPG system.[1] In a large clinical study of 222 patients (total of 257 teeth) with invasive cervical resorption, several potential predisposing factors were identified.[51] The most frequently identified single factor was orthodontic treatment (21.2%), followed by trauma (14%). Often there was a combination of factors, for example, trauma and bleaching (7.7%), whereas bleaching as a sole factor was found in 4.5% of the patients studied.[52]

Pathogenesis

Hyperplastic resorptive tissue, apparently derived from precursor PDL cells, invade the hard tissues of the tooth in a destructive, invasive, and apparently uncontrolled fashion akin to the nature of some fibro-osseous lesions. Initially, the resorptive tissue is fibrovascular in nature (Figure 15-20) but as the resorptive process extends more deeply into radicular root structure, it becomes fibro-osseous in character, as shown in Figure 15-21. Ectopic bone-like deposits can be observed both within the lesion itself and at the interface with resorbed dentin. In addition, the resorptive tissue creates a series of channels that encircle the root canal, infiltrate radicular dentin, and also interconnect with the PDL at other locations on the root surface.[53,54]

Clinical Findings

This relatively uncommon type of external root resorption may, on occasion, show no external signs, nor elicit any clinical symptoms, and may be diagnosed primarily as the result of a chance radiographic finding. An example is shown in Figure 15-22A,B. The initial clinically observable sign may be a slight reddish defect near the gingival margin of an affected tooth, or a pink discoloration of coronal tooth structure (Figure 15-23A). Ultimately, with extensive invasive cervical resorptions, there may be enamel cavitation (Figure 15-24A). The pulp remains intact until late in the process, apparently protected from the resorptive tissue by a layer of predentin and dentin

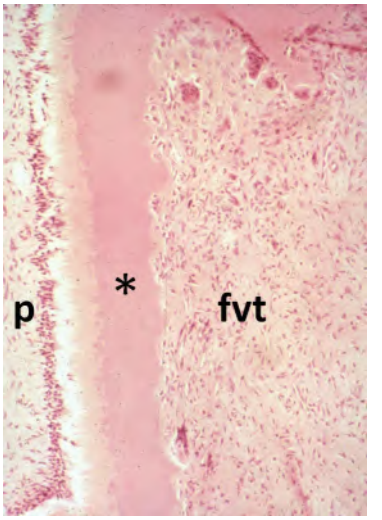

FIGURE 15-20 Histologic appearance of an incisor tooth with the first stage of invasive cervical resorption. An intact layer of predentin and dentin (*) separates the pulp (p) from the mass of fibro-vascular resorptive tissue (fvt) that contain both mononucleated and multinucleated clastic cells. (Reproduced from Heithersay GS. Quintessence Int. 1999;30:27–37 with permission, Quintessence Publishing.)

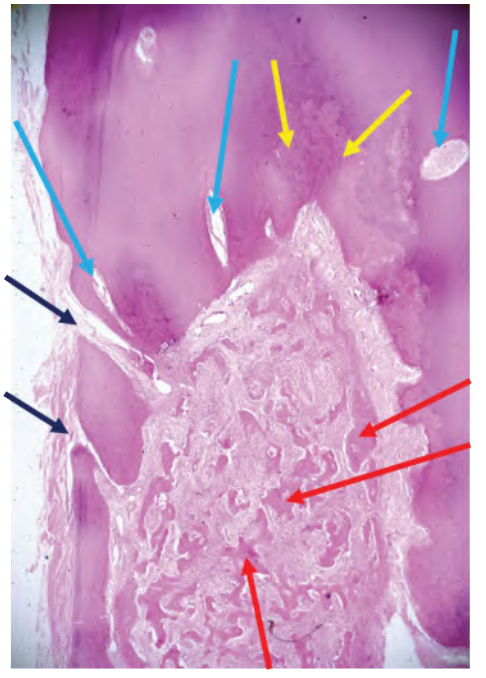

FIGURE 15-21 Histological appearance of an extensive invasive cervical resorption with radicular extensions. The resorptive tissue within the resorption cavity is fibro-osseous in character (red arrows) and there are bone like deposits at an interface with resorbed dentin, akin to replacement resorption (yellow arrows). There are also intercommunicating channels with the periodontal ligament (black arrows) and deeply penetrating small channels within the residual dentin (blue arrows). (Reproduced from Heithersay GS. Quintessence 1999;30:27–37, with permission, Quintessence Publishing.)

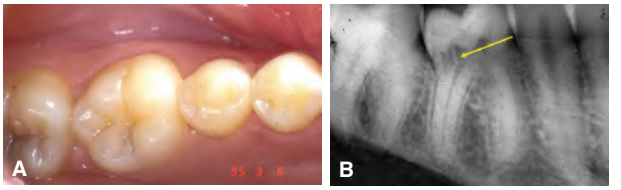

FIGURE 15-22 ***A.*** Crown of a mandibular first molar with no external signs of resorption. ***B.*** Radiograph of the tooth pictured in ***A*** showing an irregular radiolucency extending from the distal cervical region into the crown and adjacent to the pulp space but separated by a radiopaque line (yellow arrow).

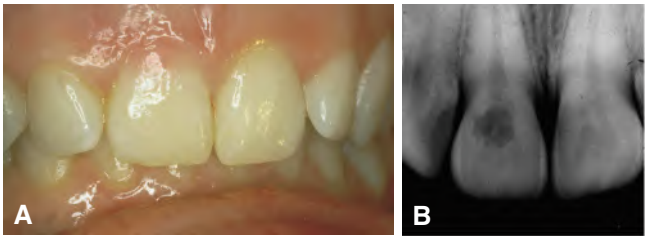

FIGURE 15-23 ***A.*** The maxillary right central incisor of a 21-year-old female shows a pink discoloration of the cervical third of the crown. ***B.*** A radiograph of the central incisor shows an irregular radiolucency overlying the root canal outline.

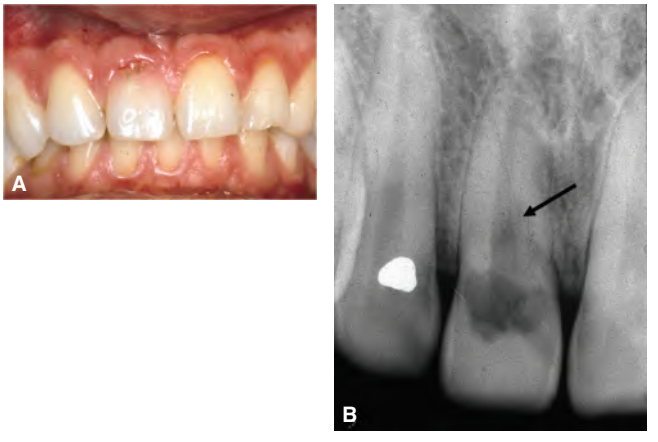

FIGURE 15-24 ***A.*** Cavitation of labial enamel with exposure of underlying resorptive tissue can be observed in the crown of the maxillary right central incisor, that also shows a pink discoloration. ***B.*** Radiograph of the tooth pictured in ***A*** shows an irregular radiolucency in the tooth crown and extending into the coronal third of the root (arrow). The pulp space is demarcated from the image of the resorptive lesion by a radio-opaque line. (Reproduced from Heithersay GS. Quintessence Int. 1999;30:27–37; used with permission from Quintessence Publishing.)

(Figure 15-20). Generally, the affected tooth remains asymptomatic unless there is superimposed infection late in the progression of the resorptive process, that will likely lead to pulpal or periodontal symptoms.[52,53]

Radiographic Findings

The periapical radiographic features will vary depending on the location and extent of the resorptive process. Initially, there may be a slight radiolucency at the cervical region but with invasion of the coronal tooth structure, the image may have an irregular appearance overlying the outline of the root canal (Figure 15-23B). A characteristic radio-opaque line in some radiographs demarcates the intact predentin–dentin protective layer from the irregular image of the invading resorptive tissue in an extensive process (Figure 15-24B) and in a mandibular molar with outwardly a normal clinical appearance[52,53] (Figure 15-22B,G,H).

Cone-beam computed tomography can be used to advantage in more extensive cases of invasive cervical resorption to identify the location of the main portal of entry of the resorptive tissue and the extent of the resorptive process (Figure 15-25A,B). This enhanced imaging can be important in treatment planning where alternative forms of management are to be considered (e.g., implant therapy). For more details, see Chapter 10, Imaging Interpretation. A clinical classification, largely based on radiographic evidence, developed in 1999 as an aid to diagnosis and treatment planning, is shown in Figure 15-26.[51]

Endodontic Implications

In class 1 and class 2 invasive cervical resorptions, the pulp is generally not involved in what is essentially a periodontally derived external resorptive process does not indicate endodontic therapy. It will only be indicated if the resorption has extended to the pulp. In the more extensive class 3 and class 4 invasive cervical resorptions, the pulp will ultimately be encroached and in the treatment of class 3 cases endodontic therapy is recommended as follows.

Treatment

Two treatment modalities have been reported.[54] The first and frequently employed method involves periodontal flap reflection, curettage of the resorptive tissue, and restoration of the defect with a composite resin or glass ionomer cement. An example of this approach is shown in Figure 15-27A–D.

The second treatment method involves the topical application of a 90% aqueous solution of trichloracetic acid (TCA) to affect a coagulation necrosis of the highly active and invasive resorptive tissue, followed by curettage, endodontic treatment, if indicated, and restoration.[54] An example of this treatment modality carried out in the class 2 invasive cervical resorption case illustrated previously in Figure 15-23A,B, is shown in Figure 15-28A,B. Five year clinical and radiographic follow-up images are shown in Figure 15-28C,D.

In class 3 cases, the coronal extension of the resorption is treated with the topical application of TCA as mentioned previously, followed by prophylactic endodontic therapy to facilitate access for treatment of the resorptive tissue encircling the root canal. The further careful topical use of TCA to this region followed by curettage should not only inactivate this encircling resorptive tissue but also aid in controlling further

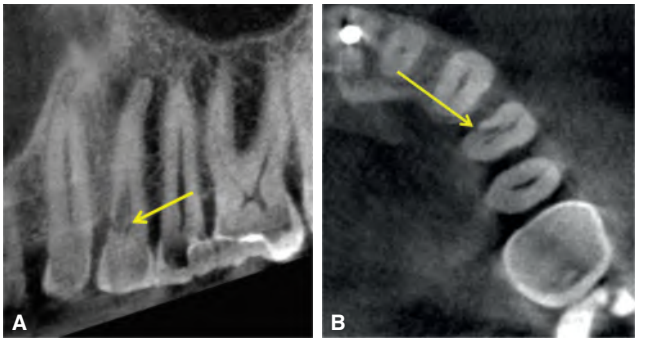

FIGURE 15-25 ***A.*** CBCT sagittal image showing radiographic evidence of an extensive invasive cervical resorption in a maxillary left first premolar. A characteristic radio-opaque line can be observed at the interface between the image of the resorptive tissue and the dental pulp (yellow arrow). ***B.*** CBCT axial view of the same tooth shows the mesio-palatal portal of entry of the resorptive defect (yellow arrow). (Courtesy of Drs. Randolf Todd and Paul Fletcher, New York, NY.)

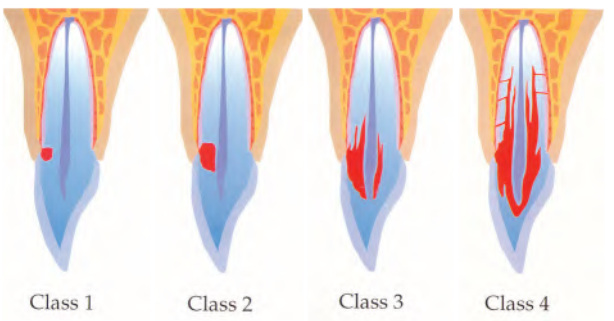

FIGURE 15-26 Clinical classification of invasive cervical resorption. Class 1 denotes a small invasive resorptive lesion near the cervical area with shallow penetration into dentin; Class 2 denotes a well-defined invasive resorptive lesion that has penetrated close to the coronal pulp chamber but shows little or no extension into the radicular dentin; Class 3 denotes a deeper invasion of dentine by resorbing tissue, not only involving the coronal dentin but also extending into the coronal third of the root; Class 4 denotes a large invasive resorptive process that has extended beyond the coronal third of the root. (Reproduced from Heithersay GS Quintessence Int. 1999;30:83–95; used with permission from Quintessence Publishing.)

progression of resorptive tissue contained in small deeply infiltrating channels (Figure 15-21). An illustrative case is shown in Figure 15-29A–H. Orthodontic extrusion can also be employed to advantage as an adjunctive therapy in some instances to render the repaired resorptive defect into a more harmonious position in respect to the gingival crevice.

The results of a prospective study of 101 teeth with varying degrees of invasive cervical resorption, using the second approach, have been reported.[53,54] The results judged by tooth survival and no radiographic evidence of

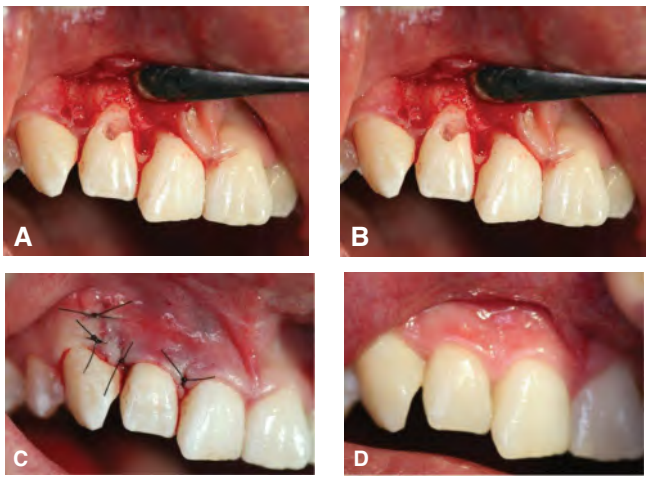

FIGURE 15-27 Management of a class 2 invasive cervical resorption by flap reflection, curettage of resorptive tissue, and restoration. ***A.*** Following periodontal flap refection and curettage of resorptive tissue, the extent of the resorption cavity is revealed showing no involvement of the dental pulp. ***B.*** Restoration of the resorptive cavity with composite resin. ***C.*** Flap repositioned and sutured. ***D.*** Excellent soft tissue healing response is evident. (Courtesy of Dr. Steve Soukoulis, Wayville, Australia.)

resorption, periradicular or periapical pathosis, showed that the treatment modality was entirely successful in class 1 and class 2 situations and acceptably successful in class 3 cases. However, as there was a low success rate for class 4 resorptions, no treatment can be generally recommended. While the progression of the resorption in class 4 cases may be slow, ultimately extraction and replacement with an implant or other prosthodontic means will be necessary.

Invasive Coronal Resorption

In rare instances, where there has been a defect in enamel integrity in an erupting tooth, an invasion of coronal tooth structure by hyperplastic resorptive tissue can occur (Figure 15-30A,B). The enamel defect is generally due to an area of hypomineralization, that can occur developmentally or as a result of intrusive luxation of a primary tooth impacting on the developing permanent successor. The same management principles apply as with invasive cervical resorption.

Invasive Radicular Resorption

When the invasion of resorptive tissue occurs in a more apical, rather than cervical location, the term invasive radicular resorption is applied. It generally occurs in patients who have had vertical bone loss due to periodontal disease so that their gingival attachment is more apically placed. This type of resorption follows a similar pattern to that of invasive cervical resorption but generally there are no extensions into coronal tooth structure.

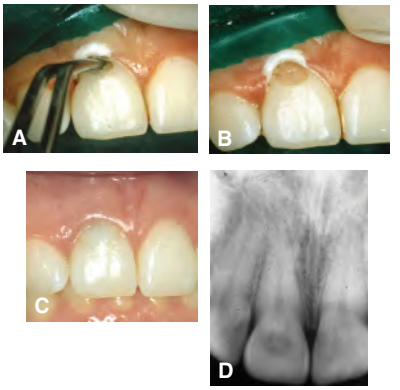

FIGURE 15-28 Management of the class 2 invasive cervical resorption shown in Figure 23-*A,B* by topical application of a 90% aqueous solution of trichloracetic acid (TCA), curettage, and restoration. ***A.*** After a protective application of glycerol to adjacent soft tissue, a dental dam "cuff" has been placed for protection and isolation. This has been supplemented with a glycerol impregnated cotton roll placed in the labial sulcus. TCA, on a small cotton pellet, has been applied to the resorptive defect with slowly increasing pressure, so that the resorptive tissue within the cavity undergoes coagulation necrosis. ***B.*** Following curettage of the avascular tissue from the resorption cavity, the glistening dentinal base of the cavity has been revealed. The incisal margin of the cavity was smoothed with high-speed bur under water spray and a glass-ionomer restoration was then placed in the cavity, and its surface protected with an unfilled resin. ***C.*** Clinical appearance of the tooth 5 years postoperatively. The original glass-ionomer cement has been faced with a composite resin restoration. ***D.*** 5-year follow-up radiograph of the maxillary right central incisor shows no evidence of periapical pathosis or extension of the treated resorptive lesion. (Reproduced from Heithersay GS, Quintessence Int. 1999; 30:96–110; used with permission from Quintessence Publishing.)

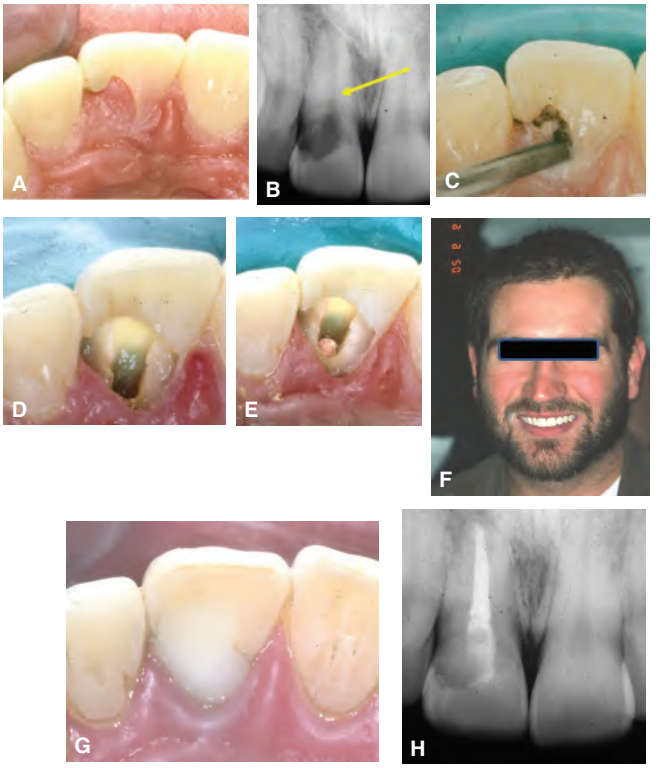

FIGURE 15-29 ***A.*** A mass of soft tissue is evident in a defect on the palatal aspect of the auxillary right central incisor of a 19-year-old male with a history of orthodontic treatment 6 years earlier. The labial surface of the patient's dentition showed a normal appearance. ***B.*** A radiograph of the tooth shows a large, irregular radiolucency extending both coronally and into the radicular tooth structure (yellow arrow). This hyperplastic invasive cervical resorption is classified as class 3. ***C.*** Topical application of TCA, on a small cotton pellet, has been carried out with slowly increasing pressure, to prevent hemorrhage and effect a coagulation necrosis of the resorptive tissue mass. ***D.*** Following curettage of the devitalized tissue in the coronal extent of the resorption, elective pulpectomy and canal enlargement with a Gates Glidden drill has been carried out to allow access to encircling resorptive tissue. Further resorptive tissue inactivation can be affected by the careful application of TCA to the area. Visualization with enhanced vision, or microscope, is important to confirm the removal of all resorptive tissue. ***E.*** Root canal filling with gutta-percha and sealer was done following initial root canal dressing with a corticosteroid antibiotic paste (Ledermix® paste) and temporization. The two stage procedure allowed a further visual check of the margins of the resorption cavity and the contents of the root canal. The cavity was then restored with glass-ionomer cement protected with an unfilled resin. ***F.*** Facial view of the patient at a 10-year recall. ***G.*** Palatal view of the treated central incisor. The original glass ionomer restoration has been refaced with composite resin due to some surface crazing. ***H.*** A 10-year follow-up radiograph shows no evidence of further resorption or periradicular pathosis. (Reproduced from Heithersay GS, Quintessence Int. 1999; 30:96–110; used with permission from Quintessence Publishing.)

MULTIPLE EXTERNAL INVASIVE CERVICAL RESORPTION, OR SITES OF ANKYLOSIS, OR INFECTION-RELATED RESORPTIONS

Etiology

These are very rare types of root resorptions; only a handful of new cases are reported each year in Denmark (6 million inhabitants). The etiology is suspected to be a defect in the RANK-RANKL-OPG system and a hereditary background is found in some cases.[2] A viral etiology acquired by contact with cats displaying feline invasive cervical resorption has also been reported.[55] The possibility of drug induction of these resorptions has been raised and a recently reported case series implicated bisphosphonate therapy.[56]

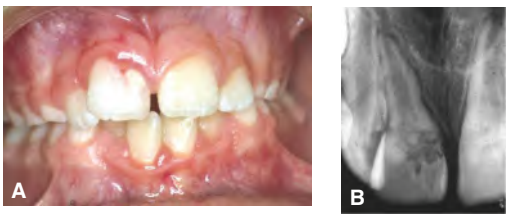

FIGURE 15-30 ***A.*** Hyperplastic invasive coronal resorption in the maxillary right central incisor of a 11-year-old male; the resorption is apparently associated with an area of hypomineralization of the crown. ***B.*** Radiograph of the maxillary right central incisor showing an extensive irregular radiolucency indicative of hyperplastic invasive coronal resorption. (Reproduced from Heithersay GS. *Aust Dent J* Suppl. 2007; 52(Suppl. 1):S105–S12; used with permission from Wiley Blackwell.)

Pathogenesis

As with the preceding section, hyperplastic resorbing tissue, apparently derived from precursor PDL cells, invade the hard tissues of multiple teeth in the cervical region in an extremely aggresive and destructive manner. Superimposed microbial invasion in advanced lesions will result in pulpal or periodontal pathosis that may assume the characteristics of ankylosis or infection-related resorptions.

Radiographic Findings

The radiographic images of multiple invasive cervical resorptions will vary from relatively small radiolucencies at the cemento-enamel junction to extensive radiolucencies indicative of tooth destruction due not only to invasive cervical resorption but may also be due to superimposed pulpal and periodontal pathosis.

Clinical Findings

The pattern of development of these multiple resorptions is unclear as it is often difficult to trace the date of the initiation of the lesions. However, it could be estimated that the process variously takes place over a matter of months to several years. Some cases indicate a spontaneous initiation in multiple teeth, while others indicate a spread of the resorptions from one tooth to another. Often the patient may be unaware of the resorptions as there may be no symptoms or external signs either in the tooth crowns or the gingival tissues. A spontaneous crown/root fracture may precipitate a dental examination where the full extent of the condition will be revealed (Figure 15-31A–D). Alternatively, some tooth crowns may show the typical pink coronal discoloration and symptoms with associated pulpal or periodontal pathosis may develop in advanced cases.

Endodontic and Periodontal Implications

Due to the extremely aggressive nature of the cells involved in these resorptions, the prognosis for successful management is poor.

Treatment

Heroic treatment could involve periodontal flap reflection, application of 90% aqueous TCA to the resorptive tissue

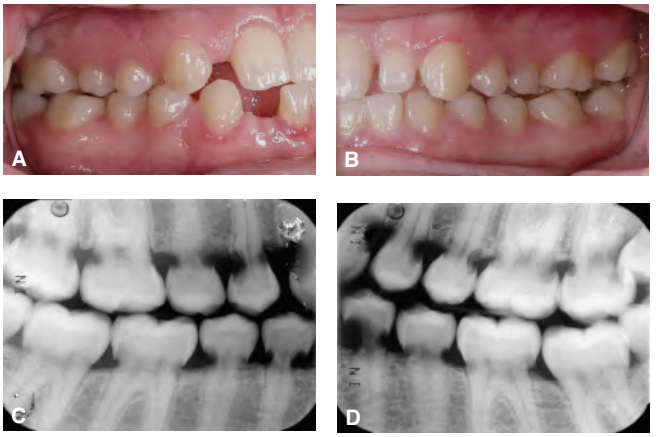

FIGURE 15-31 ***A & B***. Clinical appearance of the caries-free dentition of a 27-year-old female who sought dental advice after her mandibular right lateral incisor spontaneously fractured subgingivally Other than gingival inflammation associated with the mandibular right central incisor, the gingival soft tissues appear normal and in an apparently healthy state. ***C & D***. Radiographs of the right and left posterior segments show varying degrees of invasive cervical resorption in the entire dentition. The most severely affected teeth seen in these radiographs are the maxillary and mandibular premolars and canines. Similar gross destruction was evident in the mandibular anterior region and the retained root of the fractured mandibular right lateral incisor was still present. (Courtesy of Dr. Paul V. Abbott, Nedlands, Western Australia, Australia.)

followed by curettage, endodontic treatment (if there is pulp involvement), and finally restoration. Long term, these patients will sadly face prosthodontic replacement of the involved teeth.

INTERNAL SURFACE RESORPTION

Etiology

Internal surface resorption can be found in areas where revascularization occurs, such as in fracture lines of root fractures, and in the apical part of the root canal of a luxated tooth undergoing revascularization[57–59] (Figures 15-6B and 15-32).

Pathogenesis

Osteoclastic activity is part of the process along with the formation of granulation tissue.

Radiographic Findings

Radiographically, there appears to be a temporary widening of the root canal.

Endodontic Implications

It is very important to consider that this transient resorption process is a sign of progressing pulp healing and that any endodontic intervention may arrest this process.

Treatment

No treatment is recommended except to monitor for radiographic changes.

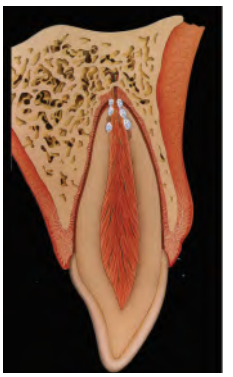

FIGURE 15-32 Internal (root canal) surface resorption.

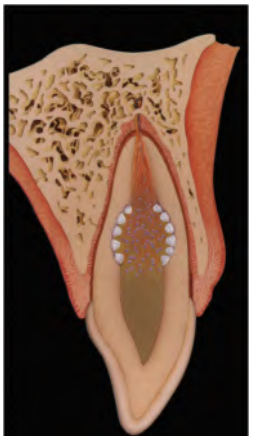

FIGURE 15-33 Internal infection-related root resorption. The resorption site represents resorbing granulation tissue interposed between vital (apically) and diseased (coronally) pulp tissue.

INFECTION-RELATED INTERNAL RESORPTION

Etiology

Coronal to the resorption site in the pulp is found necrotic infected tissue (root canal and dentinal tubules or pulp tissue with chronic inflammation). The resorption site represents resorbing granulation tissue interposed between healthy and diseased pulp tissue[60] (Figure 15-33). This type of resorption can also be found associated with root fractures.

Pathogenesis

The resorptive process will gradually expand leading to perforation or fracture of the root.

Treatment

Endodontic treatment is appropriate and requires a technique that allows management of the resorbed area of the root canal (Figure 15-34A, B).

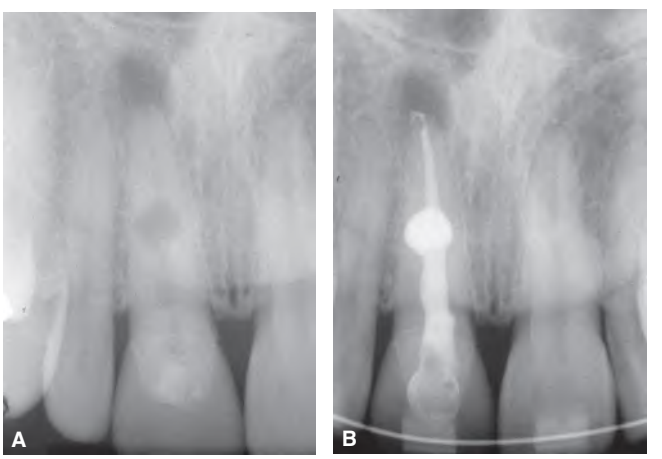

FIGURE 15-34 Teeth with internal root resorption can be treated successfully with any of the many endodontic techniques that allow filling material to be compacted into canal irregularities. ***A.*** Preoperative radiograph of a maxillary central incisor with internal resorption midroot. ***B.*** Radiograph after completion of the endodontic treatment procedure. (Courtesy of Dr. Steve Morrow, Loma Linda University, California.)

INTERNAL REPLACEMENT RESORPTION

Etiology

When damaged pulp tissue is replaced as part of a healing process, tissue metaplasia may take place leading to formation of bone tissue in the pulp canal. The damage to the pulp tissue is usually related to trauma and the damaged pulp tissue is replaced with an ingrowth of new tissue, which includes bone-derived cells.[57]

Pathogenesis

The root will gradually be replaced with bone. In some cases the bone replacement will spontaneously arrest.

Clinical Findings

The teeth are asymptomatic but if ankylosis occurs, the teeth will gradually develop infraocclusion in young patients.

Radiographic Findings

A dissecting resorptive area may be seen in the root canal; initially the root canal appears intact. Mesio-distal and ortho-radiographic exposures should verify the central location of the ankylosis site.

Endodontic Implications

Since the eventual outcome of allowing this type of condition to continue is the loss of the tooth, it may be advantageous to treat the tooth endodontically, in spite of the fact that that may lead to a poor prognosis due to the lack of root maturity.

SUMMARY

With few exceptions, pathologic tooth resorptions have endodontic implications that clinicians can address. It is

clear that most infection-related resorptions respond well to endodontic treatment. It is hoped that the future will provide avenues that currently are not available for successful management of ankylosis-related resorptions.

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Chapter 16

Rhinosinusitis and Endodontic Disease

Roderick W. Tataryn

Rhinosinusitis is the single most common chronic medical condition in the United States affecting approximately 15% of the population.[1] Sinus pain, caused by inflammation, or infection, of the maxillary sinus, is often perceived by patients as odontogenic pain, and therefore deserves important consideration when developing a differential diagnosis for a maxillary toothache.[2] Despite its prevalence, differentiating pain caused by rhinosinusitis from that of dental etiology can be one of the more difficult diagnostic challenges facing clinicians. The result of a misdiagnosis in this area can lead to unnecessary endodontic therapy, periapical surgery, and even multiple tooth extractions, with no effective pain relief for the patient.

In order to effectively distinguish rhinosinusitis from odontalgia, it is imperative that clinicians understand the anatomy and functions of the paranasal sinuses both in healthy and diseased states, and recognize the signs and symptoms associated with sinus inflammation. It should be made clear that it is not expected of the endodontist, and is in fact outside the scope of dentistry, to definitively diagnose or provide treatment for a sinus condition. This diagnosis and treatment is best left to an otolaryngologist, or ear, nose, and throat physician (ENT). Nevertheless, it is essential that the endodontist have a thorough understanding of this system and its potential disease processes, in order to effectively diagnose or rule out pain of odontogenic origin, and to make an appropriate ENT referral if rhinosinusitis is suspected. Knowledge of sino-nasal structures and their function is also crucial to understanding the effects of periapical infection on the adjacent sinus tissues and the causative role that endodontic disease has in a significant percentage of sinus infections.

THE PARANASAL SINUSES: ANATOMY AND FUNCTION

Sinuses are hollow air spaces, or cavities, located within the skull surrounding the nasal cavity. There are four pairs of paranasal sinuses: the maxillary, the frontal, the ethmoid, and the sphenoid (Figure 16-1). The sinuses are lined with a membrane of ciliated and nonciliated pseudostratified columnar epithelium that is continuous with the nasal cavities. The columnar cells are interspersed with mucous producing goblet cells that constantly produce a thin layer of mucinous fluid. The mucous is rich with immune cells, antibodies, and antibacterial proteins that serve in immune defense and air filtration by trapping and filtering particles such as dust, spores, viruses, and bacteria. The cilia beat in a coordinated, rhythmic wavelike pattern moving the mucous toward and through a small (1–4 mm diameter) opening, or ostium, into the nasopharynx where it is ultimately swallowed and dissolved by digestive acids. In addition to air filtration, the paranasal sinuses serve to humidify and warm inspired air and are also important in giving the voice resonance, defining tonal quality and voice amplification, as well as reducing the weight of the skull.[3]

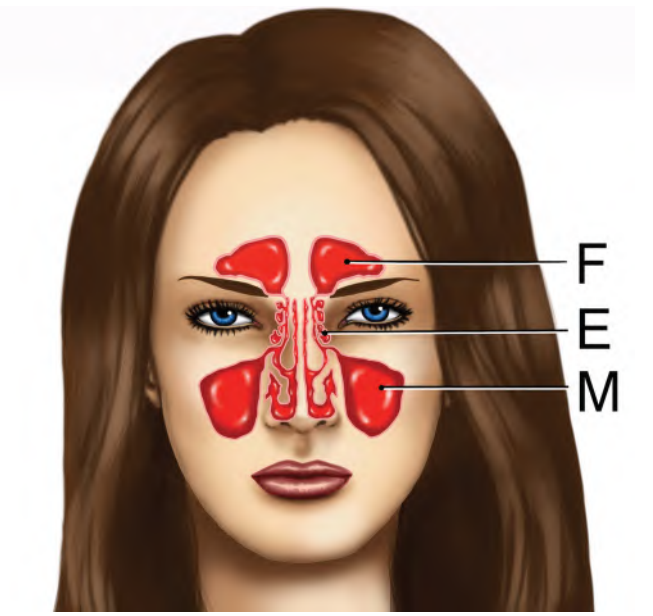


FIGURE 16-1 The paranasal sinuses: Maxillary (M), Ethmoid (E), and Frontal (F). The sphenoid sinuses are posterior to the ethmoids and are not shown.

The largest of the paranasal sinuses and most prone to infection are the maxillary sinuses. The maxillary sinus, also known as the antrum, is somewhat pyramidal in shape with the lateral wall of the nasal cavity forming the base and the apex extending into the zygoma. The floor of the maxillary sinus is the maxillary alveolar and palatine process, the roof is the floor of the orbit, and the posterior wall is adjacent to the infratemporal fossa. The maxillary sinus ostium is located near the top of the medial sinus wall and drains via the infundibulum into the semilunar hiatus of the middle meatus of the nasal cavity. Although the mucosa of the sinuses is rather insensitive to pain, the region of the ostia of the sinuses, also known as the osteomeatal complex, is the most sensitive of any of the nasal structures.[4] The osteomeatal region is highly innervated and is extremely sensitive when inflamed or stimulated during sinus-related pain episodes.[5]

RHINOSINUSITIS: ETIOLOGY, SYMPTOMS, DIAGNOSIS AND TREATMENT

The sinus ostia are considered to be the primary focal point for sinus disease, with obstruction being the major pathophysiologic factor in rhinosinusitis.[6] Sinus obstruction is commonly caused by inflammatory edema of the lining mucosa usually in response to a virus, allergen or bacteria. Ostial blockage can also occur from an anatomic obstruction such as a septal deviation or polyp, or a foreign body obstruction such as dried mucous. When a blockage occurs, mucous secretions, normally expelled through the ostia, accumulate within the sinus cavities where they begin to stagnate and thicken. The lack of sinus ventilation and stagnation of the secretions results in a lowered oxygen tension and a decrease in pH, providing an excellent environment for bacterial pathogens to colonize.[7] As the condition progresses, the cilia and epithelium become damaged, preventing fluid movement, and there is further mucosal thickening creating a more severe obstruction. The term *sinusitis* is used in the current literature interchangeably with *rhinosinusitis* due to the contiguous lining membrane of the paranasal and nasal cavities and its similar responses to therapy.[8]

Approximately one in five visits to primary care physicians in the United States result in a diagnosis of rhinosinusitis.[9] This condition is more prevalent in patients with congenital or acquired immunodeficiency, asthma, or allergic rhinitis. Symptoms may include nasal congestion, facial pain and pressure, purulent rhinorrhea, cough, headache, fever and fatigue, and pain in the maxillary posterior teeth.[10]

A diagnosis of rhinosinusitis is generally made through clinical examination and is based on the patient's history and physical signs and symptoms, with no single clinical finding being conclusively predictive of the condition.[11] In recurrent acute or chronic cases, endoscopy and CT scanning may be helpful in the diagnosis.[12] Coronally viewed sections of a sinus CT scan can reveal ostial patency or blockage, soft tissue changes, or any retained fluids that may be indicative of sinus disease (Figure 16-2). Although CT imaging has a high sensitivity, it has a low specificity for demonstrating rhinosinusitis. More than 40% of asymptomatic patients and 87% of patients with common colds show sinus abnormalities on a sinus CT.[13,14] It is also important to note that sinus inflammation and symptoms can occur in the absence of CT findings, thus the physician's overall clinical impression is superior to any single physical or radiological finding.[15]

Rhinosinusitis is classified as acute, subacute, recurrent acute, or chronic according to the temporal course of the disease.[10,12] Acute rhinosinusitis by definition lasts less than 4 weeks. Four or more episodes of acute rhinosinusitis within a 1-year period are defined as a recurrent acute rhinosinusitis. Subacute rhinosinusitis is a continuation of an acute form lasting between 4 and 12 weeks. Persistent signs and symptoms that continue unresolved for more than 12 weeks are classified as a chronic rhinosinusitis.[15] Acute

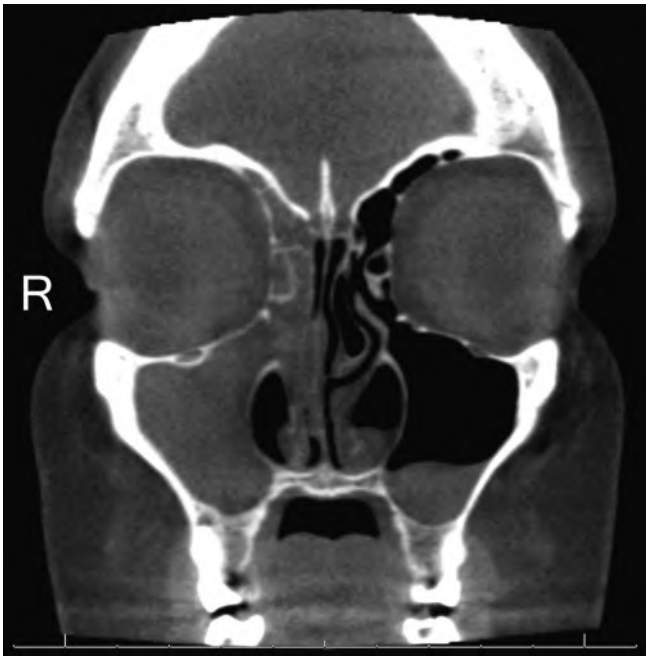


FIGURE 16-2 Rhinosinusitis. Coronal CT image shows fully obstructed right (R) maxillary, ethmoid, and frontal sinuses, as well as mucosal thickening on the floor of the left maxillary sinus.

rhinosinusitis usually has a sudden onset, usually with severe symptoms, but 40% to 50% of patients will recover spontaneously and the remainder will respond fairly well to antibiotic and/or adjunctive treatments. The predominant bacterial florae isolated in acute rhinosinusitis include *Streptococcus pneumoniae, Haemophilus influenzae, and Moraxella catarrhalis*. Amoxicillin or trimethoprim-sulfamethoxazole is often prescribed as the preferred first-line antibiotic treatment although the role of antibiotics in uncomplicated rhinosinusitis has been questioned because of concern regarding its overuse.[16–19] Sinusitis accounts for 21% of all antibiotics prescribed for adults in the United States, and 9% of all antibiotics prescribed for children.[20] The bacterial species isolated from acute rhinosinusitis has not changed in the last 60 years; however, antibiotic resistance in these organisms is a growing problem due to a significant increase in production of beta-lactamase. It has been estimated up to 40% of *H. influenza* strains and more than 90% of *M. Catarrhalis* isolates are resistant to amoxicillin.[21]

Chronic rhinosinusitis usually has less severe symptoms, yet is more difficult to resolve. Because symptoms are poorly localized and mild, this condition may be extremely difficult to recognize. The microbial flora of chronic rhinosinusitis tends to be a more mixed infection with *Staphlococcus aureus* and anaerobes being the more frequent isolates.[22] Narrow spectrum antibiotic regimens are typically ineffective against chronic rhinosinusitis. Treatment of chronic sinus disease is centered on reestablishing and then maintaining adequate drainage. Current treatment guidelines include the use of broader spectrum antibiotics such as amoxycillin-clavulanate, clindamycin, or the combination of metronidazole and

penicillin. Other adjunctive treatments include the use of steroid nasal sprays, decongestants, or saline irrigation. If antibiotic therapy and adjunctive treatments prove ineffective, then endoscopic sinus surgery is currently the recommended procedure to surgically remove any diseased tissue or blockage and open the natural ostium in attempt to reestablish drainage.[23]

REFERRAL OF MAXILLARY SINUS PAIN TO THE TEETH

The referral of pain from the maxillary sinus to the maxillary dentition is primarily due to the close anatomic relationship between the floor of the maxillary sinus and the roots of the posterior maxillary teeth. In a highly pneumatized antrum, the sinus floor will extend below and between the individual roots of the adjacent maxillary molars (Figure 16-3). In a study of adults, Eberhardt[24] found the average thickness of the bony partition between the apices of the maxillary second molar teeth and the antral musosa to be only 0.83 mm. Occasionally, no bony partition exists at all, with the roots apices and the sinus separated only by the mucosal tissue.[5] With such an intimate relationship, increased sinus pressure during sinusitis can produce the sensation of pain and pressure in the maxillary alveolar process and the corresponding teeth. Percussion of these proximate teeth produces acute tenderness due to the direct transfer of shock pressure to the inflamed sinus and sensitive ostium. For some patients, the most sensitive trigger for their sinus pain is often caused by manipulation of the maxillary posterior teeth, leading them to believe, with near certainty, that teeth are the source of their pain.

Heterotropic or neurologically referred pain can also be responsible for patients mistaking rhinosinusitis for odontalgia.[25] It has been demonstrated that stimulation of the maxillary sinus ostium can induce dental pain in the maxillary posterior teeth.[26–28] It has also been demonstrated that local topical anesthetic applied to the maxillary sinus ostium can relieve perceived dental pain in the case of referred sinus pain.[29] Anesthetic nerve blocks, however, may not be reliable in differentiating maxillary rhinosinusitis from odontalgia due to mutual dental and antral innervations. Sensory innervation of the maxillary sinus is via the maxillary division of the trigeminal nerve with branches coming from the anterior, middle, and posterior superior alveolar nerves, the infraorbital nerve, and the anterior palatine nerve.[4] These nerves travel along the floor of the maxillary sinus innervating the related teeth.[30]

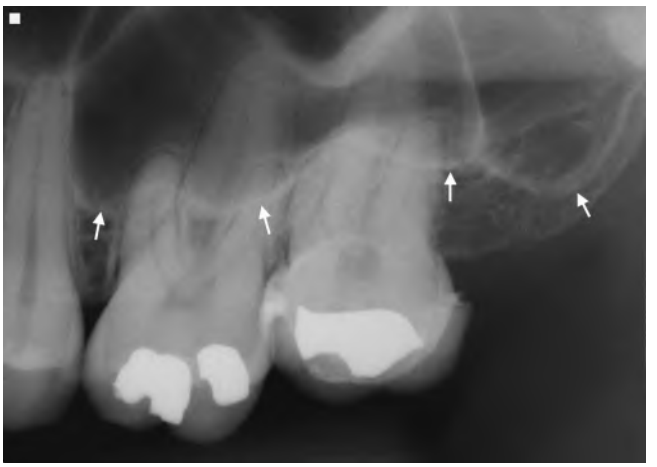


FIGURE 16-3 Periapical radiograph of a highly pneumatized left maxillary sinus in close approximation to the roots of the maxillary posterior teeth. The dense cortical bone defines the inferior border of the antrum (arrows).

DISTINGUISHING DIFFERENCES BETWEEN SYMPTOMS OF ODONTALGIA AND SINUS PAIN

Proper diagnosis always starts with a complete medical and dental history. An important question to ask the patient during the endodontic exam as well as on the medical history form is, "Do you have chronic allergies, a current cold, congestion, nasal drainage, or a history of sinus infections or problems?" Although many patients suffering from maxillary sinusitis may perceive dental pain, they usually complain of a dull aching pain that is difficult to pinpoint or localize to a single tooth. In addition, the patients often feel pressure in their cheeks and below their eyes that usually can be reproduced by external palpation of the cheeks. Erythema may also be evident. Positional changes can cause increased pain due to the movement of mucosal fluid over the sensitive sinus ostia as well as from increased intracranial pressure from blood flow.[31] The pain usually increases when patients are lying down, usually on one side more than the other, or if they bend over and place their head below their knees. In addition, patients with acute rhinosinusitis can have potentially severe symptoms, but then experience sudden and total relief if the sinus pressure is temporarily alleviated.

In contrast, pain of pulpal origin is more easily localized, unchanged with variations in position, and rarely intermittent in intensity. In cases of maxillary sinusitis, often all teeth proximate to the sinus floor test positive to percussion, whereas with odontalgia usually only the specific offending tooth is percussion-sensitive. The key diagnostic test for differentiating maxillary sinus etiology from pain of pulpal origin is thermal pulp sensibility response. If the teeth in question respond within normal limits to heat and cold stimuli when compared with other healthy teeth, then a pulpal etiology is effectively ruled out. Regardless of percussion sensitivity or the patient's complaint of spontaneous dental pain, endodontic treatment is not indicated as removal of a healthy pulp will surely not provide pain relief.

Although periapical radiographs are crucial to a thorough endodontic examination, the clinician must be careful not to establish the entire diagnosis on radiographic interpretation alone, particularly when evaluating the posterior maxillary region. The variation in bone density, presence of the maxillary

sinus and its bony septa as well as the zygomatic and palatal processes can impede important periradicular details necessary for accurate interpretation. Ambiguous radiographic conclusions are often the result. The increased availability of cone-beam computed tomography (CBCT) has vastly improved diagnostic ability in this region by eliminating superimposed structures and allowing for a much more accurate detection of periradicular lesions and sinus mucosal changes.[32,33]

MAXILLARY SINUSITIS OF DENTAL ORIGIN

The link between dental and sinus disease is widely recognized in both the dental and medical literature.[34–51] The pathological extension of dental disease into the maxillary sinus is a condition first referred to by Bauer in 1943 as *maxillary sinusitis of dental origin* (MSDO).[35] Bauer's classic study on cadavers showed histological evidence of the extension of periradicular infection causing destruction of the cortical bone, normally separating the teeth from the sinus floor, and pathological damage to the sinus tissues. Since then, other investigators have discovered that this is a prevalent and common occurrence with dental infections of posterior maxillary teeth. Abrahams et al.[44] have reported that infections of maxillary posterior teeth show maxillary sinus pathology in 60% of the cases, while Matilla[45] found sinus mucosal hyperplasia present in approximately 80% of teeth with periapical osteitis. Obayashi et al.[46] found maxillary sinus mucosal changes in 71.3% of patients with infections originating in the maxillary canines, premolars, and molars.

The increased availability of in-office CBCT has increased clinicians' recognition of MSDO. Low et al.[47] have shown that with the use of CBCT imaging, mucosal changes associated with dental infections was found with a prevalence of 77%, compared to only 19% using conventional radiographs. Lofthag-Hansen et al.[48] found that thickening of the mucous membrane of the maxillary sinus was identified more than four times as often with CBCT imaging than with periapical radiographs. If a sinus infection has a dental etiology, sinus healing cannot occur unless the source of the infection is removed. The dental literature provides numerous case reports of resolution of maxillary sinusitis following endodontic treatment or extraction.[49–54]

EFFECTS OF ENDODONTIC DISEASE ON THE MAXILLARY SINUS

Endodontic disease is characterized by pulpal microbial infection and the consequent inflammatory response in the adjacent periradicular tissues. Typically, throughout the dentition, the root apices are surrounded by alveolar bone, and thus classical assessment of endodontic inflammatory processes centers on the clinical, radiographic, and histopathologic effects of endodontic disease on osseous tissues. However, as previously discussed, in the maxillary posterior dentition the adjacent periapical tissues are typically respiratory mucosal, occasionally separated by only a very thin layer of cortical bone (Figure 16-4). Understanding the radiographic and clinical manifestations of periapical inflammation on the sinus cortical floor and respiratory tissues is critical to accurate endodontic diagnosis and treatment in this region. Endodontic pathosis

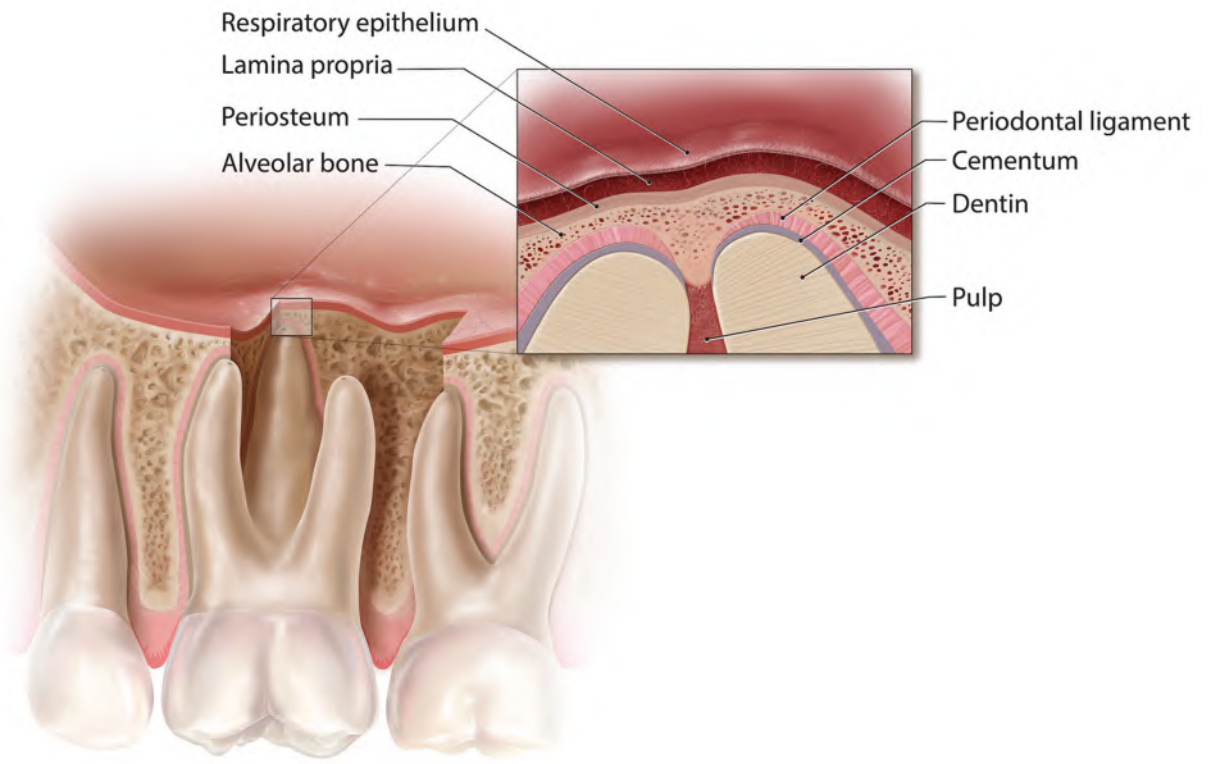


FIGURE 16-4 Illustration of the tissue layers separating the maxillary posterior root apices from the maxillary sinus in a healthy condition.

affects the maxillary sinus tissues in varying degrees: including periostitis, mucositis, and sinusitis.

PERIAPICAL OSTEOPERIOSTITIS

Inflammatory exudate from apical periodontitis, adjacent to the maxillary sinus, can penetrate the antral cortex and subsequently detach and lift the periosteum, displacing it into the sinus, and inducing periostitis (Figure 16-5). The periosteum is a dense connective tissue membrane, consisting of an outer fibrous layer and an inner cellular layer called the cambium. When irritated by inflammatory stimuli, the cambium layer contains progenitor cells that develop into osteoblasts and begin to produce new bone. This unique periosteal reaction, termed *periapical osteoperiostitis,* continues to deposit a thin layer of new bone on the inner periphery of the disease process as it expands, creating a cortical dome that produces a "halo" appearance radiographically (Figure 16-6).[55] In long-standing cases, the periosteum may continue displacing and remodeling the sinus cortical floor that, over time, can become thicker and expand deep into the maxillary sinus (Figure 16-7).

PERIAPICAL MUCOSITIS

If periradicular inflammation penetrates, or perforates, the antral cortex and associated periosteum, a localized mucosal tissue response can develop in the sinus. This localized inflammation and swelling of the sinus mucosal membrane presents as a radiopaque soft-tissue expansion called a *periapical mucositis* (Figures 16-8 and 16-9).[34,55] Mucositis can present overlying a periapical osteoperiostitis lesion (Figure 16-10), or can develop with no evident osseous changes, particularly in cases where no bony partition exists between the infected root apex and the sinus mucosa (Figure 16-11). Periapical inflammation has also been shown to be capable of affecting the sinus mucosa without perforation of the cortical bone. This is due to the extension of inflammatory mediators via bone marrow, blood vessels, and lymphatics.[35]

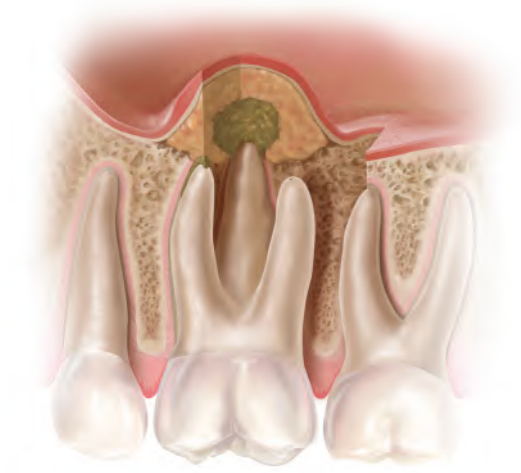
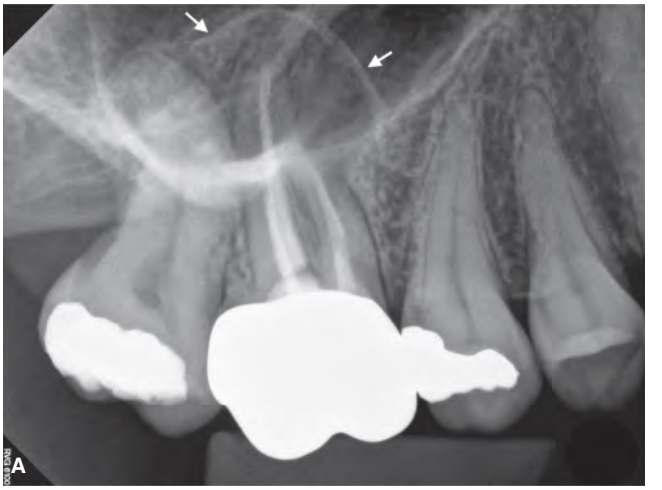


FIGURE 16-5 Illustration of periapical osteoperiostitis. Apical periodontitis displaces the periosteum into the floor of the maxillary sinus. The inflamed periosteum deposits a thin layer of newly formed cortical bone on the periphery as the lesion expands.



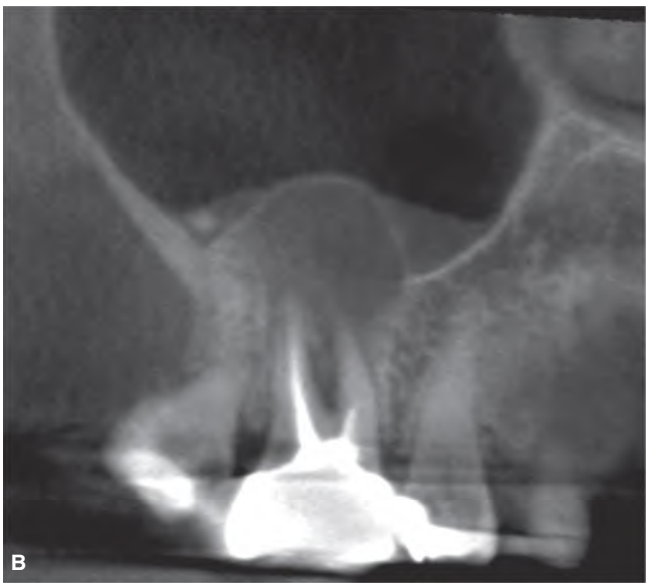
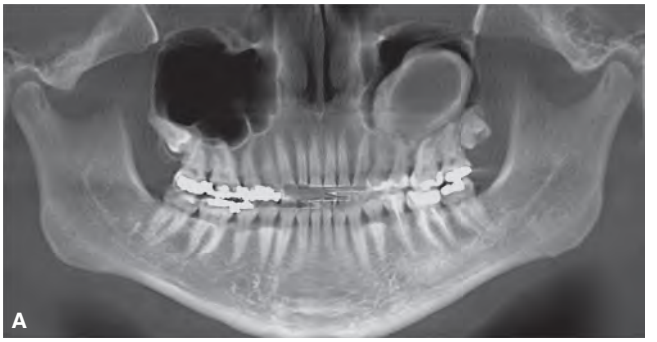


FIGURE 16-6 Periapical osteoperiostitis ("'halo" lesion). ***A***. Periapical radiograph of a failing root canal treatment of the maxillary right first molar. Apical periodontitis has displaced the periosteum into the sinus and new cortical bone has formed on the periphery of the disease process creating the appearance of a "halo" on the radiograph (arrows). ***B***. Sagittal CBCT image of the periapical osteoperiostitis lesion. Sinus mucosal edema, or fluid, is also evident on the sinus floor adjacent to the lesion.

It is important not to confuse a *periapical mucositis* with a *sinogenic mucosal hyperplasia*, a *mucocele*, or a *mucous retention cyst*. Mucosal thickening and mucous retention cysts are seen in approximately 10% of routine sinus CTs and are considered incidental findings. They can develop in all areas of the sinus and normally do not require treatment or surgical intervention by the ENT physician, unless



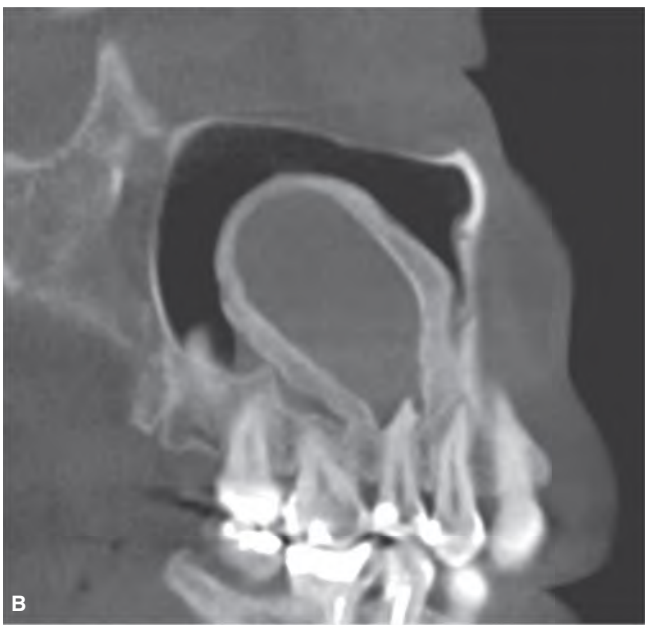
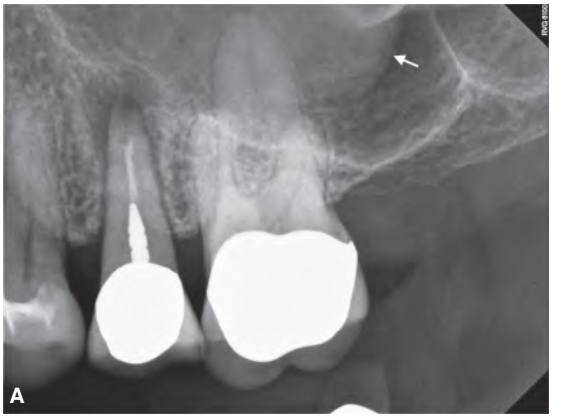


FIGURE 16-7 Advanced periapical osteoperiostitis. ***A***. Panographic CBCT image of a significant periapical osteoperiostitis caused by a long-standing, persistent apical periodontitis of the maxillary left second premolar. ***B***. Sagittal CT image showing the periosteal expansion deep into the maxillary sinus. The periosteal reaction has deposited a dense layer of cortical bone on the periphery of the disease process, separating the odontogenic infection from the sinus mucosal tissue. (Courtesy of Dr. Normand Aubre, Laval, Quebec, Canada.)



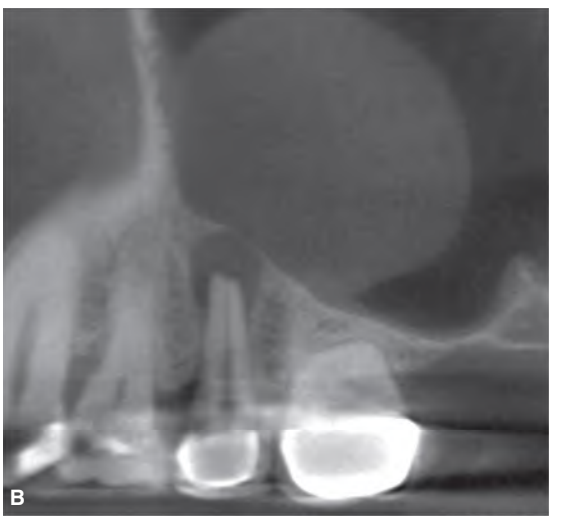


FIGURE 16-9 Periapical mucositis. ***A***. Periapical radiograph of a failing root canal treatment of the maxillary left second premolar. The distal extent of a periapical mucositis lesion is evident (arrow). ***B***. Sagittal CBCT image of the periapical mucositis lesion emanating from the apex the maxillary left second premolar. Mucositis may be indistinguishable, radiographically, from sinogenic mucoceles and mucous retention cysts, and requires an accurate endodontic diagnosis to confirm.

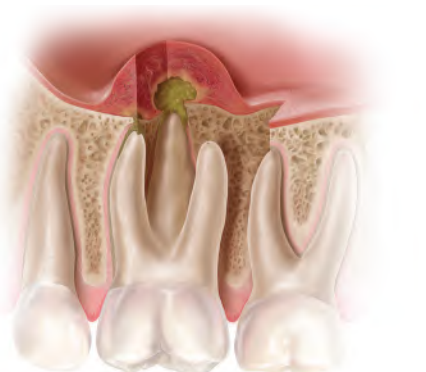


FIGURE 16-8 Illustration of periapical mucositis. Apical periodontitis perforates the antral cortex and associated periosteum causing a localized inflammatory edema in the adjacent sinus mucosa.

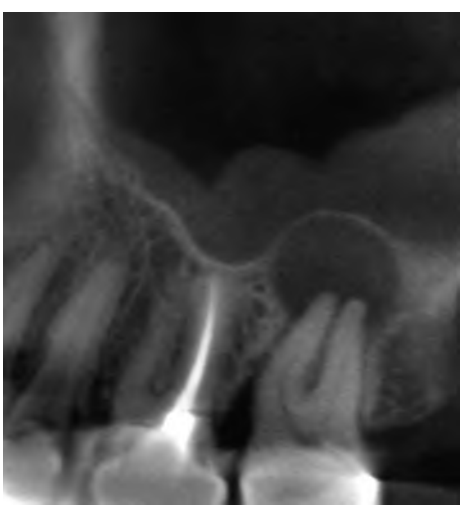


FIGURE 16-10 Periapical osteoperiostitis with mucositis. Sagittal CBCT image of a necrotic left maxillary second molar displaying osteoperiostitis ("halo" lesion) with associated mucosal edema.

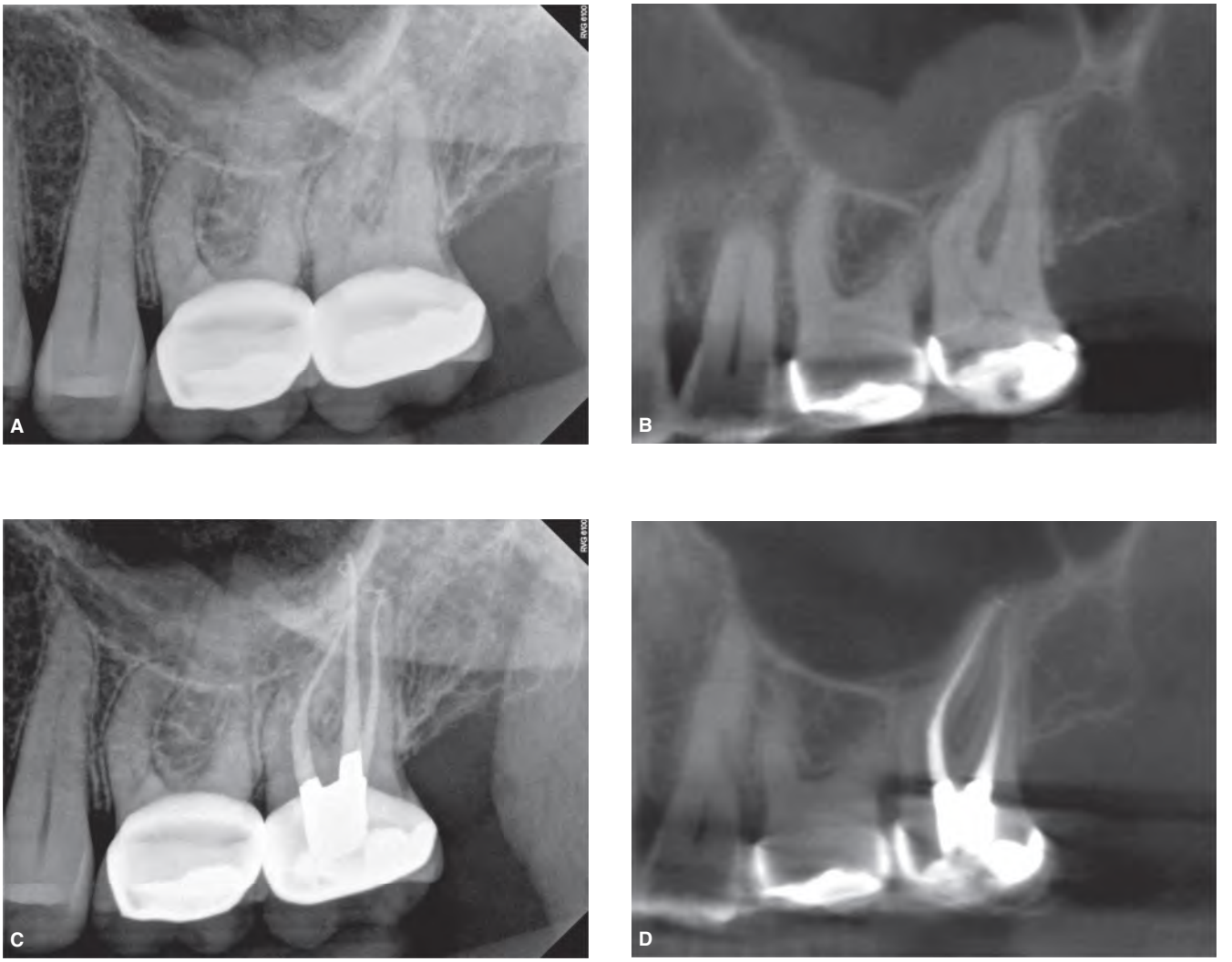


FIGURE 16-11 Periapical mucositis without osseous changes. ***A***. Periapical radiograph of the maxillary left posterior dentition. Clinical endodontic examination confirmed the second molar was necrotic. ***B***. Sagittal CBCT image of the maxillary left posterior region revealing mucosal edema in the floor of the left maxillary sinus, but without evidence of alveolar bone damage. ***C***. Periapical radiograph following endodontic treatment of the second molar. ***D***. 3-month postoperative sagittal CBCT image showing full resolution of the mucosal edema.

they are blocking the ostium.[10] If, however, sinus mucosal hyperplasia is seen adjacent to a dental root apex, on either routine periapical radiographs or CBCT imaging, it should raise the suspicion for a potential odontogenic etiology. Careful pulp testing is imperative in distinguishing a routine mucosal abnormality on the floor of the sinus from a periapical mucositis secondary to an odontogenic infection. Periapical mucositis should resolve within 3 to 6 months following endodontic treatment (Figure 16-11), however, long-term outcome studies of mucosal healing following endodontic treatment are lacking. Nurbaksh et al.[56] treated maxillary posterior teeth with apical periodontitis and found that 3 months after treatment there was full resolution of sinus mucositis in 30% of cases and partial resolution in an additional 30% of cases; 60% were considered healed after 6 months.

ODONTOGENIC RHINOSINUSITIS

Sinus inflammatory response to a dental infection may remain localized to the floor of the maxillary sinus in the form of an osteoperiostitis and/or mucositis, however, in progressed cases, a partial or full obstruction of the maxillary sinus can develop. This inflammatory process may extend even further, obstructing the nasal cavity, ethmoids, and frontal sinuses (Figure 16-12). This advanced pathological extension of dental disease, obstructing the maxillary sinus and potentially beyond, often termed *odontogenic rhinosinusitis,* is well documented. The reported frequency of odontogenic rhinosinusitis varies considerably, between 4.6% and 47% of all sinus infections.[57] This wide variation may be due to the difference in criteria and definitions as well as the inherent difficulty in establishing an exact causal relationship in maxillary

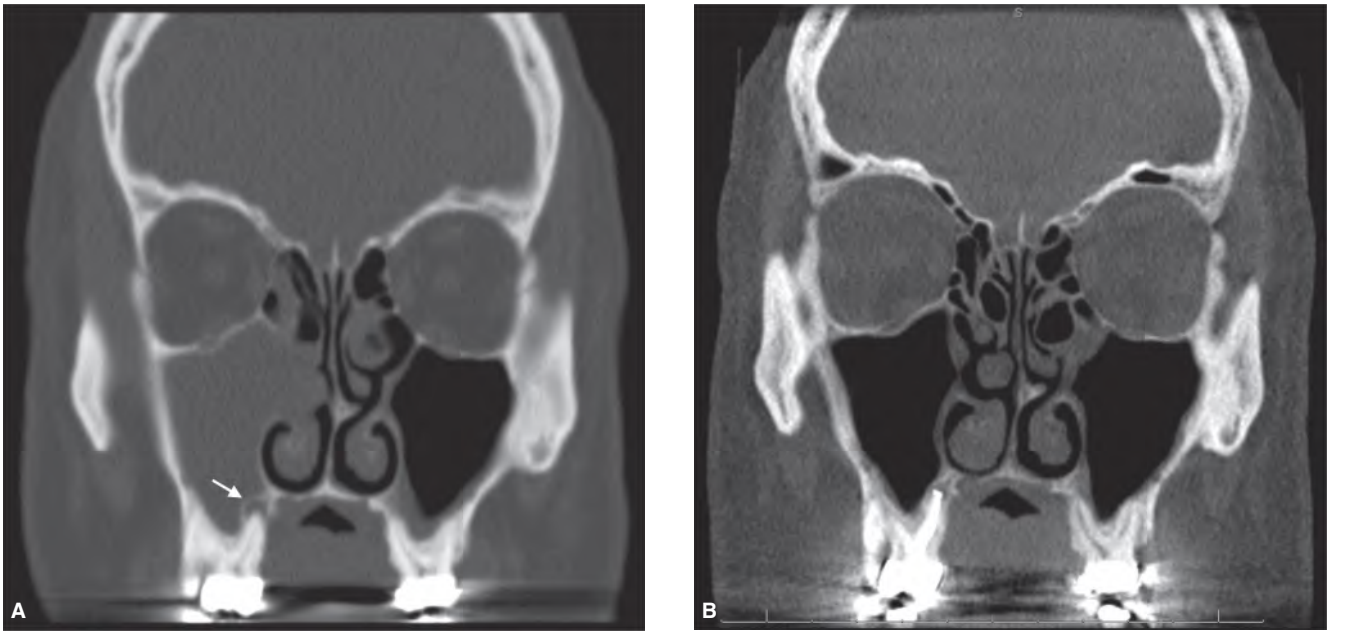


FIGURE 16-12 Odontogenic rhinosinusitis. ***A***. Coronal CT image of a fully obstructed right maxillary sinus. The patient had experienced recurrent right maxillary sinus infections and nasal obstruction for more than 4 years with no resolution, despite multiple antibiotic regimens and adjunctive sinus treatments. An associated periapical osteoperiostitis lesion is evident over the palatal root apex of the maxillary right first molar (arrow). ***B***. Three-month postoperative coronal CT image showing full resolution of the maxillary rhinosinusitis following endodontic treatment of the maxillary first molar. No other sinus treatment was performed nor antibiotics administered.

sinusitis.[58] It is often quoted and generally accepted that dental infections account for approximately 10% to 12% of all cases of maxillary sinusitis.[10,37,43,53,57] However, the primary source for this figure provides no epidemiological data to support it.[59] The actual incidence of odontogenic rhinosinusitis is likely much higher, particularly in chronic cases. Melen et al.[42], in a study of 198 patients with 244 cases of chronic bacterial maxillary sinusitis, found a dental etiology in 40.6% of the cases. Maillet et al.[60] reviewed 82 CBCT scans that had previously shown maxillary sinusitis for evidence of a dental pathology, and concluded that over 50% of these cases were of dental etiology. Bomeli et al.[61] found that the more severe the sinus disease, the more likely it was to be associated with dental pathology, such that up to 86% of severely affected maxillary sinuses were felt to have a dental etiology for the infection.

It is well documented that the predominant anaerobic bacterial isolates frequently found in chronic sinusitis are *Prevotella sp., Porphyromonas sp., Fusobacterium nucleatum, and Peptostreptococcus sp.*, the same bacterial species that are also found in endodontic infections.[22,62–64] It is unclear if the presence of these species is an indication of dental etiology or simply develops due to environmental changes within the sinus as the condition becomes more chronic.

One of the challenges in diagnosing odontogenic rhinosinusitis is that patients often have primary symptoms of maxillary sinusitis, yet are rarely able to localize pain to a specific tooth or sense any dental pain at all. These patients, typically, first seek care from their physicians or otolaryngologist who may diagnose and treat the condition as a primary sinus infection, since dental infections are easily overlooked during routine ENT examinations.[65] Otolaryngologists report that radiologists rarely comment on dental pathology on their sinus CT interpretation regardless of sinus pathology.[66] Regrettably, some patients have undergone multiple antibiotic regimens, and even one or more sinus surgeries, before a dental condition is finally diagnosed as the primary etiology (Figure 16-13).[67] This, unfortunately, occurs despite clear recommendations in the medical literature for careful dental examination and appropriate imaging in all patients with recurrent acute or chronic maxillary sinusitis.[42,61,64,65,68] The importance of properly diagnosing and treating odontogenic sinusitis is heightened with reports in the literature of dental infections spreading rapidly through the maxillary sinus causing orbital cellulitis, blindness, meningitis, subdural empyema, brain abscess, and life-threatening cavernous sinus thrombosis.[68–71] In spite of numerous medical publications emphasizing the need to rule out and treat any possible dental etiology for rhinosinusitis, to date none of the current medical guidelines for management of acute or chronic rhinosinusitis include recommendation for a clinical endodontic examination to rule out a possible odontogenic source. Longhini and Ferguson[72] reviewed 85 sinusitis guidelines published between 1998 and 2010 and found only 11 that mentioned a dental or odontogenic cause of maxillary sinusitis, and only three of these

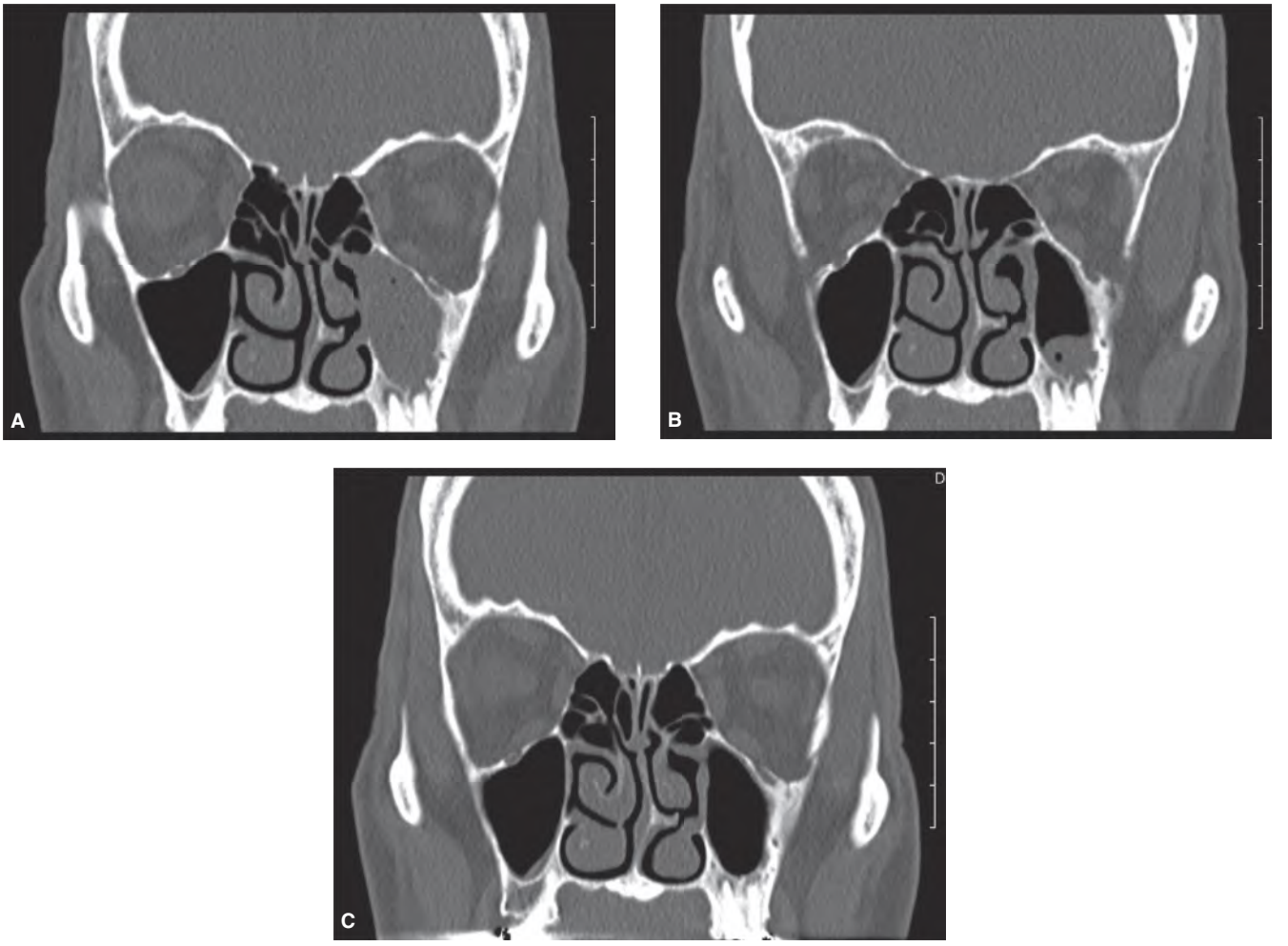


FIGURE 16-13 Odontogenic rhinosinusitis. ***A***. Coronal CT image of a fully obstructed left maxillary sinus. The patient had experienced severe maxillary sinus infections for over 7 years with no resolution, despite multiple antibiotic regimens. He was scheduled for endoscopic sinus surgery when a periapical osteoperiostitis lesion was noted over the apices of the maxillary left second molar. Endodontic treatment was recommended prior to proceeding with surgery. ***B***. 4-week postoperative coronal CT image showing significant improvement of the condition in the left maxillary sinus, with the air-fluid level less than 50%. ***C***. 10-week postoperative coronal CT image showing full resolution of the sinusitis in the left maxillary sinus. No other sinus treatment was performed nor antibiotics administered. All symptoms had resolved and the patient's sinus surgery was cancelled.

guidelines outlined diagnostic procedures for odontogenic sinusitis. The same authors also found that routine general dental examination, including dental radiographs, failed to diagnose odontogenic maxillary sinusitis in 86% of patients.[72] Melen et al.[42] similarly reported that 56 out of 99 (55%) odontogenic maxillary sinusitis cases are missed on routine dental examination and dental radiography. An improved communication between the ENT community and endodontic specialists is essential in providing improved patient care and resolving a greater number of cases of odontogenic sinusitis.

When diagnosing a possible dental etiology in a patient with rhinosinusitis, the clinician must confirm any pulpal necrosis and periapical disease, and carefully evaluate all previous endodontic treatment for possible failure in the suspected quadrant. In order for a periapical infection to occur, microorganisms must be present within the root canal system.[73] A vital pulp, whether healthy or inflamed, will not contribute to any significant periradicular or odontogenic sinus infection. Therefore, in order for odontogenic sinusitis to occur, the causative tooth must either have a necrotic pulp or a failing endodontic treatment. As discussed, periapical or panoramic radiographs may offer helpful, but limited, information regarding maxillary posterior periapical and sinus mucosal changes. Sinus CT or CBCT imaging is far more accurate and highly recommended whenever an odontogenic etiology for rhinosinusitis is suspected.[74] Other diagnostic findings that should raise the suspicion of odontogenic sinusitis are a history of unilateral maxillary sinus disease, particularly when associated with a patent sinus infundibulum or previously unsuccessful sinus surgery.[75]

If a sinus infection is determined to be caused by a periapical infection, endodontic therapy, or extraction, should provide full resolution of the disease. However, clinical and radiological follow-up is essential as concomitant management of the associated rhinosinusitis by an ENT and may be necessary in some advanced cases.[63]

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Chapter 17

Non-Odontogenic Toothache and Chronic Head and Neck Pain

Bernadette Jaeger

Pain is perfect misery, the worst of evils; and excessive, overturns all patience.

–John Milton, Paradise Lost

Patients with orofacial pain, or headache, present a true diagnostic and therapeutic challenge to the practitioner. Many patients with orofacial pain have suffered mistreatment due to misdiagnosis, and stand as proof of the complex nature of head and neck pain. Attending to patients who have been unable to obtain resolution of their pain complaint, despite extensive evaluation and treatment, requires a compassionate reappraisal and fresh approach. Often the most important first step toward resolution of a patient's orofacial pain is to seriously consider that the pain is not of odontogenic origin. Therefore, appropriate treatment often requires a referral to an orofacial pain dentist or other medical specialist.

WHAT IS PAIN?

The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage."[1] The statement "actual or potential tissue damage" emphasizes the subjective nature of pain that distinguishes it from the mere stimulation of nociceptors.

> "Many people report pain in the absence of tissue damage or any likely pathophysiological cause; usually this happens for psychological reasons. There is no way to distinguish their experience from that due to tissue damage, if we take the subjective report. If they regard their experience as pain and if they report it in the same ways as pain caused by tissue damage, it should be accepted as pain. This definition avoids tying pain to a stimulus. Activity induced in the nociceptor and nociceptive pathways by a noxious stimulus is not pain, that is always a psychological state, even though we may well appreciate that pain most often has a proximate physical cause."[1]

The IASP definition of pain makes the point that *pain is pain even if a nociceptive source is not readily identified*. Pain due to psychological causes is as real as any pain associated with actual nociception and should be treated as such. Pain is what the patient says it is.

An individual's reaction to this perceived sensation can be considered as "pain behavior." This is any behavior, physical or emotional, that follows pain perception. Culture or environment often influences these behaviors, in particular behaviors associated with chronic pain. The suffering or emotional toll the pain has on any given individual is personal and must be quantified, evaluated, and treated if the patient's pain is to be understood.

Pain may also be classified into two broad categories; protective or maladaptive. Protective pain causes the sufferer to withdraw from a potential injury; for example, pulling the finger out of a flame. It protects the organism from further injury reflexively. This type of short-lasting pain, secondary to a local injury with minimal damage, is what is known as nociceptive pain.[2] The inflammation associated with an injury such as bone fracture or even sunburn sensitizes the nociceptive and non-nociceptive nerve endings in the area resulting in signaling in responses to otherwise non-noxious stimuli. This inflammatory pain protects a patient against further trauma. Once healed, inflammation resolves and pain subsides. Pain that continues after the injury or other cause of pain has resolved is known as maladaptive pain. This type of pain is complex and involves processes as such peripheral nociceptor activation and sensitization, "central sensitization" and changes in peripheral processes such that now a non-nociceptive stimuli evokes a pain sensation. Maladaptive pain typically takes a chronic course and no longer serves any protective function.

The fact that pain is difficult to define, quantify, and understand is reflected in the numerous ways in which it has been described throughout history. Modern definitions of pain began with Descartes who defined pain as the stimulation of, or the pulling on delicate threads that led to the brain resulting in pain.[2] This idea of a peripheral receptor resulting in a central pain response was accepted for nearly 500 years until it was expanded upon by more current discoveries in neuroscience.

Fields[3] defined pain as "an unpleasant sensation that is perceived as arising from a specific region of the body and is commonly produced by processes that damage or are capable of damaging bodily tissue." He emphasized the need to be able to localize the painful source in order to distinguish it from psychological pain and suffering, for example, the "pain" of a broken heart.

To understand the nuanced and complex topic of pain better, particularly in the orofacial region, the reader is referred to comprehensive literature in the field.[4]

NEUROPHYSIOLOGY OF PAIN

Acute Pain Pathways

The body has specialized neurons that respond only to a noxious or potentially noxious stimulus. These neurons are referred to as primary afferent nociceptors and are very complex in structure. Once stimulated, these primary afferent neurons synapse with neurons known as second-order pain transmission neurons in the substantia gelatinosa of the dorsal horn of the spinal cord or, in the head and neck region, the trigeminal nucleus caudalis. From here the signals are transmitted along specialized pathways to the thalamus and brain stem respectively (Figure 17-1) and then to the somatosensory cortex where the pain location and intensity are determined. Projections to the anterior cingulate gyrus and insular cortex produce emotional responses to the stimulus. It is important to note that there is no single brain area that processes pain.[5]

Arachidonic acid is released during tissue injury and is processed by two different enzyme systems into prostaglandins and leukotriene B4 respectively (Figure 17-2). Whereas prostaglandins stimulate the primary afferent nociceptor directly, the leukotrienes contribute indirectly by causing polymorphonuclear neutrophil leukocytes to release another chemical that, in turn, stimulates the nociceptor. Bradykinin contributes further by causing the *sympathetic* nerve terminal to release a prostaglandin that also stimulates the nociceptor.[6] Additionally, in an area of injury or inflammation, the *sympathetic nerve terminal* will release yet another prostaglandin in response to its own neurotransmitter, *norepinephrine*. The presence of such an ongoing inflammatory state causes physiological sensitization of the primary afferent nociceptors.[6] Sensitized nociceptors display ongoing discharge, a lowered activation threshold to *normally nonpainful stimuli* (*allodynia*), and an *exaggerated response* to noxious stimuli (*primary hyperalgesia*).[7]

Activation of cutaneous C fibers stimulates synthesis within their cell bodies, of the neuropeptides *substance P* and *CGRP*. These neuropeptides are then transported along axon branches back to the periphery where they induce further plasma extravasation and increase inflammation. The release of these algogenic substances at the peripheral axon injury site produces the flare (redness) commonly seen around the site of injury and is referred to as *neurogenic inflammation*,[8–11] or the *axon reflex*[7] (Figure 17-3).

The primary afferent nociceptor synapses with a second-order pain transmission neuron in the dorsal horn of the spinal cord where a new action potential heads toward higher brain structures (Figure 17-2). It is at this point that repeated or intense pain fiber activation facilitates and

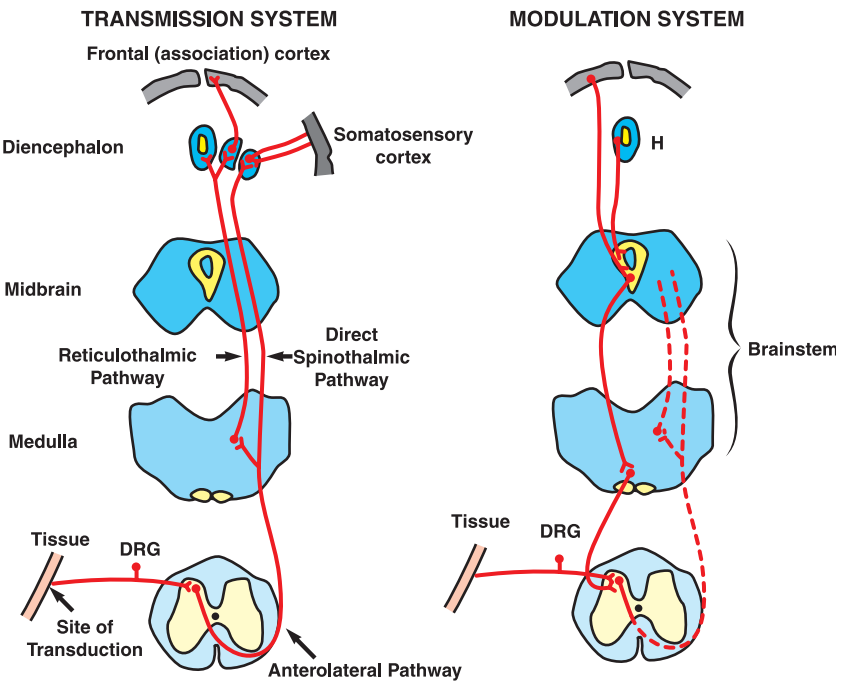

FIGURE 17-1 A diagrammatic outline of major neural structures relevant to pain. Perception of nociception begins with transduction (lower left), in which a noxious stimulus produces nerve impulses in the primary afferent nociceptor. These impulses are conducted to the spinal cord, where primary afferent nociceptors synapse with second-order pain transmission neurons that relay the message to the somatosensory cortex either directly via the spinothalamic tract or indirectly via the reticular formation and the spinoreticulothalamic pathway. From the thalamus, the message is relayed to the cerebral cortex and the hypothalamus (H). The pain modulation system depends on outflow from the cortex through the midbrain and medulla to the dorsal horn of the spinal cord, where it inhibits pain transmission cells, thereby reducing the intensity of the nociceptive signal. (Reproduced with permission from Committee on Pain, Disability, and Chronic Illness Behavior. Institute of Medicine. *Pain and Disability. Clinical, Behavioral, and Public Policy Perspectives*. M. Osterweis, A. Kleinman and D. Mechanic, eds. National Academy Press. Washington, DC, 1987.)

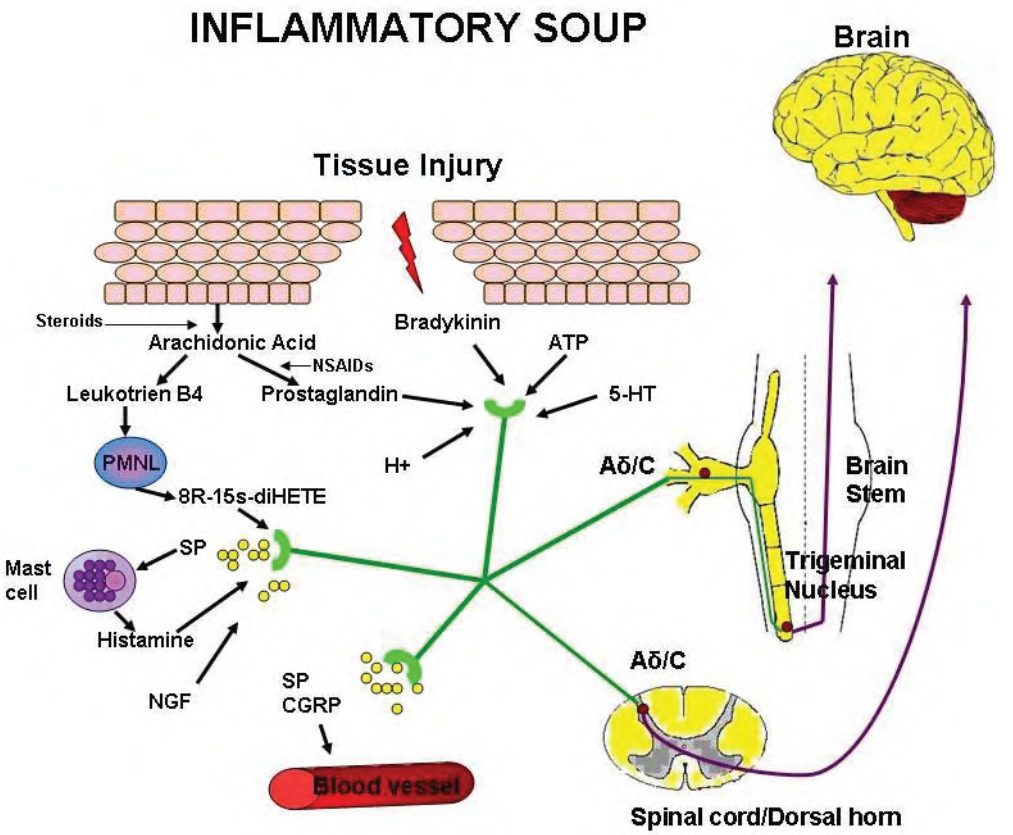

FIGURE 17-2 Arachidonic acid is produced by membrane lipids and is released with tissue injury. It is converted to prostaglandins and leukotriene B4 by two separate pathways. The cyclooxygenase pathway can be blocked by using nonsteroidal anti-inflammatory drugs (NSAIDs). Steroids prevent the synthesis of arachidonic acid altogether, thus inhibiting both pathways of prostaglandin production. Prostaglandins act directly on the primary afferent nociceptors to lower the firing threshold and therefore cause "sensitization." Leukotriene B4 causes polymorphonuclear neutrophil leukocytes to produce another leukotriene that, in turn, acts on the primary afferent nociceptor to cause sensitization.

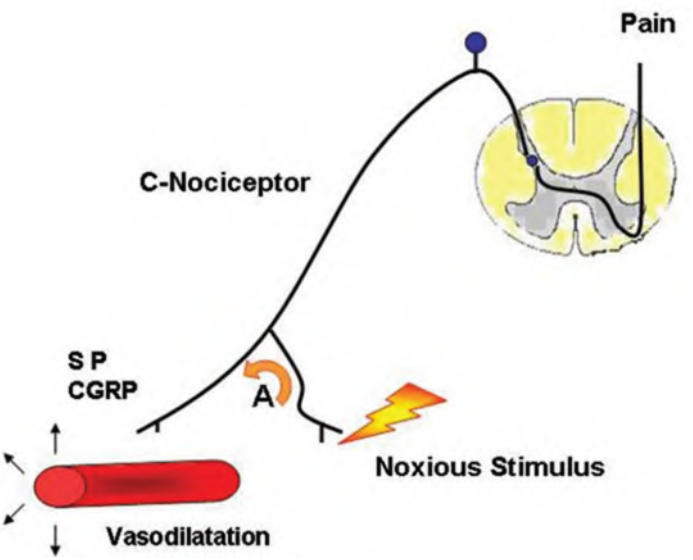

FIGURE 17-3 The axon reflex. Activation of cutaneous nociceptive C fibers elicits impulses that are conveyed centrally to induce pain and antidromically via axon branches (A). The antidromically excited peripheral C-fiber terminals release vasoactive substances, for example, calcitonin gene-related peptide (CGRP) and substance P (SP), causing cutaneous vasodilation, that produces the flare that develops around the site of noxious stimulation. (Reproduced with permission from Pain. Field HL, et al., *Neurobiol Dis*. 1998.[7])

increases responsiveness in the second-order pain transmission neuron, enhancing synaptic transmission and depressing inhibition in the dorsal horn.[12] This process further induces physiological, neurochemical, anatomical, and genetic changes,[10,13] resulting in *central sensitization*.[7,14]

The response of these spinal cord dorsal horn neurons increases progressively and is enhanced with repeated identical noxious cutaneous input from the periphery, a process called "windup."[15–17] In addition, the size of the receptive field of the second-order pain transmission neuron increases.[18] The subjective correlate of windup is "temporal summation," for which a slowly repeated noxious stimulus of the same intensity is associated with a progressive increase in perceived discomfort or pain.[7,19]

In addition, with central sensitization, stimulation of Aβ fibers (large-diameter low-threshold mechanoreceptors that normally respond only to painless tactile stimuli) will also establish connections with second-order nociceptive dorsal horn neurons, producing what is called a "secondary mechanical hyperalgesia"[7,20,21] (Figure 17-4). Aβ, when injured, gain the capacity to produce pain transmission neuropeptides not seen in unaffected Aβ fibers.[22] These plastic changes result in nonpainful stimuli causing pain. This can be observed in the hypersensitivity and allodynia present during a migraine headache or the pain to light touch of the skin after sunburn.[2] as well as in the ongoing persistent aching burning pain present in centralized trigeminal neuropathies such as painful posttraumatic trigeminal neuropathy.[23]

Modulation refers to mechanisms by which the transmission of noxious information to the brain is modified. Numerous *descending inhibitory* systems that originate supraspinally and influence spinal nociceptive transmission exist.[24] Activity in the pain modulation system results in decreased activity in the pain transmission pathway in response to noxious stimulation.

An endogenous opioid system for pain modulation also exists.[25] *Endogenous opioid peptides* are naturally occurring pain-dampening neurotransmitters and neuromodulators that are *implicated in pain suppression* and modulation because they are present in large quantities in the areas of the brain associated with these activities (subnucleus caudalis and the substantia gelatinosa of the spinal cord).[25–27] The presence of these natural opioid receptors for endogenous opiates is why morphine-like drugs work. However, evidence suggests that exogenous opioids, when used extensively, can paradoxically induce hyperalgesia.[28]

Finally, glial cells, considered the macrophages of the central nervous system, also contribute to central sensitization

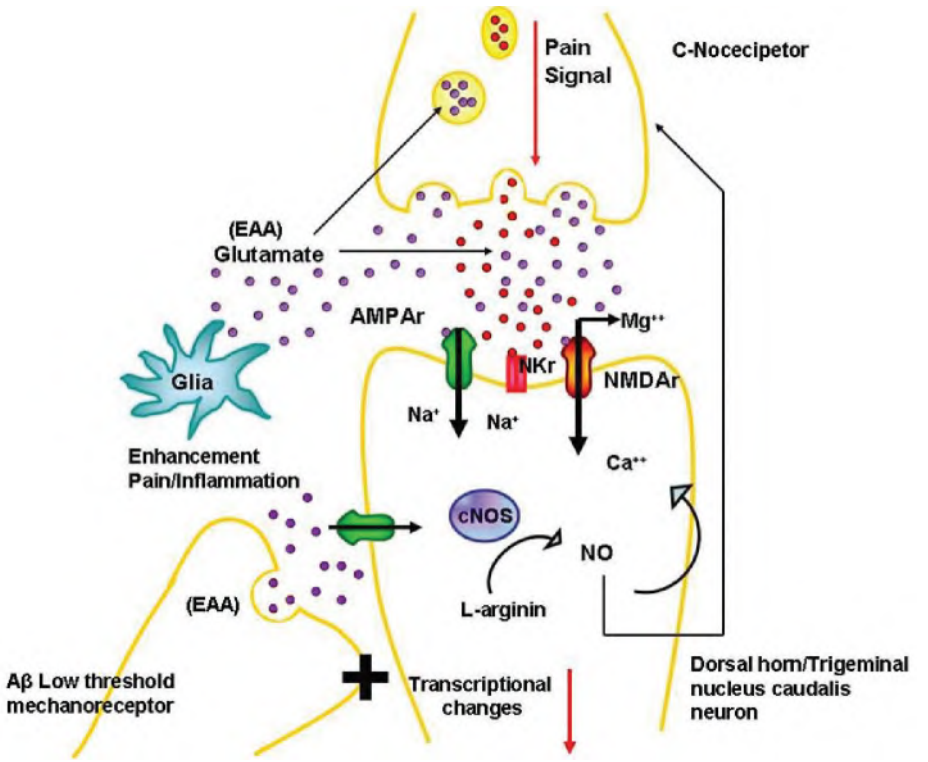

FIGURE 17-4 Central sensitization and allodynia. Input from C-nociceptors enhances the response of dorsal horn pain-signaling neurons to subsequent afferent inputs (central sensitization). This involves neuropeptides such as substance P (SP) acting at neurokinin receptors (NKR) and excitatory amino acids (EAA) acting at both the AMPA/KA and *N*-methyl-D-aspartate (NMDA) receptors, triggering secondary nitric oxide (NO) mechanisms. Large diameter low-threshold mechanoreceptive primary afferents (A fibers) respond maximally to innocuous tactile stimuli and normally produce tactile sensation. When central sensitization is present, A fibers become capable of activating central nervous system (CNS) pain-signaling neurons (+), leading to touch-evoked pain (allodynia). (Modified with permission from Fields HL et al., *Neurobiol Dis*. 1998.[7])

after physical injury to a peripheral nerve when they leave their usual homogenous distribution in the gray matter of the spinal cord to accumulate in the superficial dorsal horn of the injured peripheral nerves and even surround cell bodies of ventral horn motor neurons.[6] Glia release a variety of molecules that enhance central sensitization and persistent pain.[29]

The final step in the subjective experience of pain is *perception*. How and where the brain perceives pain is still under investigation. Part of the difficulty lies in the fact that the pain experience has at least two components: the sensory-discriminatory dimension and the affective (emotional) dimension. The affective dimension of pain is made up of feelings of expectations, unpleasantness, and emotions associated with future implications related to the pain.[30,31] Neuroimaging studies have demonstrated the involvement of the thalamus and multiple cortical areas in the perception of pain.[32]

Of significance is the fact that, with high levels of modulation or with damage in the pain transmission system, it is possible to have nociception without pain perception. Conversely, with certain types of damage to the nervous system, there may be an overreaction to pain stimuli or pain perception without nociception.[2]

Referred Pain

Pain arising from deep tissues, muscles, ligaments, joints, and viscera, is often perceived at a site distant from the actual nociceptive source. For example, cardiac problems may be perceived in the left arm or the jaw, and diaphragmatic pain is often perceived in the shoulder or the neck.[33,34] Cutaneous and mucogingival pain is sharp, burning, and clearly localized; referred pain from musculoskeletal and visceral sources is typically described as deep, dull, aching, and more diffuse. Referred pain is dependent on the primary pain source and will cease if this source is eliminated.

Referred pain presents a diagnostic dilemma. Unrecognized, it may result in ineffective treatments that, especially if invasive or irreversible, subject the patient to unnecessary risks, expense, and complications. When nothing else works, a misdiagnosis of psychogenic pain may be made.

The mechanism of referred pain is still enigmatic. The two most popular theories are convergence-projection and convergence-facilitation.

1. *Convergence-projection theory*. This is the most popular theory and was proposed by Ruch in 1965.[35] Primary afferent nociceptors from both visceral and cutaneous neurons often converge onto the same second-order pain transmission neuron in the spinal cord,[6,36] and convergence has been well documented in the trigeminal brain stem nuclear complex as well.[37] The trigeminal spinal tract nucleus also receives converging input from cranial nerves VII, IX, X, and the upper cervical nerves.[38] The brain, is programmed to interpret noxious signals as arising from cutaneous rather than visceral structures. Pain is perceived as arising from the most common source: the regions subserved by the cutaneous afferent fibers (Figure 17-5).

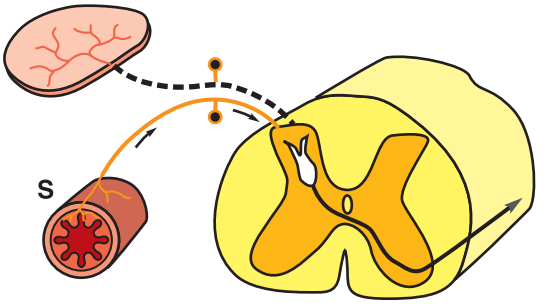

FIGURE 17-5 The convergence-projection hypothesis of referred pain. According to this hypothesis, visceral afferent nociceptors (S) converge on the same pain-projection neurons as the afferents from the somatic structures in which the pain is perceived. The brain has no way of knowing the actual source and mistakenly "projects" the sensation to the somatic structure. (Reproduced with permission from Fields HL, McGraw-Hill; 1987.[3])

2. *Convergence-facilitation theory*. This theory dates back to MacKenzie in 1920.[39] It is similar to the convergence-projection theory, except that the nociceptive input from the deeper structures causes the resting activity of the second-order pain transmission neuron in the spinal cord to increase or become "facilitated." The resting activity is normally created by impulses from the cutaneous afferents. "Facilitation" from the deeper nociceptive impulses causes a perception of pain associated with the normal, resting background activity. This theory tries to incorporate the clinical observation that blocking sensory input from the reference area, with either a local anesthetic or cold, can sometimes reduce the perceived pain.

Neither theory is considered complete, as changes in the reference zone and sympathetic involvement are not explained.[2] Spinal and supraspinal mechanisms may be involved.[2]

Trigeminal System

An appreciation of the arrangement of the trigeminal nociceptive system provides some insight into the interesting pain and referral patterns that are encountered in the head and neck region. The primary afferent nociceptors of the fifth cranial nerve synapse in the trigeminal nucleus caudalis of the brain stem.[37] The nucleus caudalis is the caudal portion of the trigeminal spinal tract nucleus and corresponds to the substantia gelatinosa of the rest of the spinal dorsal horn (Figure 17-6).

Of significance is the arrangement of the trigeminal nerve fibers within this nucleus and the fact that the nucleus descends as low as the third and fourth cervical vertebrae (C3-4) in the spinal cord. Fibers from all three trigeminal branches are found at all levels of the nucleus, arranged with the mandibular division highest and the ophthalmic division lowest.[40] In addition, they are arranged in such a manner that fibers closest to the midline of the face synapse in the most cephalad portion of the

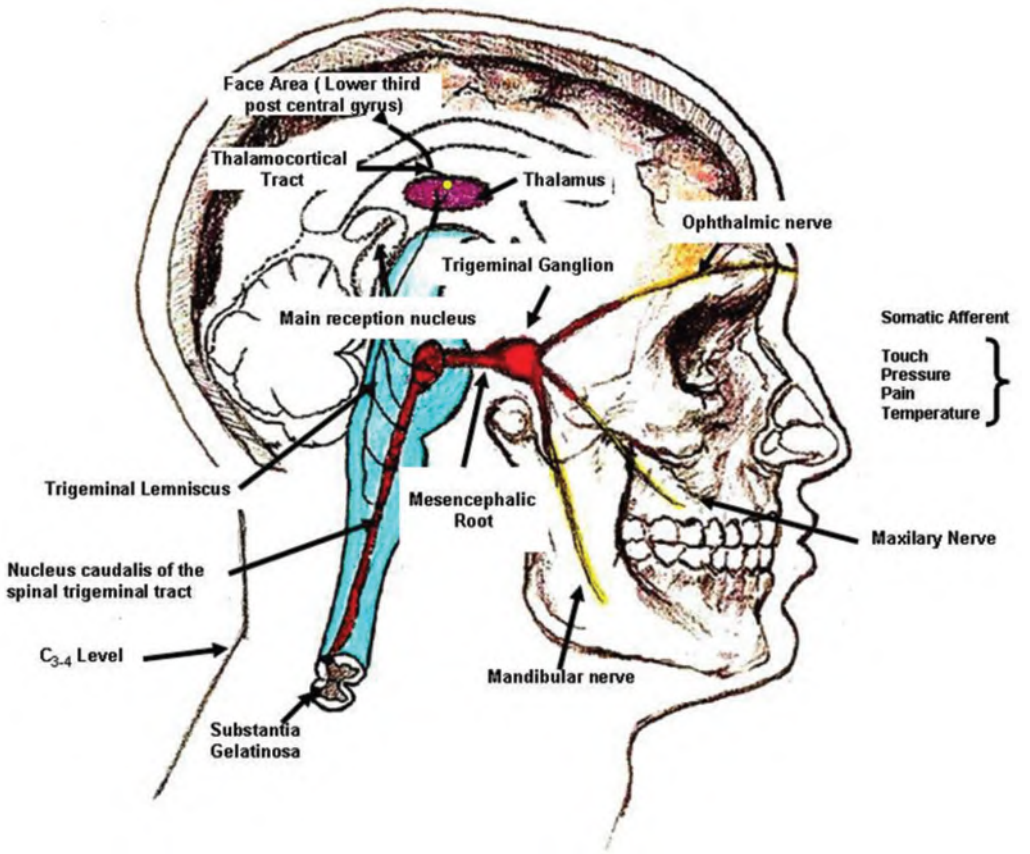

FIGURE 17-6 Primary afferent nociceptive fibers of the trigeminal nerve (cranial nerve V) synapse in the nucleus caudalis of the spinal trigeminal tract. The nucleus caudalis descends as low as C3-4 in the spinal cord. Many nociceptors from deep cervical structures synapse on the same second-order pain transmission neurons as the trigeminal nerve fibers do. This may explain why cervical pain disorders are often perceived as facial pain or headache.

tract. The more lateral the origin of the fibers on the face, the more caudal the synapse in the nucleus (Figure 17-7) Understanding this "laminated" arrangement helps to explain why a maxillary molar toothache may be perceived as pain in a mandibular molar on the same side (referred pain) but not in an incisor. Similarly, pain perceived in the ear may actually be due to (or referred from) an infected third molar.

Because the trigeminal nucleus descends to the C3-4 level in the spinal cord, primary afferent nociceptors from deep cervical structures synapse on the same second-order pain transmission neurons that subserve the fifth cranial nerve.[37] This convergence of primary afferent nociceptors from the trigeminal region and the cervical region provides a basis for understanding why *cervical pain* disorders may be perceived as pain in the head and face, particularly in the *forehead and temple*—the lateral ophthalmic trigeminal fibers synapse most caudally (Figure 17-7).

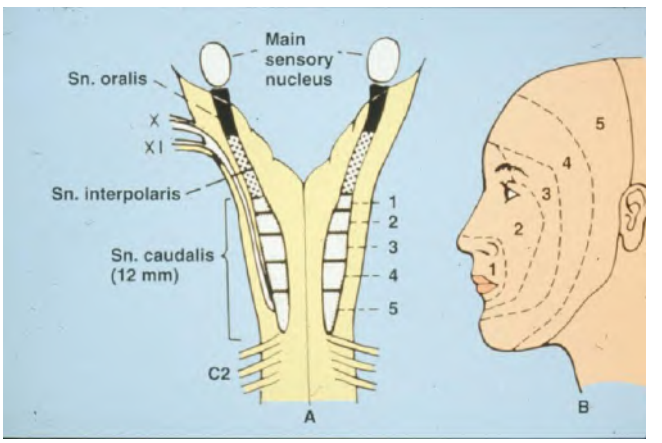

FIGURE 17-7 The arrangement of the trigeminal nociceptive fibers of the spinal trigeminal tract is significant. Fibers from all three trigeminal branches are found at all levels of the nucleus, arranged with the mandibular division highest and the ophthalmic division lowest. Fibers closest to the midline of the face synapse in the most cephalad portion of the tract. The more lateral the origin of the fibers on the face, the more caudal the synapse in the nucleus. (Modified with permission from Kunc Z, Georg Thieme Verlag, 1970.[40])

Psychological Factors That Modify Pain

Psychological and social factors are inextricably linked with the perception of pain and illness.[41] The traditional dualistic view of mind and body as separate, independently

functioning entities has been successfully challenged with the revelation of brain function during pain functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) scan imaging illustrate that various locations in the brain, not associated with pain perception, demonstrate increased activity in response to pain. Conversely, these same regions in the brain show increased activity when the subject is presented with a painful scenario or asked to view a disturbing photograph. Areas of the brain responsible for cognition, motivation, and emotions are activated during the experience of pain and play a significant role in pain modulation.[5]

In recognition of the need for a new approach to illness and pain, the biopsychosocial model for disease and illness has evolved. In this model, *disease* is defined as a "definite pathologic process with a characteristic set of signs and symptoms."[42] *Illness* refers to the subjective experience of that disease or the belief that disease is present.[43] An explosion of research in chronic pain has documented the complex interrelationship between genetics, neurophysiology, pain, and emotion.[44] For example, emotional distress may predispose a person to experience pain, amplify or inhibit the intensity of the pain, perpetuate the pain, or be a result of the pain. Anxiety and depression have long been studied with respect to their complexly intertwined relationship with chronic pain, but anger also plays an interesting role. These emotional concomitants to chronic pain are important to appreciate because they affect treatment motivation and compliance to treatment recommendations. For successful treatment, clinicians treating patients with pain, acute or chronic, must pay attention to their emotional/psychological states, as well as the somatically identified nociceptive cause.

Behavioral Factors

Suffering and pain are communicated through actions such as moaning, limping, grimacing, guarding, pill taking, or visiting the doctor. These actions are termed *pain behaviors*. Pain behaviors follow basic learning principles and tend to increase unconsciously if they are rewarded by positive consequences. Positive consequences may include attention from loved ones and/or the health care system and avoidance of various aversive tasks and responsibilities. Since chronic pain conditions, by definition, last a long time, they provide an opportunity for unconscious learning to occur. The pain behaviors observed in a patient with chronic pain may be the cumulative result of intermittent *positive reinforcement* over months or years. In addition, *well behaviors* are often totally ignored and non-reinforced, causing them to decrease. Thus, even when the nociceptive source has diminished or healed, pain perception and attendant disability may be maintained through *learned pain behaviors* along with physical, cognitive, and affective factors.[44]

MAKING A PAIN DIAGNOSIS

Diagnosing orofacial disease and headaches follows the same principles as any medical diagnosis. Of primary importance is a careful and exhaustive history. This alone will often point directly to a specific diagnosis or at least reveal a diagnostic category. The history is followed by a physical examination that should help to confirm or rule out the initial diagnostic impression. If necessary, further diagnostic studies such as pulp testing, nerve blocks, imaging, or blood tests may be ordered at this time. These may help rule out serious disorders and provide information complementing the history and physical examination. Finally, if doubt concerning the diagnosis persists, other medical specialists and health care providers may provide valuable consultation.

Classification

In evaluating orofacial pains and headaches, an easy-to-use, practical, and clinical classification of these pains will facilitate diagnosis (Table 17-1). In developing such a classification, it is important to remember that local disease of the extracranial or intracranial structures must be ruled out first. Most dentists are well trained to evaluate a patient for acute pathologic disease. Referred pains from disease in structures distant from the oral cavity must also be considered, although these disorders may be less well known to the dentist and will be reviewed.

There are other disorders, however, causing pain in the head and neck region that for the most part cannot be attributed to any obvious diseases. These disorders are less well appreciated and, for ease of clinical use, are best classified according to the apparent tissue origin of the pain (Table 17-1). For an exhaustive classification of headaches and facial pain that includes very specific diagnostic criteria, the reader is referred to the International Headache Society's Classification of Headache disorders.[45]

TABLE 17-1 Practical Clinical Classification of Craniofacial Pain

General classification	"Origin" of pain	Basic quality of pain
Local disease of extracranial structures	Craniofacial organs	Any
Intracranial disease	Brain and related structures	Any
Referred pain from remote sites	Distant structures	Aching, pressing
Neurovascular	Blood vessels	Throbbing
Neuropathic	Sensory nervous system	Shooting, sharp, burning
"Causalgic"	Sympathetic nervous system	Burning
Muscular	Muscles	Deep aching, tight
Idiopathic	Etiology as yet unknown	Deep aching, burning

The primary distinguishing feature of these practical clinical diagnostic categories is the quality of the pain. For example, neuro-*vascular* pain, such as migraine, generally has a throbbing, pulsing, or pounding quality; *neuropathic* pain, for example, trigeminal neuralgia or postherpetic neuropathy, is usually described with sharp, shooting, or burning qualities and is restricted to the peripheral distribution of the affected nerve branch; *muscle* pain is usually deep, steady, and aching or produces a sensation of tightness or pressure.

In contrast, *extracranial* or *intracranial* diseases may present with any quality of pain: a toothache may throb, a cracked tooth has sharp pain, a tumor in the mandibular canal may cause burning pain.

Once organic disease has been ruled out, a preliminary diagnostic category can be chosen based on the *quality* of the pain. Therefore, the first question to ask after location is the quality of the pain. This will help guide further questioning. For example, a constant dull ache in front of the ear along with a sharp shooting pain from the ear to the chin describes two qualities of pain. One points to a musculoskeletal diagnosis for the ear pain and the other to a paroxysmal neuralgia for the shooting pain. Further history, examination, and diagnostic tests will help establish a final or differential diagnosis.

History

The key elements of taking a pain history are delineated in Table 17-2. Chronic pain patients often have multiple pain complaints with different descriptions that may indicate multiple diagnoses. When this is the case, obtaining complete information on each complaint separately simplifies the diagnostic process.

Different pains have different *temporal patterns*. For example, a patient with a migraine would complain of unilateral throbbing head pain or toothache that lasts 4 hours to 3 days. The pain of trigeminal neuralgia is seconds in duration and may be triggered frequently throughout the day. The pain of a neuropathy is continuous.

TABLE 17-2 History of Pain

Chief complaint
Characteristics of pain
Location
Quality
Temporal patterns
Continuous or intermittent
Diurnal or nocturnal variation in intensity if continuous
Duration of each attack if intermittent
Pain intensity range (lowest, usual, and highest intensities)
Seasonal variation of symptoms if any
Associated symptoms
Precipitating factors if intermittent
Aggravating factors
Alleviating factors
Onset and history
Past and present medications or other treatments for pain
Past medical and dental history
Family history
Social history
Review of systems

Often there are *associated symptoms* such as nausea, vomiting, ptosis, tingling, or numbness. Visual changes may precede a migraine with aura. Nausea and vomiting often accompany severe headaches, especially migraine. Generalized malaise may accompany a temporal arteritis. Autonomic changes almost always accompany the trigeminal autonomic cephalalgias (TACs).

Unexplained neurological symptoms, however, such as cognitive or memory changes, transient sensory or motor loss, tinnitus, vertigo, or any of the previous symptoms not fitting an appropriate pain picture, may be signs of an intracranial lesion.

Precipitating, aggravating, and *alleviating factors* also add clues to the probable diagnosis. Withdrawal from alcohol or caffeine may trigger a migraine attack.[46] Light touch, shaving, or brushing teeth may precipitate trigeminal neuralgia. Cold weather, immobility, or over-exercise will aggravate myofascial pain (MFP).

Gathering information about the *onset* and *history* of the problem will provide further clues. What happened around the time the pain started, how has the pain changed, and what evaluations and treatments were tried? For example, a history of malaise and skin lesions (herpes zoster) typically precedes the pain of postherpetic neuropathy.

Knowing which specialists the patient has seen, which tests and radiographs were already completed, what the previous diagnostic impressions were, and which treatments were tried, helps the practitioner rule out various diagnoses and decide if other workups are needed. With acute pain, the history is usually quite short, but with chronic pain, the history may take hours to obtain, the patient having seen many different health care providers.

A *family history* should include information regarding the patient's parents and siblings. Does anyone in the immediate family suffer from a similar pain problem? For example, 70% of migraine patients have a relative who also has or had migraine. Does anyone in the family have a chronic illness? This person may provide a model for pain behavior and coping.

A *social history* should not only cover demographic information, marital status, household situation, and occupation but also seek to uncover any potential perpetuating factors. Look for potential stressors at work and at home and ask about postural habits, body mechanics with sleeping, driving, computer work, watching TV and reading, dietary habits, environmental factors, and drug and alcohol use.

The *past medical history* may reveal systemic illnesses such as lupus erythematosus or hypothyroidism that may predispose the patient to developing pain. Past surgeries and medications for other purposes, any psychiatric history, allergies, hospitalizations, and other illnesses must be included.

A *review of systems* screens the person's present state of health. It includes asking about any recent symptoms related to the head and neck; the skin; and the cardiovascular, respiratory, gastrointestinal, genitourinary, endocrine, neurological, and musculoskeletal systems.

The next step in diagnosis is a thorough physical examination.

Physical Examination

Disease of extracranial structures, particularly the oral cavity, must be sought and ruled out first. Intraoral examination that must include inspection of all intraoral tissues, teeth, and periodontium is discussed in detail in Chapter 8. Once acute disease has been ruled out, the physical examination must be augmented to include evaluation of the cranial nerves, temporomandibular joint (TMJ), cervical spine, and head and neck muscles. In specific cases, a more comprehensive neurological examination may be indicated.

General inspection can reveal a great deal about a patient to the alert clinician. A slouching posture can point to *depression*. Asymmetry, swelling, redness, and other signs may indicate inflammation, infection, or a neoplastic process. Closer inspection of the head and neck may reveal scars of past surgeries or traumas long forgotten by the patient.

The *stomatognathic examination* of the TMJs involves testing the range and quality of motion of the mandible, palpating and listening to joint noises, and palpating the lateral and dorsal joint capsules for tenderness. The normal range of jaw opening is 40–60 mm. Laterotrusive and protrusive movements should be 8–10 mm. The path of opening and laterotrusive and protrusive movements should be straight, without deflections or deviations. Joint capsule palpation anterior to the tragus of the ear for the lateral capsule and from the top of the condyle for the dorsal capsule may be "uncomfortable" but should not be painful.

A *cranial nerve examination* (Table 17-3) is indicated any time the history points to a non-odontogenic type of pain or when there are unusual complaints or symptoms. Detailed information on how to perform a cranial nerve examination is available in Bates's textbook on physical examination and history taking.[47] If intracranial disease is suspected, a complete *neurological examination* and referral to a competent neurologist is indicated.

Poor posture, especially anterior head positioning, is one of the most important contributing factors to TMJ dysfunction and MFP and also affects the occlusion.[48] The cervical spine is often the source of referred pain to the orofacial and TMJ regions. A discussion of the examination for posture and of the cervical spine is found in the section on Cervical Spine later in the chapter. For details on how and why this is important, the reader is referred to Chapter 5 in Travell and Simons' *Trigger Point Manual*, 2nd ed.[49]

Myofascial trigger point (TrP) examination requires a thorough, systematic palpation of all of the masticatory and cervical muscles, looking for tight muscle bands and the focal tenderness associated with myofascial TrPs. Myofascial TrPs are the most prevalent cause of chronic pain, both in the head and neck region and in general.[50–52] Also, it is frequently an accompanying diagnosis to other chronic pain conditions.

The next step involves choosing appropriate diagnostic studies to help rule out suspected disease or disorders and could include pulp testing; radiographs; more advanced imaging such as CT, CBCT, or magnetic resonance imaging (MRI); laboratory studies of blood or urine; and differential diagnostic analgesic blocking to name a few.

TABLE 17-3 Nerve Examination

I	Olfactory: test sense of smell of each nostril separately by using soap, tobacco, or coffee with patient's eyes closed
II	Optic: test visual acuity Examine fundi ophthalmoscopically Test visual fields
III, IV, VI	Oculomotor, trochlear, abducens: Test pupillary reactions to light and accommodation Test extraocular movements Check for ptosis or nystagmus
V	Trigeminal: Motor—palpate masseter and temporalis during contraction Sensory—Test discrimination of pinprick, V1, V2, V3 temperature, V1, V2, V3, and light touch, V1, V2, V3 Test corneal reflex
VII	Facial: Observe patient's face during rest and conversation Check for symmetry, tics Examine for symmetric smile, ability to wrinkle forehead, hold air in cheeks, and tense the platysma muscle
VIII	Acoustic: Whisper, rub fingers, or hold watch next to ear, use tuning fork for Rinne and Weber tests
IX, X	Glossopharyngeal and vagus are tested together: Check for symmetric movement of the soft palate and the uvula when patient says "Ah" Check gag reflex by touching back of throat. Note any hoarseness
XI	Spinal accessory: Have patient shrug shoulders against resistance (trapezius) (partially innervated by C4) Have patient turn head against resistance (SCM)
XII	Hypoglossal: Observe tongue in mouth: check for atrophy or asymmetry or fasciculation Check for deviation by having patient stick tongue out

A Word About Psychogenic Pain

Many clinicians use the term "psychogenic" as a diagnosis in patients in whom they cannot identify a disease process to explain the symptoms or in patients whose pain has caused a strong emotional component or in patients who have not responded well to somatic treatment. It must be emphasized, however, that while psychological factors are intimately involved in the expression of all pain, regardless of etiology or time course, it is *rare* for pain to be purely due to a psychological disorder. And whether or not the pain has an ongoing identifiable organic basis, it is still very real to the patient and can be as intense as any somatic pain. Also important

to remember is that referred pain from myofascial TrPs is frequently overlooked as an organic finding and is thus often mislabeled as psychogenic pain.[53]

"Psychogenic pain" or "somatoform disorders" are described in the DSM-V, the *Diagnostic and Statistical Manual of Mental Disorders*, and include physical complaints that, after appropriate medical workup and testing, either cannot be explained by a medical condition or substance (ab) use or, if due to an identifiable medical condition, cause physical, social, or occupational impairment greater than what would be expected given the objective findings. In addition, the complaints must have been present for over 6 months and cannot be explained by other psychological disorders such as depression or anxiety. Despite their inclusion in the *Diagnostic and Statistical Manual of Mental Disorders*, somatoform disorders have been the subject of continuing criticism by both professionals and patients[54] mostly because no empirical evidence exists to support these psychodynamic diagnoses and to date no psychodynamic treatment has been shown to be effective in treating these disorders. Thus the validity of these diagnoses is under debate.[55]

Finally, it is generally accepted that all chronic pain began at some point with a true pathological or nociceptive stimulus that elicited a "pain behavior." Most people live in an environment that systematically and positively reinforces pain behaviors while ignoring or punishing healthy behaviors.[56] In acute pain, these behaviors and their rewards subside quickly, but in chronic or persistent pain, pain behaviors and operant conditioning become more prominent and may persist even after the noxious stimulus is gone; it is this behavior that often leads some clinicians to doubt the patient's veracity or label them as having psychogenic pain.

Rather than falling into the trap of calling unidentified pain "psychogenic," the clinician should go back to the classification of pain to answer this question (Table 17-1).

SPECIFIC FOR EACH BROAD CATEGORY OF PAIN ORIGIN

The remainder of the chapter will be devoted to discussing several of the specific diagnoses that fall into each broad diagnostic category. This classification is by no means exhaustive. The disorders discussed are those with which dentists should be familiar. An exhaustive treatise of orofacial pains was published by Bell[57] and an official classification with diagnostic criteria by the International Headache Society.[45]

EXTRACRANIAL DISEASE

Local Disease of Extracranial Structures

Local disease of extracranial structures and referred pain from distant sites are listed in Tables 17-4 and 17-5. Acute pain arising from disease of the extracranial structures is commonly seen in dental practice. Most extracranial acute pain is well localized, easily identifiable, and straightforward to treat. However, some pains from local diseases are more elusive because they are referred from other head, neck, and thoracic structures.

TABLE 17-4 Local Disease of Extracranial Structures

Structures	Diseases
Tooth pulp, periradicular structures	Inflammation
Periodontium, gingival, mucosa	Infection
Salivary glands	Degeneration
Tongue	Neoplasm
Ears, nose, throat, sinuses	Obstruction
Eyes	
Temporomandibular joints	

TABLE 17-5 Referred Pain from Remote Sites

Structures	Diseases
Heart	Angina pectoris, myocardial infarction
Thyroid	Inflammation
Carotid artery	Inflammation, other obscure causation
Cervical spine	Inflammation, trauma, dysfunction
Muscles	Myofascial TrPs

Ear Pain

Patients may present themselves to the dentist with a primary pain complaint of toothache or temporomandibular joint pain, and ear pain is found to be an associated symptom. Similarly, when a medical workup for ear pain is negative, an astute physician will want to know whether the TMJ or dental infection is referring the symptoms.

Ear pain is typically seen with disorders such as otitis media, otitis externa, and mastoiditis and may be associated with headache.

Etiology. The ear is innervated by cranial nerves V, VII, VIII, IX, X, and XI and also branches of the upper cervical roots that supply the immediate adjacent scalp and muscles. Therefore, pain can be referred to the ear from inflammatory or neoplastic disease of the *teeth*, tonsils, larynx, nasopharynx, thyroid, *TMJ*, and cervical spine, as well as from inflammation or tumors in the posterior fossa of the brain.[58] Ear pains are also associated with pain from acute herpes zoster, glossopharyngeal neuralgia, and nervus intermedius neuralgia. These are discussed in the section "Neuropathic Pains."

Symptoms. Primary ear pains are usually described as a constant aching pressure. Their onset is usually recent. Inflammatory or infectious disorders may be associated with fever and malaise.

Patients with otitis externa (inflammation of the external auditory canal) may present themselves to the dentist first because *swallowing is an aggravating factor*. Otitis externa may be misdiagnosed as arthralgia of the TMJ if the condyle is palpated through the external auditory canal without first evaluating the ear. Glossopharyngeal neuralgia, also aggravated by swallowing, may need to be considered if ear and dental disease are absent.

Examination. The dentist must first rule out referred ear pain from oral or dental sources. Myofascial TrPs in the lateral and medial pterygoid, masseter, and sternocleidomastoid muscles frequently refer pain to the ear. Deep masseter and sternocleidomastoid muscle TrPs may also cause tinnitus.[49]

If primary ear pain is suspected, screening examination should include visual inspection of the ear and ear canal. Wiggling the auricle itself or tugging on the earlobe will aggravate otitis externa and will help distinguish an ear lesion from a TMJ problem. Pumping on the ear with the palm of the hand will exacerbate pain from otitis media. Hearing can be grossly evaluated by rubbing the fingers together in front of each ear.

Treatment. Primary ear pain should be diagnosed and managed by an ENT specialist.

Sinus and Paranasal Pain

The most common extraoral source of dental pain arises from the maxillary sinus and associated pain-sensitive nasal mucosa. Many teeth have been mistakenly extracted because of an incorrect diagnosis of this syndrome. For more details, see Chapter 16.

Temporomandibular Joint Articular Disorders

Table 17-6 provides a simple breakdown of TMJ disorders, the vast majority of which are non-painful. It is possible to have several of these diagnoses affecting one joint.

The term *internal derangement* applies to all joints and encompasses those disorders causing mechanical interference to normal joint functions. In the TMJs, this primarily involves displacement and distortion of the articular disk, as well as remodeling of the articular surfaces. Many of the articular disorders affecting the TMJs involve an abnormal or restricted range of motion and noise but are *relatively painless*. These include the congenital or developmental disorders, disk derangement disorders, osteoarthritis, and ankylosis listed in Table 17-6. Any pain associated with these disorders is usually momentary and associated with pulling or stretching of ligaments. In the case of *ankylosis*, pain ensues if the mandible is forcibly opened beyond adhesive restrictions. Forcible opening can cause acute inflammation. Primary or secondary *osteoarthritis*, unless accompanied by synovitis, is also associated with minimal pain or dysfunction,[50,59] although crepitus and limited range of motion may be present.

TABLE 17-6 Temporomandibular Joint Articular Disorders

Inflammatory disorders
Capsulitis and synovitis
Polyarthritides
Disk derangement disorders
Disk displacement with reduction
Disk displacement without reduction
Noninflammatory disorders
Osteoarthritis Primary
Osteoarthritis Secondary
Congenital or developmental disorders
Aplasia
Hypoplasia
Hyperplasia
Dysplasia
Neoplasia
Temporomandibular joint dislocation
Ankylosis
Fracture (condylar process)

Adapted from De Leeuw R.[59]

Inflammatory Disorders of the Joint

It is the inflammatory joint disorders that cause pain. Included in this group are *capsulitis and synovitis* and various *polyarthritides.*

Symptoms. Capsulitis and synovitis are inflammatory disorders of the joint that present with similar signs and symptoms and can only be differentiated with direct visualization of the tissues through arthroscopy or open joint surgery. Capsulitis or synovitis may occur alone or in combination with an internal derangement. The chief symptom is continuous pain over the joint, aggravated by function. Swelling may be evident, and the patient may complain of restricted jaw opening, ear pain, and an acute malocclusion (the back teeth on the same side do not touch). There is usually a history of trauma, infection, polyarthralgia, or chronic, nonpainful degenerative arthritis that is now in an acute stage.[59] Because the pain is fairly constant, the clinical picture is often complicated by central excitatory effects including myalgia or referred pain or myofascial TrPs. The pain tends to wax and wane with the course of the inflammation.

Symptoms in patients with a *polyarthritis* are similar to a capsulitis or synovitis and include pain at rest and with mandibular function, crepitus, and a limited range of motion.

Etiology. Inflammation of the capsule or synovium may occur secondary to localized trauma or infection, through overuse of the joint, or secondary to other joint disorders. Synovitis may also occur secondary to primary osteoarthritis.

The *polyarthridites* are due to systemic rheumatological illnesses that affect multiple joints. These most commonly include rheumatoid arthritis, juvenile rheumatoid arthritis, psoriatic arthritis, and gout. The autoimmune disorders and mixed connective tissue diseases such as scleroderma, Sjögren's syndrome, and lupus erythematosus may also affect the TMJ.[59]

Examination. For *capsulitis and synovitis*, palpation over the joint itself is painful. Posterior or superior joint loading and any movement that stretches the capsule may also increase the pain. Range of motion is not usually restricted except due to pain in the joint. Initiation of mandibular movement after a period of rest may be slow and "sticky." Inflammatory effusion in the joint may cause acute malocclusion with slight disclusion of the ipsilateral posterior teeth. Clenching may be painful, but biting on a tongue depressor ipsilateral to the affected joint may reduce the pain by preventing intercuspation and full closure.

In the *polyarthritides*, palpation of the joint is also painful. Range of motion is not affected except due to pain and

may be accompanied by crepitus; malocclusion is related to inflammatory effusion or lateral pterygoid muscle spasm secondary to the primary pain. In severe cases of rheumatoid arthritis, rapid resorption of the condyles causes an acute anterior open bite. Ankylosis may also occur.

Diagnostic Tests. Plain radiographs and tomography will show a few, if any, bony changes in the joint with *capsulitis or synovitis* unless these are secondary to osteoarthritis.MRI, on the other hand, will show a bright T^2-weighted signal in the presence of fluid.[59]

The *polyarthritides* will show extensive TMJ changes on radiographic examination. Radiographic changes include more gross deformities than are seen in osteoarthritis, including erosions, marginal proliferations, and flattening (Figure 17-8). Of particular diagnostic value in the polyarthritides are various serologic tests screening for evidence of rheumatoid disease such as antinuclear antibodies, elevated erythrocyte sedimentation rate, rheumatoid factor, or anemia, although not all rheumatologic diseases are seropositive. Definitive diagnosis and management of the polyarthritides are left to the rheumatologist. The dentist's role is to manage concomitant TMJ symptoms should they occur.

Treatment. In treating inflammatory TMJ disorders, palliative care is an appropriate first step. This includes jaw rest, a soft diet, and 7–14 days of *nonsteroidal anti-inflammatory drug* (NSAID) use. Intermittent application of ice or moist heat

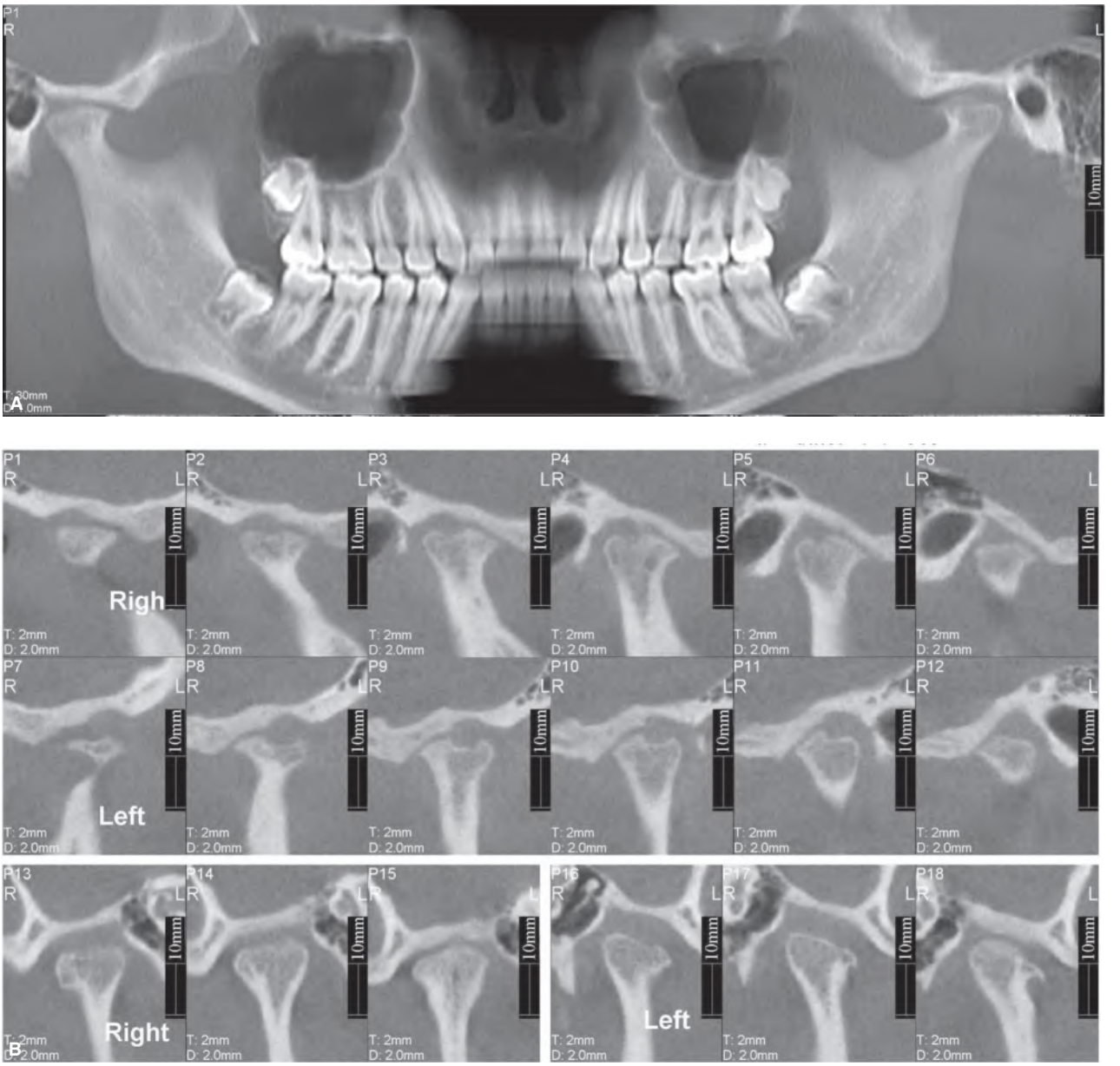

FIGURE 17-8 Tomography of a juvenile rheumatoid arthritic temporomandibular joint (TMJ). ***A.*** Observe the flattened surface of both TMJ condyles. ***B.*** Note the resorption of both articular surfaces of the TMJ and in particular, the significant resorption of the left condyle. (Courtesy of Dr. Marcela Romero, New York, NY, U.S.A.)

may also be helpful. Office physical therapy, including ultrasonography, may be indicated in some patients. Single supracapsular or intra-articular injections of corticosteroids may be effective in reducing inflammation and pain if simpler methods prove ineffective. Pain relief from steroid injection can be expected to last 18–24 months provided that the patient limits joint use appropriately. It is generally accepted that steroid injections into the TMJ should be limited: no more than two in 12 months, and two or three in a lifetime. This is because there is currently insufficient evidence about the long-term effects, efficacy, and safety of intra-articular steroid injection,[60,61] so erring on the conservative side is advised.

Definitive therapy is aimed at reducing the causative or contributing factors. This must involve the patient who controls the functional demands placed on the joint and must be motivated to limit joint use and eliminate abusive habits. Home exercises, application of heat and ice, and massage are also important. In addition to the self-help program, construction of a *stabilization splint* may help reduce symptoms. If necessary, a psychological approach to decrease emotional stress and/or daytime clenching may also be instituted. Surgery is seldom, if ever, indicated.

If the condition is due to polyarthritis, management is similar to treating the systemic disease itself. It includes analgesics and anti-inflammatory agents such as aspirin, indomethacin, and ibuprofen, corticosteroids such as prednisone, gold salts, and penicillamine, as well as the same palliative and supportive therapies mentioned previously. Surgical intervention is limited to severe cases with marked dysfunction.

Referred Pain from Remote Pathological Sites

Angina Pectoris and Myocardial Infarction

Severe pain of cardiac origin can be referred to the mandible and the maxillary region and, surprisingly, pain from a pulpalgia may refer down the ipsilateral neck, shoulder, and arm.[62] These unusual patterns of pain referral may be explained by the fact that dorsal root ganglion cells have been shown to branch in the periphery[33,34] and that, in the rat at least, the dorsal root ganglion cell supplying the heart also supplies the arm[33] (Figure 17-9). Potential convergence of nerve fibers from different sensory dermatomes in the dorsal horn of the spinal cord provides a basis for a "convergence-projection" phenomenon for the pain referral (Figure 17-5).

Symptoms. Angina pectoris is typically characterized by heaviness, tightness, or aching pain in the mid or the upper sternum. These symptoms may radiate upward from the epigastrium to the mandible—the left more frequently than the right. This pattern is similar to that seen with myocardial infarction (Figure 17-10A). Precipitating factors include exertion and emotional excitement or ingestion of food.[63] Angina attacks are usually short-lasting, rarely longer than 15 minutes. Anginal pain referred to the teeth may be experienced with or without concomitant chest pain.[64]

Myocardial infarction is characterized by a symptom complex that includes a sudden, gradually increasing precordial pain, with an overwhelming feeling of suffocation. The pain

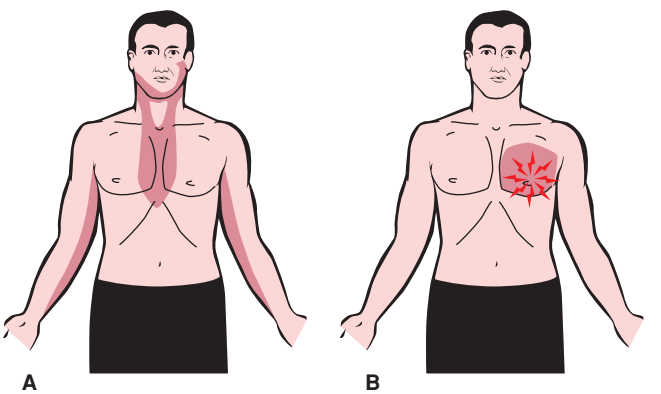

FIGURE 17-10 ***A.*** Pattern of pain and referred pain emanating from myocardial infarction. Central necktie pattern and greater left jaw and arm pain than on the right side are typical. ***B.*** Harmless noncardiac pain in the left chest. (Reproduced with permission from Turner GO, Med World News; April 6, 1973.)

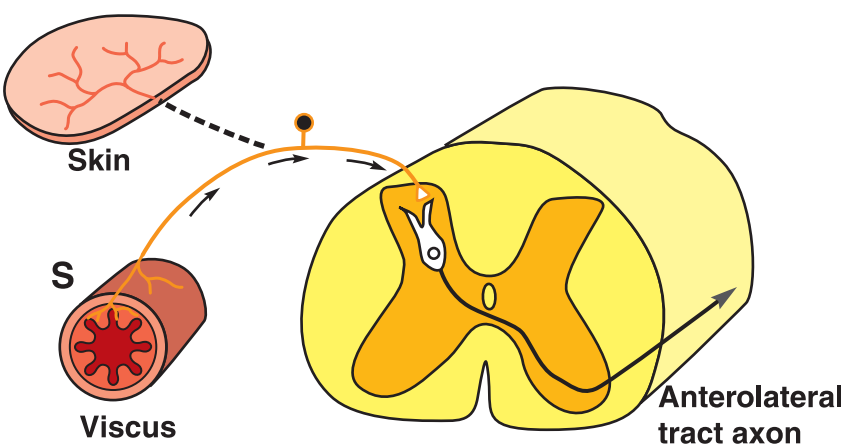

FIGURE 17-9 Branched primary afferent hypothesis of referred pain. According to this theory, a single primary afferent branches to supply both the deep structure stimulated and the structure(s) in which the pain is perceived. (Reproduced with permission from Fields HL, McGraw-Hill; 1987.[3])

of myocardial infarction is similar to angina but is more pronounced, long-lasting, and does not resolve with rest.

Severe pain in the left maxilla and mandible related to angina pectoris or myocardial infarction *may occur without any other symptoms*. Bonica reported an incidence as high as 18% for the presentation of cardiac pain as jaw or tooth pain alone.[65]

Examination. A careful history is important in diagnosing the referred oral pain of cardiac origin. The dentist must rule out dental disease quickly and efficiently.

Treatment. If previously undiagnosed cardiac pain is suspected, the patient must be referred to an emergency room immediately. The patient should be transported by emergency services personnel and not allowed to drive. It is wise to call the hospital and have them ready to receive the patient.

If the patient loses consciousness, basic cardiopulmonary resuscitation should be applied until help arrives. This may be lifesaving. Every dental office should be equipped with a defibrillator.

Thyroid

Disorders of the thyroid gland are prevalent in medical practice, second only to diabetes as an endocrine disorder.[47] *Subacute thyroiditis* (viral inflammation of the thyroid gland) is of interest to the dentist because it may refer *pain to the jaws and ears*.

Symptoms. The typical symptom picture includes a sore throat with pain over at least one lobe of the thyroid gland or pain radiating up the sides of the neck and into the *lower jaw, ears*, or occiput. *Swallowing may aggravate the symptoms*. This is usually associated with a feeling of pressure or fullness in the throat. There may be mild fever, asthenia, and malaise.[47] Symptoms may wax and wane over a period of months and then finally resolve with return of normal thyroid function.

Etiology. Subacute thyroiditis is reported to occur 2–3 weeks after an upper respiratory infection and is viral in origin.

Examination. The thyroid gland may be visibly enlarged and will be exquisitely tender to palpation with nodularity (Figure 17-11). If thyroiditis or other thyroid disease is suspected, referral to the patient's physician should be made.

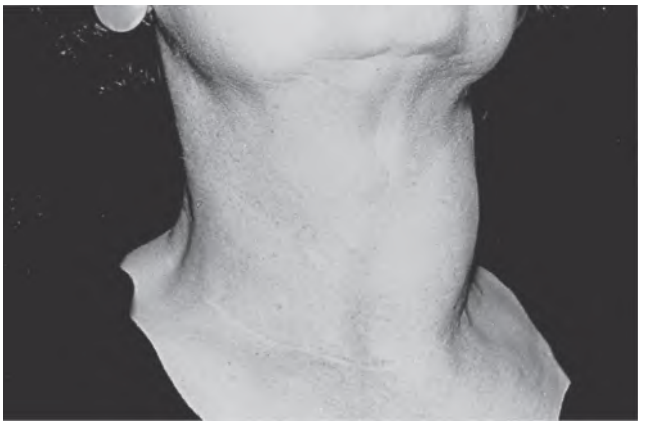

FIGURE 17-11 Benign thyroid tumor that recurred following thyroidectomy was referring pain into the mandibular first molar on the homolateral side.

Treatment. Subacute thyroiditis may resolve spontaneously. Large doses of aspirin and occasionally steroids are used to control the pain and inflammation. Thyroid supplements are sometimes indicated.[47]

Carotid Artery

Carotidynia is a nonspecific symptom of unilateral vascular neck tenderness that was first described by Temple Fay in 1927.[66] Stimulation of various parts of the carotid artery in the region of the bifurcation was shown to cause pain in the *ipsilateral jaw, maxilla, teeth, gums*, scalp, eyes, or nose. Long thought to be a benign complaint, frequently associated with migraine, and often responding to migraine therapy,[67] carotidynia was removed from International Headache Society (IHS) classification in 2004 due to an apparent lack of radiological or pathological findings.[45] More recently, various systematic MRI and ultrasound and PET-CT studies have found evidence of abnormal soft tissue infiltration surrounding the symptomatic carotid arteries, renewing the interest in carotidynia as a potential distinct disease entity.[68]

Symptoms. Continuous or intermittent dull aching, rarely pulsing, jaw and neck pain, with occasional spread to the *masseter muscle, temple and TMJ region*. Aggravating factors may include *chewing, swallowing*, bending over, or straining at stool. Precipitating factors may include an inflammatory response secondary to physical trauma or bacterial or viral infection, drugs, or alcohol.

Etiology. Recent studies support the idea that there may be inflammation or abnormal soft tissue infiltration surrounding the symptomatic tender carotid bulb.[69]

Examination. Palpation may reveal tenderness and swelling over the ipsilateral carotid artery and aggravate the pain. The thyroid is nontender. All other tests and examinations are normal. Carotidynia has an extensive *differential diagnosis*, including such conditions as carotid artery dissection, aneurysm, fibromuscular dysplasia, elongated stylohyoid process, and temporal arteritis.

Treatment. Treatment recommendations range from reassurance and local heat to NSAIDs, steroids, or migraine medications.

Cervical Spine

There is a close functional connection between the cervical spine and the cranium and mandible. Many of the ligaments and muscles of the neck attach to both the head and the jaw, and all movements of the head and the neck affect the muscle activity in the masticatory muscles and the position of the mandible.[70] Because of these connections and the fact that disorders of the cervical spine and neck area may refer pain into the *facial region* due to the convergence of cervical and trigeminal primary afferent nociceptors in the nucleus caudalis of the spinal trigeminal tract, the cervical spine and musculature must be included in the assessment of orofacial pain complaints.

The normal cervical spine has 37 individual joints, making it the most complicated articular system in the body.[71] Most

of the structures in the neck have been shown to produce pain when stimulated.[72] Pain may be elicited through several different pathological processes including trauma; inflammation or misalignment of the vertebral bodies; inflammation or herniation of the intervertebral disks; trauma or strain in the spinal ligaments; and strains, spasms, or tears of the cervical muscles, and cervical dysfunction (lack of normal anatomical relationship or restricted functional movement of individual cervical vertebral joint segments).

Non-musculoskeletal pain-producing structures of the neck include the cervical nerve roots and nerves and the vertebral arteries.[72] Trauma, inflammation, and compression are implicated in the etiology of pain from these structures as well.

Acute cervical trauma and primary pathological processes of the neck are obviously not in the realm of the dentist to diagnose and treat; therefore, these will not be discussed. However, the cervical spine must be recognized as a potential source of dermatomal and referred pain into the head and the *orofacial* region.

Neck pain is very common. The pain is usually felt in the suboccipital region, may originate in any cervical structure, and is often referred to the frontal, orbital, temporal, parietal, and vertex regions.[72] Vertebral artery involvement or convergence of cervical neural input onto the spinal accessory and vagus nerves may induce visceral symptoms such as nausea, dizziness, or visual disturbance.[73] Recognizing the cervical contribution to these symptoms, along with prompt referral to an experienced physical therapist, may prevent the development of joint restrictions, cervical dysfunction, and progressive cervical disease such as bulging discs or spondylosis.

The tactile and painful dermatomes of the upper cervical nerves, C2 and C3, include the lower border of the mandible and the area surrounding the TMJs, a good portion of the external ears, posterior temporal and parietal regions of the head (Figure 17-12). Pain or dysfunction of these upper cervical areas may present as *mandibular or TMJ pain, lower molar toothache, throat tightness or difficulty swallowing*. Entrapment or chronic irritation of the nerve roots in the C4 to C7 area generally produces pain in the respective dermatomes in the shoulder and arm regions.[74] *Dermatomal pains* may have an aching or neuritic quality and typically follow the distribution of the cervical nerve root involved. Greater and lesser occipital nerves are often implicated as contributing to various headache presentations and nerve blocks are sometimes part of the treatment.[75] *Referred pains* on the other hand, are usually deep and aching and may present as headache that is not consistent with the dermatomal distribution of any of the cervical nerves. Referred symptoms from the neck may include *ipsilateral masticatory muscle tenderness* and *pain in the preauricular area*, *body of the mandible* and mastoid.

Examination. Postural evaluation is essential for all facial pain patients. Of particular importance is anterior head position. One simple quick test is to view the standing patient from the side. The ear should be in line with the acromioclavicular joint. If the ear is anterior to this landmark, the posterior cervical muscles, upper trapezius, levator scapula, and

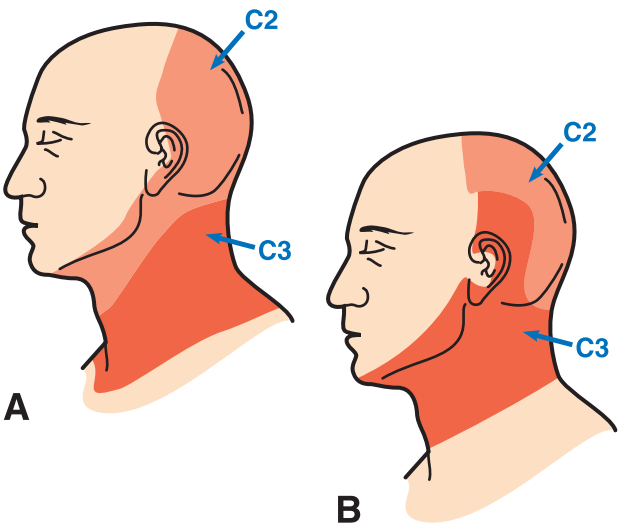

FIGURE 17-12 A study by Poletti revealed a slightly different distribution of C2-C3 dermatomes for pain than those previously defined by using tactile criteria. ***A.*** Upper image depicts the C2 and C3 tactile dermatomes previously defined by Foerster. (Reproduced with permission from Foerster O. The dermatomes in man. *Brain* 1932;56:1–38.) ***B.*** Lower image depicts C2 and C3 pain dermatomes as defined by Poletti. (Reproduced with permission from Poletti CE. C2 and C3 pain dermatomes in man. Cephalalgia. 1991;11:155–9.)

SCM muscles are activated to keep the head rotated up and the eyes looking forward. This results in flexion of the lower cervical spine and extension of the upper cervical spine with potential for compression of neural and vascular elements between the occiput and C1 and C1 and C2. With anterior head positioning, the jaw elevator muscles are also activated as they counteract the gentle downward forces on the mandible from the supra and infra hyoid muscles that are now on the stretch. Changes in head position have been shown to affect occlusion and maximum mandibular opening.[76]

The examination of the cervical spine should include range of motion in flexion, extension, lateral flexion, and rotation. Additionally, the movement of the individual upper cervical segments (occiput-C1 and C1-C2) should be evaluated.[48] Cervical range of motion is often restricted with cervical dysfunction. Upper cervical dysfunction may be unilateral or bilateral.

Treatment. Physical therapy, including cervical joint mobilization along with a comprehensive home exercise program and postural retraining, is required to treat pain of cervical origin.

Local treatment of referred symptoms does not provide long-lasting, if any, relief. If the patient has a true dental or temporomandibular disorder *and* dysfunction of the cervical spine, both problems must be addressed.

INTRACRANIAL DISEASE

Although rare, intracranial lesions can cause pain referral to all areas of the head, face, and neck, including the oral cavity. Neurological or neurosurgical evaluation is critical to rule

out space-occupying lesions, intracranial infections, or neurological syndromes.

Intracranial causes of head and neck pain include those caused by *traction on pain-sensitive structures* (the venous sinuses, dural and cerebral arteries, the dura mater, and the cranial nerves) and those caused by *specific CNS syndromes*, such as neurofibromatosis or thalamic pain[77] (Table 17-7). Different types of cerebrovascular accidents and venous thrombosis may also cause painful CNS lesions.

Intracranial lesions are usually associated with different focal neurological signs or deficits such as weakness, dizziness, difficulty in speaking or swallowing, ptosis, areas of numbness, memory loss, or mental confusion. When painful, the quality of pain may vary from a generalized throbbing or aching to a more specific paroxysmal sharp pain. Systemic symptoms such as weight loss or fever, sudden onset of pain, or a pattern change of existing pain should all be cause for concern and raise red flags that the cause may be intracranial.

NEUROVASCULAR PAINS

This category of pain encompasses several of the primary headache disorders (headaches not of pathological origin) such as migraines and the trigeminal autonomic cephalalgias (TACs), as well as some headaches associated with pathological vascular disorders, such as temporal arteritis (Table 17-8).

TABLE 17-7 Intracranial Causes of Pain

Neoplasm	Vascular malformations
Infection	Neurofibromatosis
Abscess	Thalamic pain
Meningitis	High/low intracranial pressure
Encephalitis	Ischemic cerebrovascular disease
Hemorrhage	Venous thrombosis

TABLE 17-8 Neurovascular Pain

Migraines
- Migraine without aura
- Migraine with aura

Trigeminal Autonomic Cephalalgias (TACs)
- Cluster headache
  - Episodic
  - Chronic
- Paroxysmal hemicrania
  - Episodic
  - Chronic
- Short-lasting unilateral neuralgiform headache attacks (SUNHA)
  - With conjunctival injection and tearing (SUNCT)
    - Episodic
    - Chronic
  - With cranial autonomic symptoms (SUNA)
    - Episodic
    - Chronic
  - Hemicrania Continua

Headaches associated with vascular disorders
- Arteritis
- Carotid or vertebral artery pain

Adapted from Headache Classification Committee of the International Headache Society, 2013.[45]

The list includes only those headaches that have a higher likelihood of presenting in the dental office due to the documented likelihood of occasionally presenting in an intraoral site.[78,79]

Benoliel et al[78] and Sharav[80] proposed creating an additional diagnostic subcategory in the IHS criteria[45], namely that of *neurovascular orofacial pain* in order to encompass those migraines and TAC type of pains that occur in the lower two-thirds of the face.

In general, these headache types share the following features. They all have primarily a deep, throbbing or pulsating quality, occasionally with superimposed sharp pain, and occasionally with an aching or burning background. The pain is exclusively or *predominantly unilateral* with pain-free or almost pain-free periods between attacks. The main difference between the different headache types lies in their temporal patterns (Figure 17-13) and their associated symptoms.

Migraine

Migraine is a debilitating, inherited, episodic disorder and a form of sensory processing disturbance in the CNS.[81] Migraine without aura and migraine with aura are the two most common forms. Migraine headaches are more common in females and 50% begin before the age of 20.[82]

Symptoms. The migraine headaches typically start on one side but can spread to involve both sides. There is often a premonitory period before the headache emerges that lasts hours and up to 2 days. This consists of combinations of vague symptoms such as fatigue, difficulty concentrating, neck stiffness, nausea, blurred vision, food cravings, and/or depression. These symptoms are part of the migraine process and are not considered to be triggers.

Some migraine patients do report having specific triggers to their headaches. These may include foods such as chocolate, cheese, fruit and alcohol (especially red wine), menstruation, weather changes, lack of sleep, histamines, and vasodilators.

The headache itself is predominantly unilateral in the frontal, temporal, or retrobulbar areas, although about 3% of migraine patients will complain of intraoral pain,[83] increasing the likelihood that an undiagnosed migraine patient will seek consultation with a dentist or endodontist before seeing their physician or an orofacial pain specialist.

The pain may begin as an ache but usually develops into a pain of throbbing, pulsating, or beating nature and, if untreated, will last 4–72 hours with variable pain-free periods (days to years) between attacks. Aggravating factors include exertion; patients will avoid activities such as walking, climbing stairs, coughing or moving the head. Associated symptoms may include photo~ and phonophobia, nausea, and/or vomiting. Sleep helps the headache to abate.

"Facial migraines" or *neurovascular orofacial pains* is pain with many similarities to migraine that occurs in the lower two thirds of the face, most frequently in the *alveolus* occasionally with an *associated mucosal site*. There may be referred *pain to the lips or chin, or to the infraorbital or preauricular*

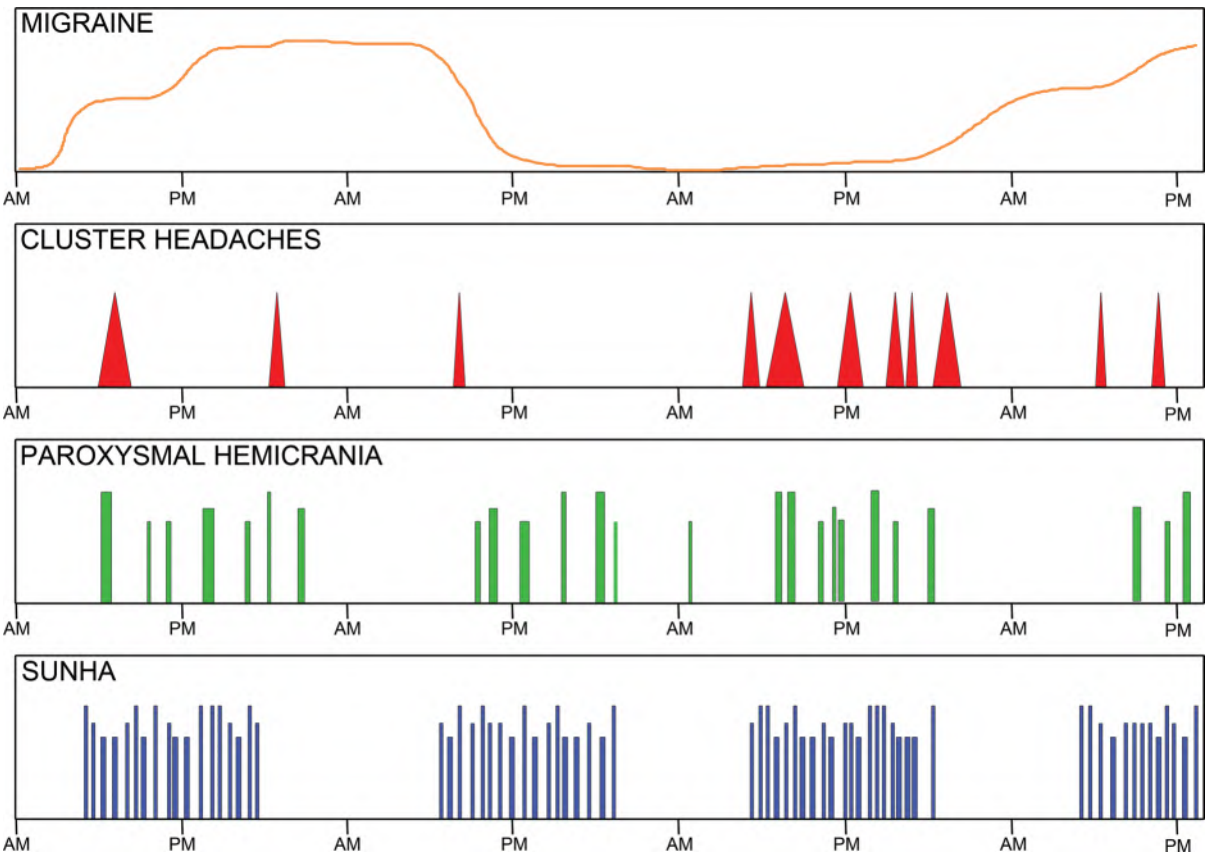

FIGURE 17-13 Comparison of temporal pattern of four main "neurovascular" headache types. (Reproduced with permission and courtesy of Drs. Bernadette Jaeger and Austin Skootsky, Los Angeles, CA, U.S.A.)

regions.[78] Neurovascular orofacial pain, similar to migraine, is also more common in women, primarily unilateral in location, and throbbing in quality. The pain may last minutes to hours, some patients reporting that the pain lasts over 24 hours. Over 36% of patients have associated autonomic symptoms[79] such as tearing, nasal congestion, photo~ or phonophobia, and nausea.[78] A potentially misleading sign is that some of these patients also report *dental sensitivity to cold*.[84] Often patients will present having had one or more root canal treatments in the teeth in the painful region without relief of their pain.

Migraine with aura is distinguished from *migraine without aura* by the occurrence of transient, reversible, focal neurological symptoms (the aura), 5–20 minutes prior to the onset of headache pain. Visual auras are most common and may present as flashing lights, halos, or loss of part of the visual field. Somatosensory auras are also common and consist of dysesthesias that start in one hand and spread up to involve the ipsilateral side of the face, nose, and mouth.[45,85,]

Often patients with migraine, especially migraine without aura, have *overlying MFP*, causing additional dull, aching headache pain.[86,87] The prevalence of temporomandibular disorders (TMDs) and various orofacial pain disorders in relation to headaches has been reported.[87]

Etiology. The exact etiology and pathogenesis of *migraine with aura* headaches remain controversial, the past attribution to intra and extracranial vascular changes having been disproven through magnetic resonance angiography studies.[89]

Current evidence suggests that migraine arises from a "brain state of altered excitability" that leads to activation and sensitization of the trigeminovascular system in genetically susceptible people."[4] The trigeminovascular system consists of the trigeminal neurons and the blood vessels they innervate. In migraine, this consists largely of the ophthalmic division of the trigeminal nerve and the large pain producing cerebral blood vessels.

The migraine aura is associated with a brief hyperaemia in the brain that correlates with visual changes, followed by a pathognomonic spreading oligemia (decrease in blood flow) that starts in the visual cortex and spreads forward and persists throughout the headache phase.[90] These regional cerebral blood flow changes have not been found in migraine without aura, although blood flow changes in the brain stem may occur.[45] The cortical spreading depression has been shown in animals to cause sustained activation of the meningeal nociceptors and central trigeminovascular neurons and is thereby thought to activate headache mechanisms.[4]

The pain of migraine is thought to be *referred* from a sterile neurogenic inflammation, caused by the release of vasoactive neuropeptides from nerves surrounding the affected intracranial vasculature, to the more superficial cranial structures through convergence of vascular and somatic nerve fibers on single second-order pain transmission neurons in the trigeminal nucleus caudalis.[91] While the mechanism of pain

generation in migraine is still controversial, evidence suggests that central sensitization in the trigeminocervical complex (the trigeminal nucleus caudalis and the dorsal horns of C1 and C2), together with the reflex connections of the trigeminal system with the cranial parasympathetic neuronal flow mediated through the pterygopalatine, otic and carotid ganglia, results in headache and cutaneous allodynia.[92–95]

Examination. The diagnosis of migraine can usually be made by history, but in the case of neurovascular orofacial pain, examination of the patient complaining of undiagnosed intermittent toothache or facial pain should include a thorough dental, TMJ, and muscle evaluation. Once obvious dental and joint pathology have been ruled out, and the qualitative and temporal pattern of the pain raises the possibility of dental or facial migraine, referral to an orofacial pain dentist should be made. Dental heroics should be avoided.

Diagnostic Tests. There are no diagnostic tests that will confirm the diagnosis of migraine headache. If the headache is of recent onset or the pattern of a preexisting headache has changed, a neurological evaluation including a CT scan or an MRI may be indicated.

Treatment. Long-term management of migraine headache typically requires identification and control of any obvious contributing factors such as diet, sleep, and stress. Restriction of alcohol intake, regular meals, exercise, relaxation therapy, and good sleep hygiene may help prevent the onset of migraine. If myofascial TrPs or musculoskeletal dysfunction is present, competent treatment of these will significantly reduce the frequency of vascular headache symptoms and reduce the need for migraine medications. For those patients in whom stress, depression, or anxiety play a role, psychological interventions such as stress management and relaxation training, biofeedback, or cognitive behavioral therapy may be indicated.

Medications used in the treatment of migraine include abortive and prophylactic drugs, the choice of which depends on the frequency of migraine attacks. Individuals who suffer less than three or four migraines per month may do well with abortive drugs that are taken during the onset of the headache or migrainous facial pain.

Abortive drugs are particularly useful in migraine with aura for which the aura warns the patient that a headache is pending. The serotonin 5-HT(1B/1D) agonists or "triptans" are highly effective. They improve headache as well as nausea, photo~ and phonophobia, and cutaneous allodynia.[95]

Prophylactic medications are used when headache frequency exceeds 3 or 4 per month since the triptans may cause coronary vasospasm or ischemia. Prophylactic medications include β-blockers, tricyclic antidepressants, calcium channel blockers, 5-HT antagonists, and antiepileptic drugs such as valproic acid, gabapentin, and topiramate.

Despite a plethora of migraine specific medications, up to half of migraineurs do not seek medical advice and self-medicate. The most common remedy is bed rest and over 50% of patients use over the counter analgesics. This is despite the fact that studies have shown that there is an improved quality of life when patients use triptans.[96]

The use of botulinum toxin has been approved by the US Food and Drug Association for use in chronic migraine headaches, cases in which a person has more than 15 days of headache per month.[97] The protocol consists of injection of 5 units of Botox in 31 sites along the corrugator, procerus, frontalis, temporalis, occipital, cervical paraspinals and upper trapezius muscles bilaterally for a total of 155 units.

Trigeminal Autonomic Cephalalgias

Cluster headaches, paroxysmal hemicranias, short-lasting neuralgiform headaches (SUNCT and SUNA), and hemicrania continua (HC) belong to the TACs. Because the *pain quality is similar to dental pulpitis*, any of these headache types may present first in the dental office.[78,98] All three share several clinical characteristics: unilateral, pulsatile or sharp, severe trigeminal pain with significant autonomic features such as tearing, rhinorrhea, and conjunctival injection and often cause awakening from sleep. They may share similar etiological or pathophysiological mechanisms. Nonetheless, they are easily distinguishable clinically and clearly differ from migraine.

Symptoms. Cluster headaches are probably one of the most painful conditions known to man and derive their name from their temporal pattern. They tend to occur in "clusters," a series of once every other day to eight 15- to 180-minute attacks per day lasting for several weeks or months, followed by remissions of months or years[45] (Figure 17-13). These headaches are found four to five times more frequently in men than in women, particularly in men aged 20–50 years *who smoke*.[99] The pain is a severe, unilateral, continuous, intense ache or burning that often occurs at night. The most common site is around or behind the eye radiating to the forehead and temple or around and behind the eye radiating infraorbitally *into the maxilla and occasionally into the teeth*, rarely to the lower jaw and neck.[100] Because of the oral symptoms, *serious diagnostic errors are committed by dentists*. Researchers from the University of California, Los Angeles reported that 42% of 33 patients suffering cluster headache had been seen by dentists and that 50% of these patients *had received inappropriate dental treatment*.[101]

Visual or sensory auras are absent with cluster headaches, but nasal stuffiness, lacrimation, rhinorrhea, conjunctival injection, perspiration of the forehead and face, and Horner's syndrome (ptosis of upper lid and miosis) are characteristic. Precipitating factors may include alcohol or other vasodilating substances, such as nitroglycerine. Cluster headache patients typically pace the floor during their headaches due to the intensity of pain. This is in contrast to migraine patients who typically retreat to a dark room to sleep.

Cluster headaches may progress from the more classic episodic form, occurring in periods lasting 1 week to 1 year, with at least a 2-week pain-free period in between, to a chronic form in which remissions are absent or last less than 2 weeks.[45] Rarely is the chronic form present from the onset.

Paroxysmal Hemicrania has similar characteristics to cluster headache, including *pain in dental structures*, but the attacks are shorter lasting (2–30 minutes), more frequent (5 per day for more than half the time),[45] and occur almost equally in men and women,[102] with a mean age of onset between 30–40 years. Most attacks are spontaneous, but certain triggers have been identified including neck movement, stress, alcohol, and exercise.

The quality of pain is throbbing, stabbing, sharp, or boring with an intensity that parallels that of cluster headaches, the patient often choosing to pace the floor during an attack. Pain location is predominantly oculotemporal, frontal and maxillary, always on the same side, and can spread to involve the entire side of the head and the neck, shoulder, and arm.[102,103] Cases involving paroxysmal hemicrania presenting as *intermittent toothache* have been reported.[104]

As with cluster headache associated symptoms include cranial autonomic symptoms and while they are predominantly unilateral, both sides may be affected. As with cluster, paroxysmal hemicrania may wake the patient from sleep, although this is not considered characteristic.[105] The awakenings with both headache types is considered to be REM sleep related.[106] Nausea and vomiting are usually absent, as are visual or somatosensory auras. Paroxysmal hemicrania is completely responsive to indomethacin.

The *short-lasting, unilateral, neuralgiform headache attacks (SUNHA)* are characterized by moderate to severe, extremely short-lasting attacks (from 5 seconds to 3 minutes) of unilateral, stabbing, sometimes electrical or burning, rarely pulsatile[106] ocular or periocular pain that *may radiate into the teeth*. Attacks occur 3–200 times per day, and there is prominent redness and tearing of the ipsilateral eye.[45]

There are two subtypes of SUNHA, short-lasting, unilateral, neuralgiform headache attacks *with conjunctival injection and tearing (SUNCT)* and short-lasting, unilateral, neuralgiform headache attacks *with cranial autonomic symptoms (SUNA).*[45] These two subtypes are differentiated primarily by the presence of both conjunctival injection and tearing in SUNCT and only one or none in SUNA. SUNCT may be a variation of SUNA.[4]

Because of the electrical, stabbing, short-lasting characteristics, SUNCT and SUNA may be difficult to distinguish from first division trigeminal neuralgia, although the pain is reported to be less intense. Triggering factors include light mechanical stimuli but, in contrast to trigeminal neuralgia, there is a short latency between the stimulus and the onset of pain.[107] Neck movements may also set off an attack. Also in contrast to trigeminal neuralgia, SUNCT/SUNA has no refractory period after an attack.[108] Trigeminal neuralgia and SUNCT/SUNA may occur together in the same patient. Because the signs and symptoms overlap, the differentiation may be clinically quite difficult and in those cases, patients should receive both diagnoses.[45]

Hemicrania Continua. This headache is strictly unilateral and completely responsive to indomethacin.[45] The pain is a continuous throbbing, sometimes dull and pressure-like, with mild autonomic symptoms, and daily or every other day exacerbations that last from 30 minutes to 10 hours, rarely several days. When severe, it resembles migraine and even has similar associated symptoms such as nausea, photo~ and phonophobia. Because of the similarity of symptoms to other chronic daily headaches (chronic or transformed migraine, chronic tension-type headache, new daily persistent headache) HC is often misdiagnosed. Up to *33% of patients consulted a dentist first* and *20% were given a dental diagnosis.*[109] HC, similar to paroxysmal hemicrania, is completely responsive to indomethacin, the headache abating within hours of being given an adequate dose of the medication.

Etiology of TACs. Headache presentations consistent with any of the TACs may be secondary to head trauma, or other pathologic phenomena such as malignancy, benign tumors, carotid artery dissection, vertebral artery injury, brain stem infarction, or arteriovenous malformations to name a few.[4] Thus, careful history taking and thorough evaluations are very important in all cases of unusual pain presentations, especially in the absence of obvious dental pathology.

The exact etiology or pathophysiology of TACs is as yet unknown; however, the similarity of the symptoms, the rhythmicity and autonomic features suggest a common pathophysiology. Because of the rhythmicity and predilection for attacks to occur at night, involvement of the posterior hypothalamus was suspected and confirmed by various neuroimaging techniques.[110,111] The location of the pain and the accompanying autonomic symptoms, implicates activity in the first and second divisions of the trigeminal nerve. Vasodilation, once considered an important element in the pathogenesis of cluster, is now thought to be secondary to trigeminovascular activation.[112] This is because the pain of cluster headache typically precedes any extracranial blood flow changes, and intracranial blood flow studies have shown inconsistent results.[112] Instead, trigeminal activation is implicated in causing neurogenic inflammation in the peripheral sites subserved by V1 and V2, resulting in clinical redness, swelling and pain. The parasympathetic nervous system is activated through connections in the salivary nucleus in the brain stem resulting in the autonomic phenomena (the trigemino-parasympathetic reflex).[113]

Examination. The diagnosis of all of the TACs is based on signs and symptoms. There are no diagnostic tests to confirm a diagnosis of TAC. Either the patient presents with a typical history and no abnormalities on physical and neurological examination or the examination causes suspicion of organic lesions with an ultimately normal neuroimaging scan.[45] Mild ptosis and miosis with or without periorbital swelling and conjunctival congestion may be present on the side of the headache if the patient is seen during or shortly after an attack. Dental pathology, part of the differential in the presentation of many TACs, must be carefully ruled out.

Treatment. Definitive treatment of TACs is best left to the orofacial pain dentist or other health care providers with a specific interest in headache management.

Cluster Headaches. Many of the treatments used for migraine therapy are also useful in cluster headaches, including symptomatic use of subcutaneous or intranasal triptans.[4] In general, however, prophylactic medications are more appropriate in cluster headache because of the frequency of attacks and because prodromal warning symptoms are rare. Cluster headache patients are often wakened from sleep, and the pain reaches its high intensity very quickly. Once the cluster period subsides, patients are weaned from their prophylactic medications until the headaches recur.

Oxygen inhalation (100% 7–8 L/min with a nonrebreathing mask), given at the very beginning of an attack for 15 minutes, may be successful in aborting an attack. In rare cases of resistant chronic cluster headache, microvascular decompression or lysis of the trigeminal ganglion, deep brain stimulation, gamma knife treatment, or stimulation of the posterior inferior hypothalamus may be considered.[4]

Paroxysmal Hemicrania and HC. Indomethacin is the medication of choice for paroxysmal hemicrania. If the pain does not resolve with this medication, it is unlikely to be paroxysmal hemicranias or HC. Aspirin and naproxen have a partial effect, but the relief is not as dramatic as with indomethacin.[114]

SUNHA. SUNCT and SUNA appear to be quite resistant to most of the usual first line medication choices for neurovascular headaches and also those medications used for neuropathic pains such as trigeminal neuralgia. The medication that has shown the most promise is lamotrigine and this drug is now considered the first line of treatment, followed by gabapentin and topiramate.[107] Neurovascular decompression of the trigeminal nerve has been shown to have high efficacy in resistant cases,[115] further supporting a potential connection between SUNCT, SUNA, and trigeminal neuralgia.

Headaches Associated With Vascular Disorders

This category includes headaches that are attributable to demonstrable disease, such as *giant cell arteritis*. Other pathological vascular causes of headache include vertebral or carotid artery dissection, subarachnoid hemorrhage, arteriovenous malformations, or venous thrombosis. Headaches due to such intracranial disease are unlikely to appear in the dental office and are therefore not further described. *Giant cell arteritis*, however, does on occasion present with *dental symptoms*, and may produce serious, irreversible consequences, such as blindness, if left unrecognized and untreated.

Giant Cell Arteritis (Temporal Arteritis)

Symptoms. The patient with giant cell arteritis is usually over 50 years old and may have other rheumatic symptoms, such as muscle aches, morning stiffness and polymyalgia rheumatica. Most have head, face, or neck pain, and involvement of the temporal artery may cause pain or tiredness with mastication ("jaw claudication"), inefficient chewing and perhaps even temporomandibular joint arthralgia, that may be the first or only complaints.[116] *Burning tongue or dental pain* has also been reported and missing the correct diagnosis has resulted in *inappropriate, ineffective endodontic surgery*.[117]

The pain is usually unilateral and in the anatomic area of the artery. However, it may radiate down to the ear, *teeth*, and occiput with generalized scalp tenderness and arthralgia.[118] Temporal arteritis may resemble a migraine attack because it, too, has a persistent throbbing quality that may last hours to days and the location is unilateral, over the temple area. *Temporal arteritis*, however, has an *additional burning, ache-like quality*. The pain increases with lowering of the head, *mastication*, and movements that create increased blood flow to that artery.

The patient may present with complaints of malaise, fatigue, anorexia, and weight loss if the arteritis occurs as a febrile illness. In advanced cases, patients may complain of transient visual loss on the side of the headache. This is a particularly severe symptom and requires immediate, aggressive treatment since thrombosis of the ophthalmic artery may result in partial or complete blindness. In up to 38% of patients, visual symptoms may be the only presenting symptom.[119]

Etiology. Arterial inflammation or chronic vasculitis, that may often be associated with immunological disorders, is the causative factor in this headache. Arterial biopsy often reveals frayed elastic tissues and giant cells in the vessel walls on histological examination.[118]

Examination. Dental examination is negative. The temporal artery may be tender to palpation, thickened, and enlarged and may lack a normal pulse.

Digital pressure with occlusion of the common carotid artery on the same side will frequently alleviate the symptoms. Ophthalmological examination or evaluation of optic ischemia is mandatory.

Diagnostic Tests. In giant cell arteritis, erythrocyte sedimentation rate, although a nonspecific test, will be significantly elevated. Definitive diagnosis is based on arterial biopsy. The entire temporal artery is typically removed to ensure sampling of diseased sections; skip lesions are common. Treatment is never delayed if visual disturbance is present.

Treatment. The dentist who suspects temporal arteritis should immediately refer the patient to a rheumatologist or an internist for complete workup. If optic symptoms are present, emergency ophthalmological examination is essential without delay.

Treatment of the condition consists of emergency dosages of steroids. When the elevated sedimentation rate has been reduced, maintenance doses of *prednisone* are administered as clinically determined.

Carotid or Vertebral Artery Pain. The dissection of the carotid or vertebral arteries, sometimes secondary to trauma such as whiplash and sometimes completely spontaneous, causes headache and cervical pain on the same side as the dissection. This serious, life-threatening condition is typically acute and accompanied by symptoms of transient ischemic attack or stroke and patients will seek medical

evaluation. However, sometimes patients with carotid dissection have symptoms consistent with one of the TACs and, due to *referral of pain into the jaws and teeth*, may, on occasion first seek dental care. Careful history and awareness of unusual symptoms and signs will help the clinician realize that this is not a dental problem and make appropriate and urgent referrals.

NEUROPATHIC PAINS

Neuropathic pain is pain that is due to some form of lesion or disease of the somatosensory system. This is in contrast to the normal transmission of noxious signals along nerves and to the central nervous system and cortex.

Neuropathic pain may occur after trauma, infection, or other disease process that affects the nerves or central nervous system directly such as mechanical damage from compression (such as with intraosseous dental implants), trauma, entrapment, traction or scarring; metabolic disorders such as diabetes; alcoholism or nutritional deficiencies; neoplastic disorders such as multiple myeloma; toxic reactions to drugs, metals, or certain organic substances; infectious or inflammatory processes such as herpes, hepatitis, leprosy; immune diseases such as multiple sclerosis; or genetic disorders such as porphyria.

It may also be due to changes that occur after an insult that leaves the normal sensory processing in the peripheral or central nervous systems in disarray, such as is seen in posttraumatic or postherpetic neuropathies.

Not all neuropathies are painful. When they are, they may be dramatically so. The distinguishing feature of peripheral neuropathic pains in the head and neck region is the quality of pain, that is burning, sharp, shooting, lancinating, or electric-like. The distribution of the pain is limited to the anatomical pathways of the nerve involved and is almost always unilateral. Sensory abnormalities may include diminished pain sensation in the presence of hypersensitivity to typically nonpainful stimuli.

In general, *neuropathic pains* in the head and neck can be divided into two main groups based on their temporal pattern, the *paroxysmal neuralgias* or *continuous neuropathies*

Some neuropathic pains are stimulus dependent, some occur spontaneously and are stimulus independent or both. Some neuropathic pains are accompanied by hyperalgesia and/or areas of numbness.

Paroxysmal Neuralgias

Symptoms. Paroxysmal, lancinating, sharp, unilateral pain that follows a distinct dermatomal pattern is common to all paroxysmal neuralgias. The pain is often described as electric-like, shooting, cutting, or stabbing. The attacks may last only a few seconds to minutes, with virtually no discomfort between attacks. Sometimes patients notice a vague prodrome of tingling and occasionally ache or burn after an attack. The attacks may occur intermittently, with days to months between a series of attacks. Usually, patients complain of "trigger areas" that, when stimulated, precipitate an attack. These are frequently located within the distribution of the nerve affected, usually on the skin or the oral mucosa. Neural blockade of the trigger area almost always relieves the pain for the duration of action of the local anesthetic. Should neural blockade fail to relieve the symptoms, either the diagnosis or the nerve block technique must be questioned. Each type of paroxysmal neuralgia has its own distinct characteristics.

Trigeminal Neuralgia and Pretrigeminal Neuralgia

Trigeminal neuralgia usually affects one or at most two divisions of the fifth cranial nerve, the maxillary and mandibular divisions being most common.[45] The maxillary or mandibular divisions alone are the next most frequently affected, with ophthalmic division neuralgias being the least common. The pain is unilateral 96% of the time. Touching and washing the face, tooth brushing, shaving, chewing, talking, or even cold wind against the face may set off the trigger and result in pain. Deep pressure or painful stimuli are usually tolerated without a painful episode. Patients go to extraordinary lengths, such as not shaving, washing, or brushing their teeth, to avoid stimulating the trigger area. Between attacks, patients are completely pain-free. Long remissions for months or years are not uncommon but tend to decrease with increasing age. Because the vast majority of trigeminal neuralgia cases involve the jaws, *dentists are commonly the first ones to see these patients*.

Trigeminal neuralgia is almost twice as common in women as in men and usually starts after the age of 50 and increases with age.[120] Anyone under the age of 40 with this disorder should be referred to a neurologist to be worked up for a structural lesion or multiple sclerosis.

Trigeminal neuralgia may rarely present comorbidly with any of the TACs, in which case the pain condition may be called cluster-tic or paroxysmal hemicranias-tic.[4] Certainly, SUNCT has several features that make it difficult to distinguish from trigeminal neuralgia and the two disorders do occasionally occur together.[45]

A syndrome of pain preceding the onset of true paroxysmal trigeminal neuralgia, known as *pretrigeminal neuralgia* (PTN), has been described.[121,122] Patients may present themselves up to 2 years before developing trigeminal neuralgia, with pain usually *localized to one alveolar quadrant* and sometimes a sinus. They may describe this pain as dull, aching, and/or burning, or as a sharp (burning) toothache, not unlike pain arising from the dental pulp. Some patients may also report a "pins and needles" sensation. The duration of PTN pain may be 2 hours to several months, with variable periods of remission. Movement, usually opening of the mouth, may trigger the pain.

Dental disease may be minimal or absent. Retained root tips are the most common finding, and their removal has been

associated with remission of pain. Obvious dental disease should be treated, but dental heroics in the absence of clinical or radiographic findings should be avoided. It was reported that 61% of the cases with PTN or trigeminal neuralgia were incorrectly diagnosed and treated for dental conditions.[123] Onset of classic trigeminal neuralgia may be quite sudden and occurs in the same division affected by the PTN symptoms. The onset may follow remission produced by previous dental treatment.

Glossopharyngeal Neuralgia

Glossopharyngeal neuralgia is 70–100 times **less** common than trigeminal neuralgia.[120] The symptoms include unilateral and 25% of the time nonconcurrent bilateral stabbing pain in the distribution of both the glossopharyngeal nerve (lateral posterior pharyngeal and tonsillar areas, the base of the tongue, down into the throat), and also the auricular and pharyngeal branches of the vagus nerve (the eustachian tube or ear, and down the neck).[45] Sometimes pain radiates into the vagal region and *may be associated with salivation or unilateral oral dryness*, flushing, sweating, tinnitus, cardiac arrhythmias, hypertension, vertigo, or syncope. *Throat movements*, pressure on the tragus of the ear, *yawning*, or *swallowing* may trigger the pain. Again, local anesthesia of the trigger area temporarily prevents precipitation of attacks. This can be accomplished by spraying the posterior pharynx with a topical anesthetic.

Eagle's syndrome may be the cause of symptoms similar to those of glossopharyngeal neuralgia,[124] although some believe that this syndrome is not sufficiently validated.[45] The symptoms that include a "sore throat" and *posterior tongue and pharyngeal pain* are thought to be related to the compression of the area of the glossopharyngeal nerve by a calcified elongation of the *styloid process* of the temporal bone. Precipitating factors include fast rotation of the head, swallowing, and pharyngeal motion from talking and chewing. Blurring of vision and vertigo are rarely seen.

Nervus Intermedius Neuralgia

Nervus intermedius neuralgia is extremely rare. The pain is often described as a lancinating "hot poker" in the ear. It may occur anterior to, posterior to, or on the pinna; in the auditory canal; or, occasionally, in *the soft palate*. A duller background pain may persist between attacks. Attacks may be accompanied by *salivation, tinnitus*, vertigo, or *dysgeusia*.[45,125] The trigger area is usually in the external auditory canal. This neuralgia has also been termed *Ramsay Hunt syndrome* or geniculate neuralgia, and is often associated with *herpes zoster* (or shingles). It may in some patients be due to a variant of glossopharyngeal neuralgia with pain in the ear.[45]

Superior Laryngeal Neuralgia

Superior laryngeal neuralgia is also a rare neuralgia with paroxysmal neuralgic pains of varying duration, minutes to hours, *located in the throat, submandibular region, or under the ear*. Triggering factors include *swallowing*, turning the head, loud vocalizations, or stimulating the site overlying the hypothyroid membrane where the nerve enters the laryngeal structures.

Occipital Neuralgia

Occipital neuralgia occurs in the distribution of the greater or lesser occipital nerves to the back of the head and mastoid process. The *ear and the underside of the mandible* may be involved because of the dermatomal patterns of C2 and C3 (Figure 17-12). The pain often radiates into the *frontal and temporal regions*, occasionally with the same sharp, electric-like character of the other neuralgias, but may last hours instead of seconds. Sometimes the pain takes on a more continuous burning, aching nature. A case of maxillary right posterior quadrant dental pain due to occipital neuralgia has been reported.[126] Trauma, especially rotational injuries to the neck, may precede the onset. Trigger zones, such as those seen with trigeminal neuralgia, are rare. The pain may be associated with neck and back pain, and emotional stress is a common aggravating factor.

Etiology. The paroxysmal neuralgias are considered *symptomatic* if a specific pathological process affecting the involved nerve can be identified and *classical* if not.[45] Paroxysmal neuralgias rarely occur in young people unless there is a distinct compression of the nerve by a tumor or other structural lesion. Compression of the nerve either peripherally or centrally by bone, scar tissue, tumors, aberrant arteries, or arteriovenous malformations causes *axonal loss and focal demyelination*, which is postulated to result in ectopic firing and reduced segmental inhibition of the low-threshold mechanoreceptors and wide-dynamic-range relay neurons.[127] The net effect is a *lowered threshold* of neuronal firing for which ordinary orofacial maneuvers such as chewing, swallowing, talking, or smiling may precipitate a neuralgic attack.

Trigeminal neuralgia, along with other paroxysmal cranial neuralgias, is typically considered idiopathic, although nerve compression by intracranial arteries that have become slack and tortuous with age is thought to be the likely culprit.[45] The *demyelinating* lesions associated with *multiple sclerosis* may also precipitate trigeminal neuralgia in younger individuals. The symptoms of trigeminal neuralgia associated with diabetic polyradiculopathy have also been reported.[128]

Glossopharyngeal neuralgia is more commonly, secondary to another pathologic disorder. For example tumors are found in 25% of cases. Local infection, neck trauma, elongation of the styloid process (Eagle's syndrome), and compression of the nerve root by a tortuous vertebral or posterior inferior cerebellar artery are other symptomatic causes of this ninth nerve neuralgia.[129] Unlike trigeminal neuralgia, glossopharyngeal neuralgia is almost never associated with multiple sclerosis,[130] and has been reported to be associated with an anomaly of the nerve itself.[131]

Occipital neuralgia may be secondary to hypertrophic fibrosis of subcutaneous tissue around the occipital nerve following trauma, irritation of the nerve by the atlantoaxial ligament, spondylosis of the upper cervical spine, spinal

cord tumors, or tubercular granulomas.[132] Myofascial TrPs in the semispinalis capitis and splenius cervicus muscles may mimic this type of pain or may cause neuralgia-like symptoms due to the entrapment of the greater occipital nerve as it passes through the tense semispinalis muscle fibers.[49] Since treatment of myofascial TrPs is noninvasive, with very low morbidity and no mortality, they must be carefully ruled out before neurectomy or other neuroablative techniques are considered.[133]

Examination. A patient presenting with symptoms of *trigeminal neuralgia* must be worked up for dental disorders, sinus disease, and head and neck infections or neoplasms. Since nerve compressions from intracranial tumors, aneurysms or vascular malformations, or central lesions from multiple sclerosis may also cause symptomatic trigeminal neuralgia, all patients with trigeminal neuralgia-like symptoms should be worked up with an *imaging study* of the head, either *contrast-enhanced CT or MRI or magnetic resonance tomographic angiography*, in addition to routine films.

Diagnosis of *PTN* is based on the clinical presentation of constant *dull toothache pain* in the absence of dental or neurological findings and normal radiographic, CT, or MRI examinations.

Since *glossopharyngeal neuralgia* is more often associated with nasopharyngeal, tonsillar, or posterior fossa tumors or other disease than is trigeminal neuralgia, imaging studies including skull films, panoramic films, and MRI are essential.

In *Eagle's syndrome*, the diagnosis is usually established on the basis of radiographs, as well as intraoral finger palpation of the posterior pharyngeal area. Panoramic films should reveal a *styloid process that is so long* that its image projects beyond the ramus of the mandible. Anything shorter is not significant, and other causes for the patient's pain should be sought. Pain is alleviated temporarily by neural block of the suspected compressive area. Treatment is primarily surgical shortening of the styloid process either through an intraoral or an extraoral approach.[124]

Occipital neuralgia requires a thorough musculoskeletal evaluation in addition to a cervical spine radiographic series to rule out neoplasms or other local destructive lesions. Digital palpation of the greater occipital nerve along the nuchal line may be painful. Caudal pressure to the vertex of the head while it is flexed and rotated toward the side of the pain may reproduce cervical compression symptoms.[132] Sensory loss is rare. Because myofascial TrPs may mimic or accompany this disorder, careful palpation of the posterior cervical muscles for the tight bands and focal tenderness with referred pain, characteristic of myofascial TrPs, is essential.[45,133]

Treatment. Obviously, if a neuralgia is "symptomatic" or the result of identifiable pathologic entity or structural lesion, treatment is directed at correction of the cause. However, for classical paroxysmal neuralgias, the first treatment of choice is the drug *carbamazepine (Tegretol)*. Carbamazepine and oxcarbazepine are most efficacious in trigeminal neuralgia and remain the first line of therapy.[134] These drugs have some success in glossopharyngeal or nervus intermedius neuralgias as well. Lamotrigine and baclofen (Lioresal) are the next most efficacious. Other medications such as topiramate, gabapentin (Neurontin), pregabalin (Lyrica), and Botox are also used, alone or in combination. All of these medications may cause varying degrees of dizziness, drowsiness, and mental confusion. In addition, carbamazepine may cause hematopoietic changes and baclofen may affect liver enzymes. Although such side effects are not as common as once thought and are less common with gabapentin and pregabalin, patients taking any of these medications must be monitored very closely initially.

Certainly, when medications become ineffective, or the patient cannot tolerate them, either due to severe drowsiness or frank allergy, neurosurgical intervention remains an option. As the success rates with neurosurgical interventions such as gamma knife, radiosurgery, percutaneous rhizotomies, and microvascular decompression improve and the morbidity goes down, surgical interventions are recommended earlier as more patients may benefit with fewer side effects.[135]

Therapies, especially medications, used to treat true trigeminal neuralgia have been reported to work for *PTN* as well.[136]

Classical *glossopharyngeal neuralgia* can be managed with the same medications and surgical techniques used for trigeminal neuralgia.[4]

Because of its predominantly musculoskeletal etiology, *occipital neuralgia* lends itself best to non-pharmacological and nonsurgical treatments. Consequently, physical therapy, postural reeducation, corrected ergonomics, home neck-stretching exercises, TrP injections, and even C2 nerve blocks are the first line of approach.[137]

Surgical section of the nervus intermedius or the chorda tympani has been reported to relieve the pain of *nervus intermedius neuralgia*[138] if management with anticonvulsants fails.

Neuromas

Neuromas are nonneoplastic, nonencapsulated, tangled masses of axons, Schwann cells, endoneurial cells, and perineurial cells in a dense collagenous matrix (Figure 17-14).

Etiology. Neuromas tend to develop when a nerve axon is transected, as might occur with a dental extraction, surgery, or trauma. The proximal stump of a transected nerve is still connected to its cell body and, in a few days postinjury, starts to sprout axons, in an attempt to reestablish continuity with its distal segment. Usually, this process is unsuccessful, especially in soft tissue; therefore, a tangled mass of tissue results.[139] In the orofacial region, neuromas most commonly develop in the area of the mental foramen, followed by the lower lip, tongue, and buccal mucosa, all easily traumatized sites. Neuromas are least likely to occur in the inferior alveolar canal because the bone guides the tissue growth.

Symptoms. A neuroma may be completely asymptomatic, and, in fact, approximately 25% of them are. However, neuromas are capable of generating very prolonged electrical impulses in response to a variety of stimuli, including touch.[139] The nerves in the trigeminal system have been

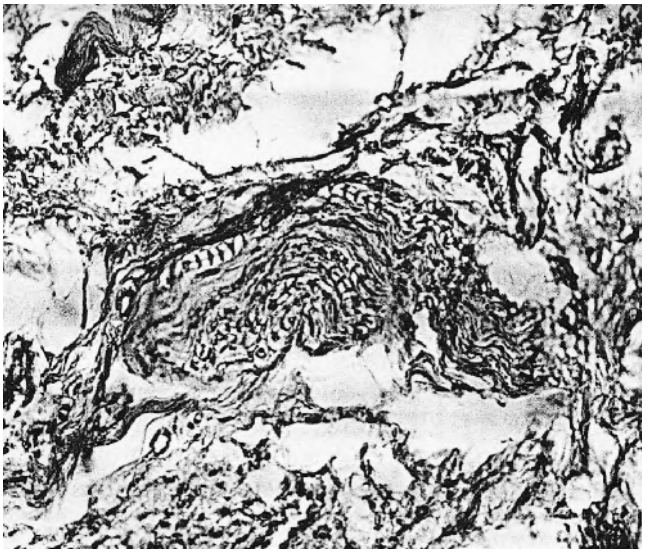

FIGURE 17-14 Amputation neuroma revealed by biopsy. Strikingly clear-cut, tangled, and well-myelinated fibers, cut at various angles, are massed with irregular sheath and perineural connective tissues into an abnormal aggragate of varying size and surrounded by well-vascularized fibrous tissue. Biopsy was occasioned by sharp, intermittent pricking "nerve pain" 6 months in duration, becoming more diffuse and steady. (Courtesy of Dr. Gordon Agnew, U.S.A.)

shown to be less sensitive to spontaneous or touch stimulated discharge than neuromas in other parts of the body.[139]

The diagnostic features of a neuroma include a history of prior surgery or a lacerating injury at the site of the pain, along with precipitation of the pain with local pressure or traction. The pain is short-lasting, with a burning, tingling, radiating quality. A drop of local anesthetic will abolish the response. If the neuroma has developed on the inferior alveolar nerve, it may be visible radiographically as a widening of the mandibular canal.

Neuromas may also occur in the *TMJ postsurgically* or postlacerating trauma.[140] Symptoms include restricted joint movement due to adhesions and pain with neuropathic qualities (sharp, shooting, itching, burning) on stretching of the adhesions. The deep aching pain of musculoskeletal disorders is lacking.

Examination. In the oral cavity, neuromas may appear as nodules of normal surface color but usually are not visible or palpable. Of greater interest is the presence of a scar, indicating previous injury or surgery. In the TMJ, movement against adhesive restrictions, as with jaw opening, should elicit short-lasting neuropathic pain.

Treatment. A one-time excision of a sensitive neuroma located in the accessible scar tissue is worth trying provided that the pain is localized and can be relieved by infiltration with a local anesthetic.[141] Unfortunately, neuroma excision and other peripheral neuroablative procedures provide significant relief in very few patients.[141] *Neuromas often re-form*, and the pain may return. Medications such as carbamazepine (Tegretol) or amitriptyline (Elavil) may be helpful but should be used only if the patient feels that the pain is severe enough to warrant tolerating the side effects that may accompany these medications.

Neck Tongue Syndrome. This is a very rare condition where rotation of the head causes occipital and cervical pain along with *paresthesias, dysesthesias, or anesthesia of the tongue or even a sensation of movement* on the same side as the neck pain.[45,142] The pain lasts seconds to 2 minutes.[142] Patients may have restricted range of motion of the neck and radicular symptoms into the arm.[142]

The proprioceptive fibers of the tongue run from the ansa hypoglossi to the C2 ventral ramus. During sudden rotation of the neck, especially when there is subluxation of the atlanto axial joint, stretching and/or compression of the second cervical root is thought to cause these symptoms.[143]

Continuous Neuropathies

Continuous trigeminal neuropathies typically occur after some kind of peripheral nerve damage or "deafferentation." This category includes postherpetic, posttraumatic, and postsurgical neuropathies, as well as anesthesia dolorosa. As with paroxysmal neuralgias, the continuous neuropathies follow the distribution of the damaged nerve but differ in that the pain is more or less continuous, with some fluctuation over time. Patients report altered sensations, dysesthesias, or pain in the distribution of the nerve that varies from tingling, numbness, and twitching to prickling or burning. They may also report "formication," which is a sensation of worms under the skin or ants crawling over the skin. The dysesthesias are generally discomforting to the patient because they are continuous and exacerbated by movement or touching of the area. As with paroxysmal neuralgias, local anesthetic blocks of the nerve eliminate all paresthesias except numbness.

Painful Trigeminal Neuropathy Due to Acute Herpes Zoster

Herpes zoster is a painful acute viral infection produced by the varicella zoster virus. Varicella zoster causes chickenpox in the young and afterward remains in the ganglia of sensory nerve endings in a dormant "provirus" form. Periodically, the virus reverts to its infectious state and is held in check by circulating antibodies. If an individual is immune-compromised or is older and has a low antibody titer, the infectious virus is able to retrace its path down the sensory nerve and escape into the skin, where it causes the typical skin eruptions and vesicles of zoster. Acute hemorrhagic inflammation with demyelination and axonal degeneration of dorsal root ganglion cells has been demonstrated in early[144] and more recent[145] pathological studies.

The incidence of acute herpes zoster outbreaks has diminished dramatically since the varicella vaccine became available in 1995, reducing the acute disease by 75%.[146] Without the vaccine, acute herpes zoster affected individuals over 70 years of age 12 times more frequently than persons under 10 presumably because their antibody titer decreased as their exposure to children with chickenpox diminished. The older the individual, the more severe the rash and the acute pain,

and, the more severe the prodromal pain, the higher the likelihood that that person will develop postherpetic neuropathy (PHN).[147]

Symptoms. Continuous pain in the distribution of the affected nerve and malaise commonly precedes the eruption of *acute herpes zoster* by up to 7 days,[45] sometimes along with paresthesia and shooting pains. Vesicles form, become infected, scab over, and heal. Small and sometimes severe scars are left behind. The scars are usually anesthetic, the skin between being hyperesthetic.

Ten percent to 15% of patients with herpes zoster develop unilateral lesions in the head and neck region, and 80% of these are in the ophthalmic division of the trigeminal nerve (Figure 17-15). C1-3 are also sometimes affected. When the first division of cranial nerve V is affected, vesicles may appear on the cornea, and the risk of impaired vision is high. If the infection affects the *maxillary division of the trigeminal nerve*, intraoral eruptions may develop concomitantly with those on the skin. Involvement of the oral mucosa without cutaneous involvement may occur and must be differentiated from aphthous stomatitis. Multiple devitalized teeth, isolated in a single quadrant, have been reported as a rare complication of herpes zoster infection.[148]

If the *facial nerve (cranial nerve VII)* is involved, facial palsy may occur due to pressure on the nerve from inflammatory swelling in the bony canal. The pupil may become permanently paralyzed, and the upper eyelid may droop. Involvement of the geniculate ganglion causes lesions to erupt in the external auditory canal, may cause acoustic symptoms, and is known as Ramsey Hunt syndrome.[45]

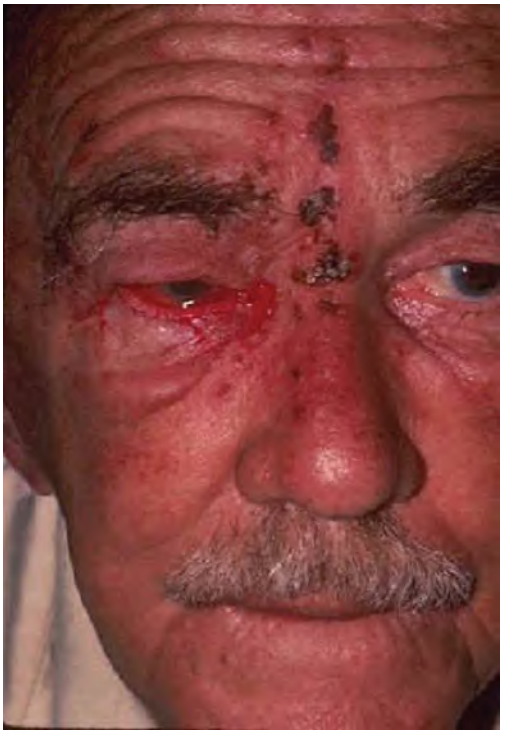

FIGURE 17-15 Herpes zoster infection of the first division of the trigeminal cranial nerve. (Courtesy of Faculty of Medicine, University of Toronto, Canada.)

Treatment. Treatment of herpes zoster is primarily focused on pain relief, reducing the rash, and reducing the risk of complications such as local infection, spread to other areas, and development of postherpeticneuropathy. The medications of choice are the antiviral drugs valaciclovir and famciclovir, which both performed equally for resolution of the lesions and pain control.[149] Newer medications are being tested and appear to offer improvements such as reduced dosing, fewer side effects, and improved effectiveness.[150] Systemic steroids help reduce the clinical symptoms significantly if started within 3 days of eruption of vesicles, but have little impact on the development of PHN.[151]

Most important is the control of pain. This can be achieved with over the counter analgesics such as acetaminophen. Amitriptyline has been shown to provide pain relief and shorten the duration of the illness. The problem is that older individuals may have difficulty taking amitryptiline due to the cardiac effects. Nortriptyline or desipramine may be better choices.

Postherpetic Neuropathy (PHN)

PHN may follow an acute attack within 3 months of *herpes zoster* ("shingles") in up to 22% of people who get the acute disease.

PHN results when the pain of acute zoster does not subside as the acute eruption clears. Risk factors include increased age, the severity of the prodromal pain of the acute zoster, the severity of the pain with the acute zoster, and the severity of the rash.[152]

The pain of PHN often has several distinct components. Patients complain of a steady, deep, aching, burning pain along with superimposed sharp, stabbing pains similar to trigeminal neuralgia. In addition, many complain of *allodynia*, pain in response to light brushing of the skin, an innocuous event under normal circumstances. "Postherpetic itch" occurs in 30–50% of patients and, despite a mild to moderate intensity, it rated as more bothersome than pain.[153] The pain is limited to the distribution of the affected nerve, and there is usually cutaneous scarring and sensory loss.[148] Complaints of *formication* are common. Since this pain is severe and unrelenting, it places a large emotional burden on the elderly patients who suffer from it causing interruption of sleep, drug reliance, depression, and even contemplation of suicide.

Examination. The history of acute zoster infection and the obvious scars it leaves behind, including pale, red or purplish skin discoloration, make diagnosis relatively simple in most cases. PHN, on the other hand, may occur up to 3 months after the vesicular stage of herpes zoster has healed. If the patient knows he has had herpes zoster infection, the diagnosis will be self-evident. The dentist, however, should carefully check the mouth to ascertain that a concomitant severe pulpitis is not superimposed on the condition and perform temperature and pinprick evaluations.

Difficulty in diagnosis arises when the severe vesicular herpetic attack is not manifested but rather one or two small

aphthous ulcers appear in the oral cavity or on the lips or face. This mild attack is often forgotten by the patient, and a careful history is essential.[154]

Treatment. Although age is the most important risk factor for the development of this painful chronic disorder, early treatment during the *acute phase of herpes zoster* with antiviral agents or certain tricyclic antidepressants such as amitriptyline combined with psychosocial support, may be effective in preventing the development of PHN.[155] Tricyclic antidepressants, gabapentin, and opioids are the only medications that have shown efficacy in randomized clinical trials.[153,156] Although sympathetic nerve blocks do tend to reduce acute herpetic pain, they have not been proven to be effective in preventing PHN.[148,157]

The treatment of PHN is often difficult and unrewarding. The longer the infection continues and the longer the patient has PHN, the more difficult pain management becomes. *Amelioration of the depression* that invariably accompanies this condition is as significant in therapy as reduction of the primary pain.

Painful Traumatic Trigeminal Neuropathies (PTTNs)

Traumatic injury to or disease of the peripheral nerves often results in sensory changes qualitatively different from PHN that are sometimes accompanied by pain. The most common dental causes of painful traumatic trigeminal neuropathy (PTTN) are dental implants, third molar extractions, nerve blocks and root canal therapy.

Anesthesia dolorosa is an extreme form of PTTN that occurs after a deliberate or iatrogenic lesion of one of the branches the trigeminal nerve or the occipital nerve. There is "persistent and painful anesthesia or dysesthesia within the area of the distribution of [the damaged nerve]," accompanied by diminished sensation to pinprick and sometimes other sensory loss.[45]

Dental implants may cause direct trauma to a major nerve (usually the mandibular nerve) or irritation of smaller nerve fibers (in the maxilla) immediately upon placement. Sometimes dental implants cause no pain until the crown is cemented and they are loaded.[158] The pain after crown placement may be due to extensive peri-implant innervation.[159]

Third molar extractions may be followed by short-term hypoesthesia, which improves in most patients.[160] Unless there is iatrogenic damage to the mandibular nerve during surgery, there appears to be little risk of neuropathic pain.[161] However with iatrogenic damage, pain occurred in 70% of cases.[162]

Root canal treatment is accompanied by about a 3–13% incidence of persistent pain.[163] Surgical endodontic procedures were associated with painful trigeminal neuropathy in 5% of cases.[164]

Symptoms of PTTN. The pain is described as a continuous tingling, numb, twitching, or prickly sensation, sometimes with burning or shooting triggered by touch or movement. The pain occurs unilaterally along the injured nerve and rarely spreads to another dermatome. Patients may describe sensations of hot, cold, swelling, local redness, or flushing. There is significant allodynia and electric pains may be precipitated with percussion over the nerve.

Pathophysiology of PTTN can be explained in part by several of the pain phenomena described at the beginning of this chapter, namely peripheral sensitization from tissue injury and inflammation, central sensitization, glial cell activation, and ectopic activity from nerve injury.

Treatment. The best treatment for traumatic trigeminal neuropathies is prevention. To this end, preventive analgesia, the use of preventive interventions such as preoperative opioids, perioperative gabapentin and/or pregabalin, or local anesthetic blocks during surgery have been studied and while they seem to reduce postoperative pain, there is no solid data to indicate that they prevent the emergence of persistent pain.[4] There is interest in developing screening tools to identify patients at higher risk of developing neuropathy after surgery as there is evidence that environmental, psychosocial, and genetic factors play a role.[165]

Treatment of established PTTN may be met with varying success and can include any of the pharmacological therapies used for the other neuralgias as well as acupuncture, transcutaneous electrical nerve stimulation. Cognitive behavioral therapy to combat comorbid anxiety and depression and address quality of life issues was not shown to be effective.[166]

Surgical therapies to repair damaged nerves do improve the level of sensation,[167] and the success has been good for painful neuropathies where nerve repair was aimed at clear sources for the neuropathy such as scar tissue removal or relief of compression (dental implant removal) or neuroma excision.[168] Central procedures have not been systematically studied but may be useful in intractable cases.[4]

"CAUSALGIC PAIN" AKA CHRONIC REGIONAL PAIN SYNDROME (CRPS)

Causalgia is a word derived from the Greek words *kausos*, meaning "heat," and *algia*, meaning "pain." Causalgia was first described by Mitchell in 1864 as a syndrome of burning pain that appeared in the extremities following partial injury to a major peripheral nerve by a high-velocity missile trauma.[169] In 1947, Evans[170] used the term reflex sympathetic dystrophy (RSD) to describe the same burning pain. Evans noted that the pain had many features of sympathetic stimulation such as redness, swelling, sweating, and atrophic changes in the skin, muscles, and bones. He also found that minor injuries, such as fractures or sprains, could precipitate this pain, not just major nerve trauma. That same year, Bingham published the first report of RSD of the face.[171] Since then additional publications, but relatively few, have documented this pain syndrome in the face.[172–176]

In the early 1990s, this pain disorder was renamed "*complex regional pain syndrome (CRPS)*" *type I and type II*: Type 1 for those pains after minor injuries (RSD) and Type II for pain after major injuries (causalgia), although the distinction seems to be unnecessary.

In 1994, the IASP came up with research diagnostic criteria that had excellent sensitivity but low specificity.[177,178] Additional clinical diagnostic criteria were added to the IASP diagnostic criteria ("the Budapest Criteria") in order to improve specificity for research purposes.[179] An additional subtype was added CRPS- not otherwise specified (NOS) in order to account for cases that did not meet all of the typical criteria.

Symptoms. The characteristic pain is a *continuous, deep, hot, burning ache* with painful cutaneous hypersensitivity (allodynia) and hyperalgesia that is disproportionate to the inciting injury and *spreads nondermatomally beyond the injury site*. The pain is present at rest and aggravated by light touch, heat, cold, movement, or emotional stress.

Typically, *CRPS* begins a few days to several weeks after an injury. In the head and neck region, CRPS/RSD has been reported to develop after maxillofacial surgery for cancer, head injury, *molar extraction*, and sinus surgery.[172]

In the limbs, undiagnosed or untreated CRPS passes through an initial or "traumatic" stage characterized by a burning ache, edema, and hyperthermia to a second or "dystrophic" stage, with cool, cyanotic skin, spreading pain, and edema. The third or "atrophic" stage is evidenced by muscle atrophy, osteopenia or osteoporosis, smooth, glossy, mottled-appearing skin, and intractable pain. However, because of the abundant collateral blood circulation in the head and neck region, the bony, vascular, and trophic changes so typical in the extremities are less common in the face.[172] This is also probably why CRPS is recognized so infrequently in the head and neck region. In the absence of sympathetic changes, CRPS of the face resembles PTTN, except that the pain spreads beyond the expected dermatome based on the site of the insult or trauma.

Etiology. Similar to PTTN, CRPS may be due to sensitization of the primary afferent nociceptors and spinal tract neurons. In addition, disinhibition of central nociceptive neurons and reorganization of thalamocortical somatosensory maps seem to play a role.[4,180]

Clinically, the sympathetic nervous system is implicated in these pains[181] but while sympathetic blockade provides profound relief in some cases, it does not in others.[182]

Examination. The patient with CRPS complains of pain at the slightest touch and is difficult to examine. The skin may be flushed and dry or cold and sweaty, and a temperature difference may be discernible.

Diagnostic tests. Definitive diagnosis of the condition in the head and neck may be accomplished through immediate reduction of pain and hyperesthesia with a successful local anesthetic block of the stellate ganglion, although systematic review of the literature has shown some mixed results.[182] This procedure requires a trained anesthesiologist since the stellate ganglion is located in close proximity to several vital structures, including the vertebral artery and the apex of the lung.

Treatment. If the stellate ganglion block was successful in eliminating or significantly reducing the pain, repeated sympathetic blocks are the treatment of choice.[172] Early intervention is best. Oral medications such as steroids, low-dose tricyclic antidepressants with or without an anticonvulsant may be a useful adjunct or alternative.[183] Biofeedback and relaxation techniques may help reduce generalized sympathetic activity. Transcutaneous electrical nerve stimulation or acupuncture in combination with physical therapy may be helpful in providing relief and increasing function.

When dealing with any neuropathic or sympathetically maintained pain, referral to a neurologist should be considered if there are any subjective complaints or objective findings of cranial nerve deficits such as areas of facial hypesthesia, persistent motor weakness or paralysis, or a depressed corneal reflex.

MUSCLE PAINS

Pain of muscular origin is generally described as a continuous, deep, dull ache or as tightness or pressure. Once dental pathology has been ruled out, it is undoubtedly the most prevalent cause of pain in the head and neck region.[50,184] The most common muscle disorder to be familiar with is myofascial pain (MFP).

Local muscle soreness or "*noninflammatory myalgia*" is usually an acute process seen in the dental office from protective co-contraction of muscles secondary to an inflammatory or infectious process such as pericoronitis or other source of deep pain or a heavy occlusal contact after crown placement, that responds well to correction of the inciting factor.[185]

Myospasm, an involuntary CNS induced continuous contraction of a muscle or group of muscles, is less common in the jaws than previously thought.[185] When encountered, it is also usually associated with deep pain input from other sources such as joint inflammation, or dental infection, although fatigue or overuse of the muscle or idiopathic mechanisms may also be involved.[186,187] Treatment involves reducing the pain with massage, vapocoolant spray, ice, or local anesthetic injection (plain, NO vasoconstrictor!) and gently stretching passively to achieve full muscle length. Elimination of the causative factor, if present, will prevent recurrence.

Myofascial Pain (MFP)

MFP, a regional referred pain syndrome associated with focally tender TrPs in muscle, is a prevalent cause of pain in all parts of the body and has been reported as a common source of pain in numerous medical specialties.[49] For example, almost 30% of patients presenting themselves with a complaint of pain in an internal medicine practice[51] and over 80% of patients in a chronic pain center[188] had MFP as a primary diagnosis. It is also the most prevalent cause of painful symptoms in TMDs.[50]

Symptoms. In *MFP*, the presenting pain complaint is usually a *referred symptom* with a *deep, dull aching quality* located in or about normal muscular or, importantly, nonmuscular structures. In the head and neck region, the patient may complain of such things as *toothache*, sinus pain, *TMJ pain*, or

headache, yet evaluation of these areas or structures does not reveal any pathological change. In fact, *any undiagnosed deep, dull, aching pain may be myofascial in origin*. The intensity of MFP should not be underestimated: the pain intensity has been documented to be equal or slightly greater than pain from other causes.[51]

Associated symptoms, thought to be due to the physiological sensory, motor, and autonomic effects seen with prolonged pain, are common and may confuse the clinical picture.[49,189] Associated sensory complaints may include tenderness in the referred pain site, such as scalp pain on brushing the hair, or *abnormal sensitivity of the teeth*.[190,191] Motor effects include increased electromyographic (EMG) activity in the pain reference zone, although patients rarely complain about this specifically.[189,190] Autonomic changes such as localized vasoconstriction (pallor), sweating, lacrimation, coryza, increased salivation, and nausea and vomiting have also been reported.[49,190,192]

Patients note worsening of symptoms with increased psychological stress, cold weather, immobility, and overuse of involved muscles. Hot baths, rest, warm weather, and massage are typical alleviating factors.

Etiology. Myofascial TrPs tend to develop with protective muscle splinting after acute trauma, with chronic muscle overload, secondary to other chronically painful conditions, and secondary to referred pain from other TrPs. They can be activated by additional acute muscle overload, and also through psychological stress.

Acute trauma is easily identified and includes injuries such as those caused by falls, blows, sports injuries, motor vehicle accidents, excessive or unusual exercise, or even *prolonged jaw opening* at the dental office.

Chronic muscle overload is more insidious and includes long-term muscle overuse due to poor posture and body mechanics, abnormal strain, and repetitive motion-type injuries.

Secondary myofascial TrPs develop in response to prolonged underlying disease, especially if painful (migraine, persistent pulpalgia, post herpetic neuropathy) since any process that activates nociceptors may induce motor effects and TrP development. Secondary MFP may prolong and complicate pain due to other causes. MFP needs to be identified and treated to reduce pain and improve response to other therapies. Patients with what appears to be primary MFP, in whom TrPs and pain recur despite appropriate initial response to TrP therapies and compliance to home exercise, must be reevaluated for occult underlying disease or other perpetuating factors.

Secondary or *"satellite" TrPs* develop in muscles in the site of referred pain from referred motor activity.[189] These satellite TrPs are extremely common and may cause their own referred pain pattern, further confusing the clinical picture. For example, upper anterior toothache may be due to referred pain from a temporalis muscle TrP but if treatment of that TrPs only provides temporary relief, there may be a "key" TrP in the upper trapezius muscle and definitive resolution of the myofascial referred toothache will require inactivation of the trapezius trigger point.

In MFP, the presenting pain complaint is always associated with a TrP located in a taut band of skeletal muscle that is often distant from the site of pain. TrPs are focally tender, firm, nodular areas in muscle that, with appropriate palpation, produce spontaneous referred pain or intensify existing pain in local or distant locations.[193–195] Patients are typically unaware of the existence of these TrPs. TrPs are considered active when the referred pain pattern and associated symptoms are clinically present and latent when they are not. TrPs will vacillate between active and latent states depending on the amount of psychological stress the individual is under and the amount of muscle overload placed on the affected muscle.

The location of TrPs and their associated referred pain patterns are predictable and reproducible from patient to patient[49] (Figures 17-16 to 17-19). A meticulous discussion of MFP, as well as a complete compendium of the pain referral patterns for most muscles of the body, has been brilliantly detailed by Travell and Simons.[49,196]

Pathophysiology. With some training, myofascial TrPs are relatively easy to palpate. Despite the comparative ease of clinical identification of TrPs, controversy still exists about their structure, exact pathophysiology and the role of the central nervous system. Suffice it to say that research over the last 25 years has led to the conclusion that MFP may be

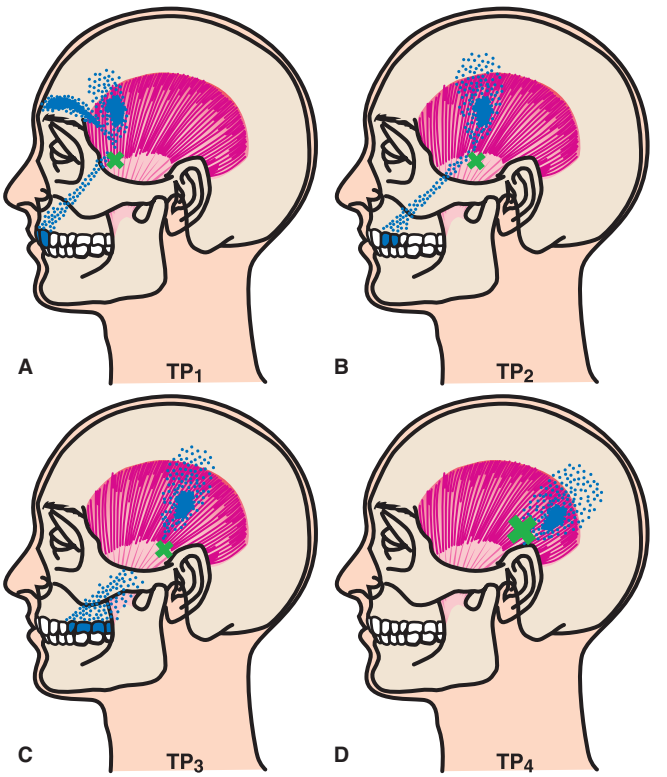

FIGURE 17-16 Referred pain patterns from trigger points (green "x"s) in the temporalis muscle (main referral zone, blue, spillover zone, stippled blue). ***A.*** Note referral to anterior teeth from anterior TrP labeled TP1. ***B,C.*** TP2 and TP3 refer to the maxillary posterior teeth, sinus, and zygomatic area. ***D.*** Posterior TrP, TP4, refers to the supra-auricular area. (Reproduced with permission from Travell JG, Simon DG, et al, 1999.[49])

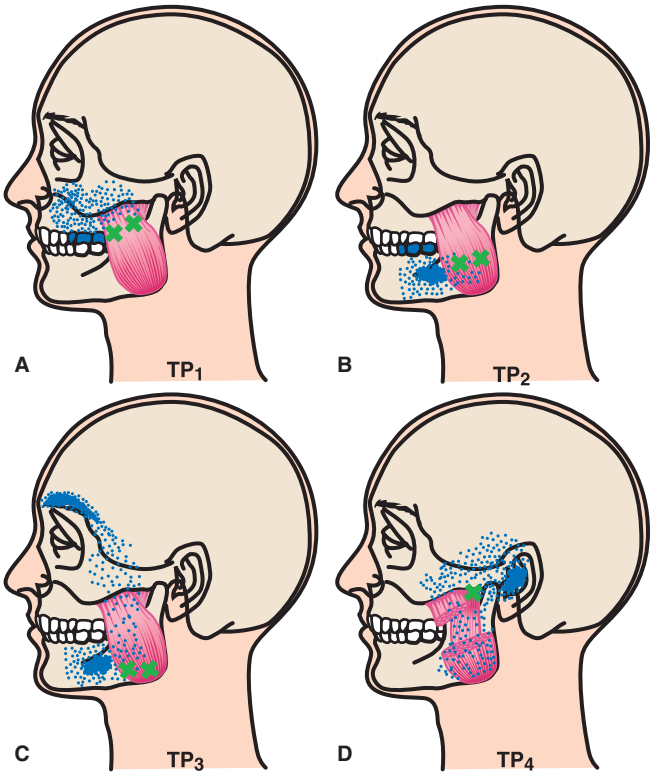

FIGURE 17-17 Referred pain from TrPs in the masseter muscle. The green "x"s locate TrPs in various parts of the masseter; the blue areas show main referred pain zones and stippled areas are spillover pain zones. ***A.*** Superficial layer, upper portion. Note referral to maxillary posterior teeth. ***B.*** Superficial layer, midbelly. Note spillover reference to the body of the mandible and posterior teeth. ***C.*** Superficial layer; lower portion refers to the body of the mandible and over the eye producing frontal "headache" symptoms ***D.*** Deep layer; upper part refers to the temporomandibular joint area and ear and may cause tinnitus. (Reproduced with permission from Travell JG, Simons DG, et al, 1999.[49])

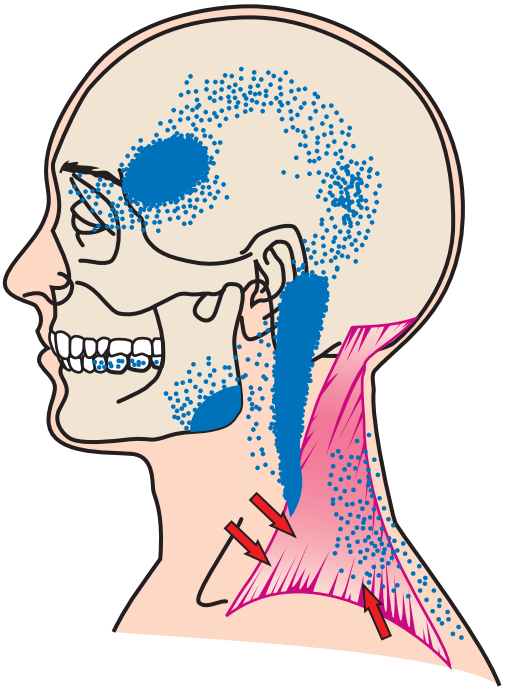

FIGURE 17-18 Composite pain referral patterns from TrPs in the upper trapezius muscle, suprascapular region. TrPs are indicated by red arrows, the main pain reference zones by solid blue. Spillover pain zones are stippled. Note temporal "tension headache" pattern and referral to the angle of the mandible with spillover to the mandibular posterior teeth. (Reproduced with permission from Travell JG. J Prosthet Dent 1960;10:745.)

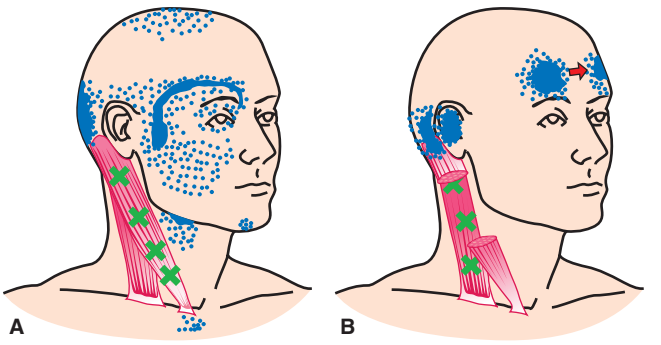

FIGURE 17-19 Pain referral patterns from the sternocleidomastoid muscle. TrPs (green "x"s), main referral zones (blue), and spillover areas (stippled). ***A.*** The sternal division. ***B.*** The clavicular (deep) division refers the pain of frontal "tension headaches." The referred pain to the forehead may cross the midline (red arrow). (Reproduced with permission from Travell JG, Simons DG, et al, 1999.[49])

a complex form of neuromuscular dysfunction where neurogenic inflammation, wide dynamic range neurons, and limbic system structures contribute to muscle sensitization, somatovisceral interactions, the development of chronic pain and the objective physical findings of allodynia, hyperalgesia, and referred pain.[128,197–199]

Simons' integrated trigger point hypothesis, namely that the myofascial TrP represents an area of isolated sustained muscle contraction due to excess acetylcholine release, is still considered the most credible theory to date.[197] Isolated sustained muscle contraction would result in uncontrolled metabolism, and localized ischemia that is initiated by acute muscle injury or strain.[190] This theory does provide a credible explanation for the palpable nodules and taut muscle bands associated with TrPs. The TrP nodule is described as a group of "contraction knots" in which a number of individual muscle fibers are maximally contracted at the end-plate zone, making them shorter and wider at that point than the noncontracted neighboring fibers. If enough fibers are so activated, a *palpable nodule* could result. As for the taut band, both ends of these affected muscle fibers would be maximally stretched out and "taut," producing the palpable taut band (Figure 17-20). This theory overlaps with the "Cinderella hypothesis" proposed by Hagg[186] where the idea is that TrPs could develop in response to muscle recruitment patterns during submaximal level exertions with moderate or low physical load, not unlike those encountered with computer work or typing.[187]

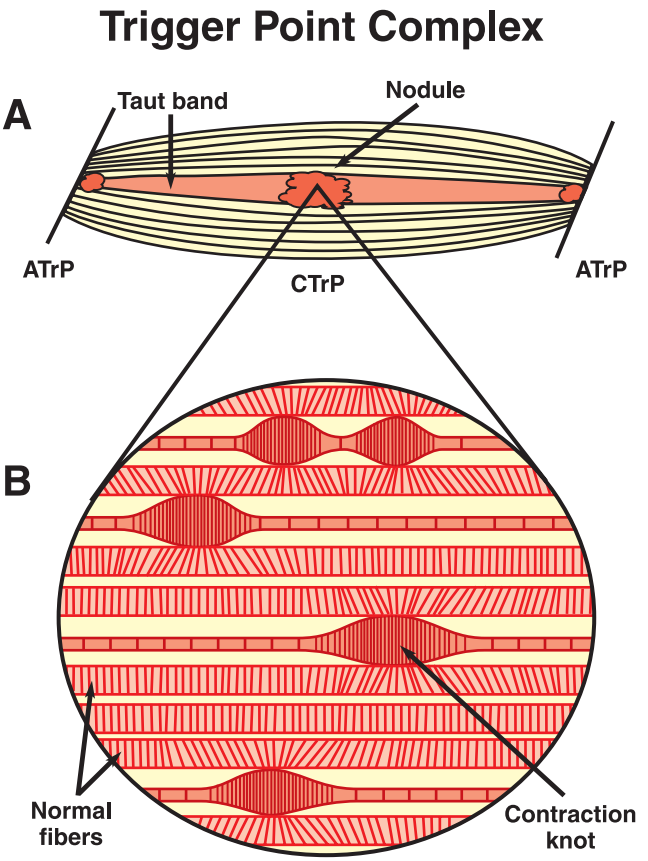

FIGURE 17-20 Schematic conceptualization of a trigger point (TrP) in a longitudinal section of muscle. ***A.*** The central TrP (CTrP), which is found in the muscle where the motor nerve axons enter (end-plate zone), is clinically tender to palpation. This area contains numerous "contraction knots," maximally contracted individual muscle fibers that are shorter and wider at the motor end plate than the noncontracted neighboring fibers. ***B.*** Enlarged view of the central TrP showing the distribution of five contraction knots. The vertical lines in each muscle fiber identify the relative spacing of its sarcomeres, the sarcomeres within the contraction knots experiencing maximal contracture and being markedly shorter and wider than the sarcomere of the neighboring normal muscle fibers. In fibers with these contraction knots (note the lower three individual knots), the sarcomeres on the part of the muscle fiber that extends beyond both ends of the contraction knot are elongated and narrow compared with normal sarcomeres. In patients, the central TrP would feel nodular as compared to the adjacent muscle tissue because it contains numerous "swollen" contraction knots that take up additional space and are much more firm and tense than uninvolved muscle fibers. If enough fibers are so activated, a palpable nodule could result. As for the taut band, both ends of these affected muscle fibers would be maximally stretched out and "taut," producing the palpable taut band. (Reproduced with permission from Travell JG, Simons DG, et al, 1999.[49])

The mechanism of referred pain from myofascial TrPs is also under speculation. According to Mense[200] and Vecchiet et al.,[201] the convergence-projection and convergence-facilitation models of referred pain (described earlier under section "Referred Pain") do not directly apply to muscle pain because there is little convergence of neurons from deep tissues in the dorsal horn.[200] These authors propose that convergent connections from other spinal cord segments are "unmasked" or opened by nociceptive input from skeletal muscle and that referral to other myotomes is due to the release and spread of substance P to adjacent spinal segments[200] (Figure 17-21A). Simons[190] has expanded on this theory to specifically explain the referred pain from TrPs (Figure 17-21B).

Examination. Because the patient's pain complaint is typically a referred symptom, it is usually distant from the muscle containing the guilty TrP,[49] although some TrPs also cause more localized pain symptoms. Multiple TrPs can produce overlapping areas of referred pain (Figure 17-22). The pattern of pain can be used in reverse to identify possible etiological TrPs.

Systematic fingertip or pincer type examination of suspected muscles and their contralateral counterparts, looking for taut bands and focal tenderness, is *essential* (Figure 17-23A–C). Effective TrP palpation is a skill that must be learned and practiced. Depending on the muscle, the tip of the index finger should be used for flat palpation or the index finger and the thumb for pincer-type palpation.[49] Once a TrP is found, algometry studies support the application of 2 to 3 kg/cm^2 of pressure to elicit pain.[193–195] The recent DC/TMD diagnostic criteria recommend 1-1.5kg/cm^2 of pressure.[195] The pressure should be applied for at least 5-10 seconds to elicit the referred pain pattern, if any. The examination may replicate the patient's pain so precisely that there is no doubt about the diagnosis. If uncertainty exists, specific TrP therapies, such as "spray and stretch" or TrP injections, described in the following, may be used diagnostically.

All head and neck muscles should be routinely examined in patients with a persistent pain complaint. This will help identify both primary and secondary MFP in addition to help identify TrPs in key muscles (e.g., upper trapezius or sternocleidomastoid) that may be inducing or perpetuating satellite TrPs in muscles located in the pain referral sites (e.g., temporalis).[49,189]

Treatment. Recommended treatment of MFP involves most importantly identification and control of causal and perpetuating factors, patient education, and specific home stretching exercises. Therapeutic techniques such as "spray and stretch," (Figure 17-24), voluntary contract release, TrP pressure release, and TrP injections are useful adjunctive techniques that usually facilitate the patient's recovery

Perpetuating factors most commonly include mechanical factors that place an increased load on the muscles. Teaching patients good posture and body mechanics will go a long way in reducing referred pain from myofascial TrPs, especially in the head and neck region.[187,202] An *intraoral stabilization appliance* may be indicated if the patient clenches or bruxes their teeth at night.[4,59,185]

Psychological factors, such as *stress* that has been shown to cause TrP activation,[203] or *depression* that lowers pain thresholds, will contribute to MFP. Sleep disturbance and general inactivity are also common perpetuating factors. Simple stress management and relaxation skills are invaluable in

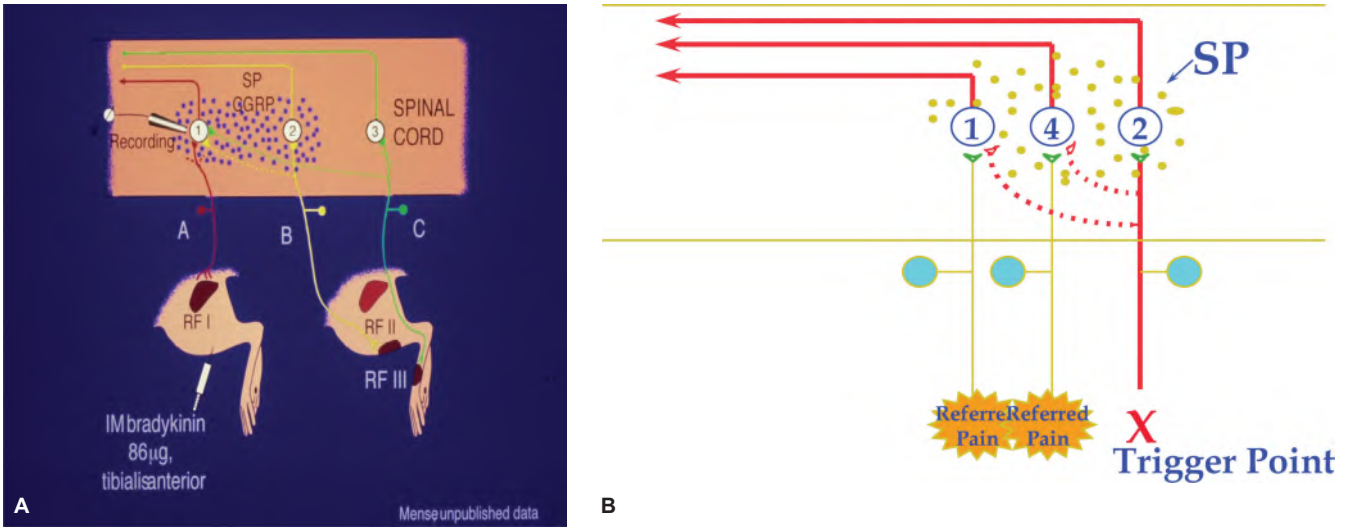

FIGURE 17-21 ***A.*** Neuroanatomic model explaining the appearance of new receptive fields (RFs) by unmasking latent connections in the dorsal horn. The activity of the second order pain transmission neuron #1 was recorded with a microelectrode introduced into the spinal cord. This neuron was connected by pathway A to its original RF I in the biceps femoris muscle. Solid lines represent synaptically effective connections, dashed lines ineffective or latent connections. The injection of pain inducing bradykinin was made outside RF I into the RF of neuron 2 (RF II). The bradykinin-induced excitation of nociceptive fibers of pathway B is assumed to release substance P (SP) and calcitonin gene-related peptide (CGRP) in the dorsal horn, which diffuse to neuron #1 (denoted by stippling) and increase the efficacy of latent connections from pathways B and C to this cell. Within 5–15 minutes neuron #1 could also be activated by stimulation of RF II and RF III. ***B.*** Extension of Mense's model of deep referred pain to explain some TrP characteristics not accounted for by his model. No direct experimental evidence substantiates this modification. Second order pain transmission neurons #1 and #2 correspond to neurons #1 and #2 in the Mense model. Neurons #1 and #4 are connected by yellow lines to their respective RFs. These RFs are the areas that would be identified as the source of nociception when neurons #1 and #4 are activated. Nociceptive input from the TrP would activate second order pain transmission neuron #2 and could account for the initial localized pain in response to pressure applied to the TrP. This activity is assumed to release SP and CGRP in the dorsal horn that diffuses (stippling) to neurons #1 and #4. This increases the efficacy of latent connections (dashed red lines) to these cells. Now neurons #1 and #4 can be activated by nociceptive activity originating in the TrP and would be perceived as referred pain. (Figure 17-21A reproduced with permission from Mense S, 1993.[201] Figure 17-21B reproduced with permission from Simons DG, 1993.[191, 201])

controlling involuntary muscle tension if this is a problem.[204] Mild depression and associated sleep disturbance can be treated with low doses of *tricyclic antidepressant drugs* and a structured exercise/ activation program.

Other perpetuating factors include metabolic, endocrine, or nutritional inadequacies that affect muscle metabolism.[49,205] Patients should be screened for general good health and referred to their physician for management of any systemic abnormalities. In secondary MFP, the primary causative painful disorder, such as TMJ capsulitis, pulpitis, or chronic neuropathy, must also be treated or managed.

Spray and stretch is a highly successful adjunct for the management of myofascial TrPs that uses a vapocoolant spray (Gebauer, Co., Cleveland, OH) to facilitate muscle stretching. Muscle stretching has been shown to reduce the intensity of referred pain and TrP sensitivity in patients with MFP.[206] The vapocoolant is applied slowly in a systematic pattern over the muscle being stretched and into the pain reference zone (Figure 17-24). This technique and alternatives not using a vapocoolant are described in detail elsewhere.[49] Long-term improvement is dependent on **control of the perpetuating factors** that overload the affected muscles.

Needling of TrPs with or without injection of solution has also been shown to be helpful in reducing the TrP activity to allow stretching.[207,208] Impaling the TrP and eliciting a twitch response are critical for a superior clinical effect.[208] Dry needling or injection of "key" myofascial TrPs has been shown to reduce the activity and tenderness in related satellite TrPs.[189] Although dry needling is effective, the use of a local anesthetic reduces postinjection soreness.[208] If a local anesthetic is to be injected, 0.5% procaine or 0.5% lidocaine is recommended. Longer-acting amide local anesthetics or local anesthetics containing epinephrine cause permanent muscle damage.[209] Injection must always be followed by stretch.[49] Trigger point injection in the absence of a home program that addresses relevant perpetuating factors will provide only temporary relief. Randomized controlled studies, looking at the efficacy of botulinum toxin in comparison to dry needling, saline or other placebo solutions in TrP injections, have shown that overall Botox is no more effective than dry needling or other solutions.[210,211]

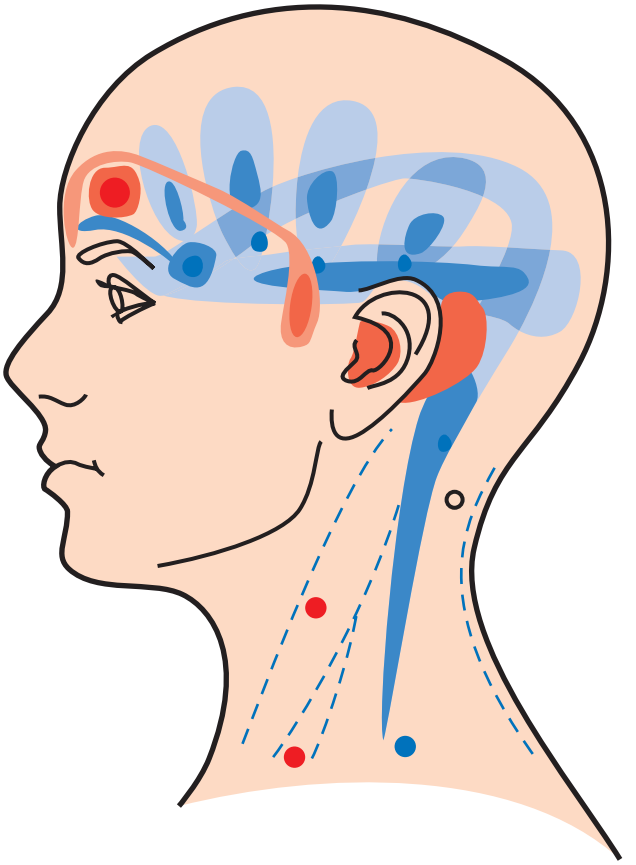

FIGURE 17-22 Overlapping pain referral patterns from the temporalis, sternocleidomastoid, upper trapezius, and suboccipital muscles produce a clinical picture of "tension-type headache." Unilateral symptoms may mimic migraine without aura. (Reproduced with permission and courtesy of Dr. Bernadette Jaeger, Los Angeles, CA, U.S.A.)

Tension-type headaches. *Tension-type headaches* are usually bilateral, with a pressing, non-pulsating quality. They last 20 minutes to 7 days when episodic and may be daily without remission when chronic.[45] Overlapping referred pain patterns from myofascial TrPs can produce a typical tension-type headache (see Figure 17-22). Studies have documented the presence of focally tender points and referred pain in this type of headache[212–214] Others have postulated that MFP and tension-type headaches may share a common underlying pathophysiology.[4] Thus, the same treatment strategies used for MFP also work well for the reduction of tension-type headache.[213,215]

PERSISTENT IDIOPATHIC FACIAL PAIN

Phantom tooth pain, atypical odontalgia, and atypical facial pain are terms used in the literature that fall under the current IHS category of *Persistent idiopathic facial pain*, dull, aching, mostly unilateral burning or throbbing pain that recurs daily for 2 or more hours for more than 3 months, may have sharp or stabbing exacerbations, is aggravated by stress and may radiate to a wider region.[45] There are rarely any sensory changes and no apparent pathologic processes. The onset may be related to an injury or invasive procedure but the pain persists without any demonstrable local cause.[45] However, based on the retrospective analysis of data collected on 493 consecutive patients, 35 who met the inclusion criteria for atypical facial pain, all but one had diagnosable physical problems and sometimes multiple overlapping physical diagnoses causing the pain.[216] Over half (19 of 35) were found to have MFP due to TrPs as a primary cause or a significant contributing factor to the pain. This data, underscores the importance of a careful diagnostic evaluation in the evaluation of patients with unusual pain presentations.

Two types of persistent idiopathic facial pain that commonly appear in the dental office are *atypical odontalgia* (idiopathic or phantom toothache, trigeminal dysesthesia) and *burning mouth syndrome*. Although deafferentation (neuropathic pain) is believed to play a role in both of these pain syndromes, the mechanism and the etiology are still largely speculative.

Atypical Odontalgia—Persistent Dentoalveolar Pain

IHS defines atypical odontalgia as a continuous dental or tooth site pain without any identifiable dental cause.[45] This pain may be a PTTN, but its throbbing nature along with the tendency for the painful site to move around implicates a potential neurovascular source for the pain as well.[4]

It is extremely important for dentists to be aware of the existence of this disorder to avoid performing unnecessary dental procedures and extractions. It has been postulated that as many as 3% to >7% of endodontic patients may suffer from this type of tooth pain, especially if they had pain prior to pulp extirpation.[217]

Symptoms. The chief complaint is a *deep, dull, aching, sometimes burning pain in a tooth or tooth site* that is unchanging over weeks or months. The pain is fairly continuous with some diurnal fluctuation, and usually worsens as the day progresses. The vast majority of patients present themselves with unilateral pain, although other quadrants may become involved and other oral and facial sites may hurt.

Most patients are *female* and *over 40 years* of age. The onset of the pain may coincide with or develop within 1 month or so after dental treatment (especially endodontic therapy or extraction) or trauma or medical procedures related to the face, increasing the impression that the pain is neuropathic.[218,219] There is evidence to support the diagnosis of PTTN in many of these cases and neurovascular pain in others, again emphasizing the importance of careful evaluation and diagnosis of unusual pain presentations.[220]

Examination. Intraoral and radiographic examinations are typically unrevealing. If the pain is in a tooth as opposed to an extraction site, responses to percussion, thermal testing, and electric pulp stimulation are variable. The majority of patients report little or no relief with diagnostic local anesthetic blocks.

Treatment. Invasive, irreversible treatments, such as endodontic therapy, exploratory surgery, extraction, or even

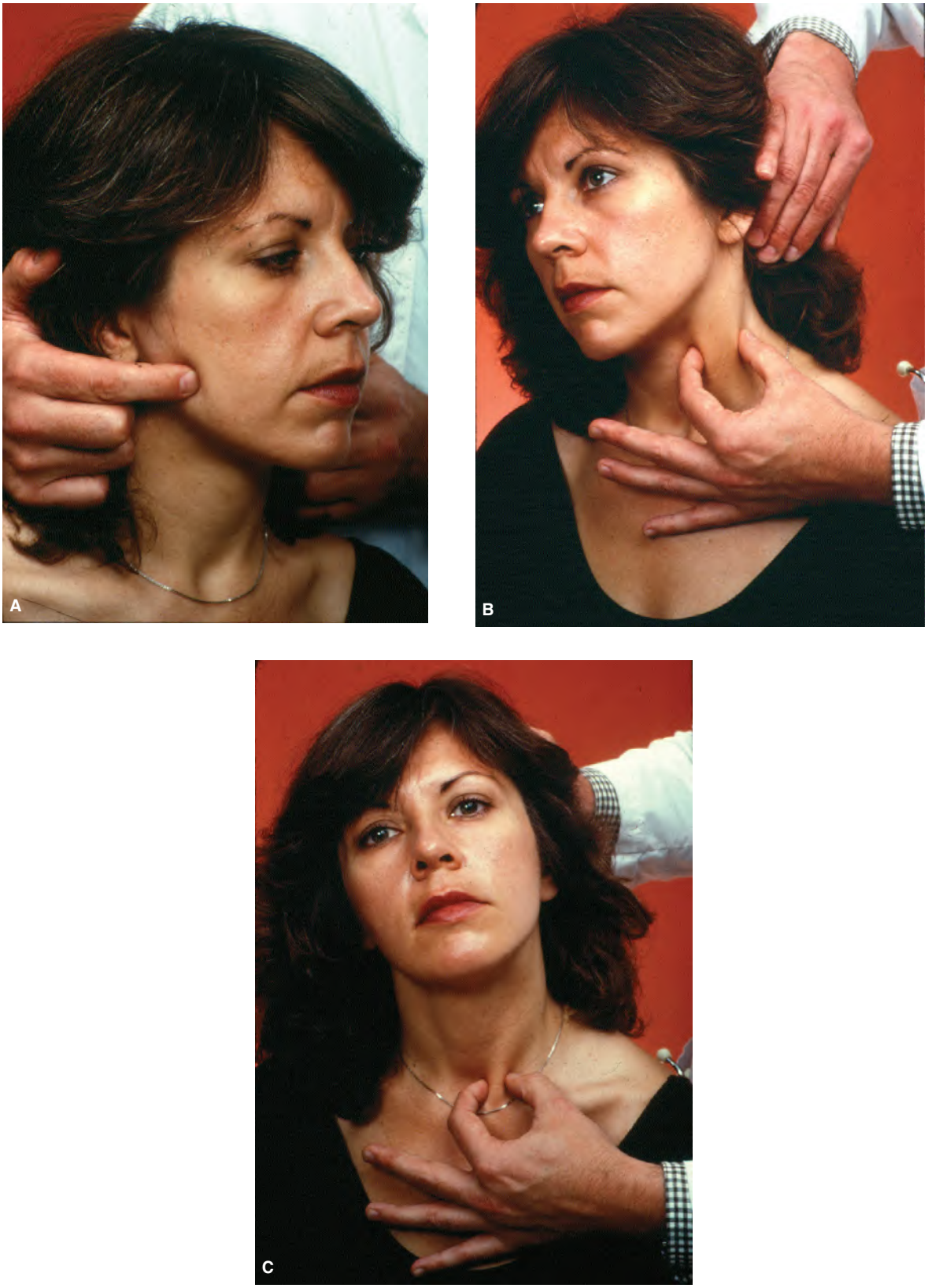

FIGURE 17-23 Muscle palpation for myofascial trigger points. ***A.*** "Flat" fingertip palpation of the masseter muscle looking for taut bands and focal tenderness characteristic of myofascial TrPs. The flat palpation technique is also useful for temporalis, suboccipital, medial pterygoid, and upper back muscles. ***B.*** "Pincer" palpation of the deep clavicular head of the sternocleidomastoid muscle. ***C.*** "Pincer" palpation of the superficial sternal head. Heads should be palpated separately. The upper border of the trapezius also lends itself to pincer palpation. (Reproduced with permission and courtesy of Dr. Bernadette Jaeger, Los Angeles, CA.)

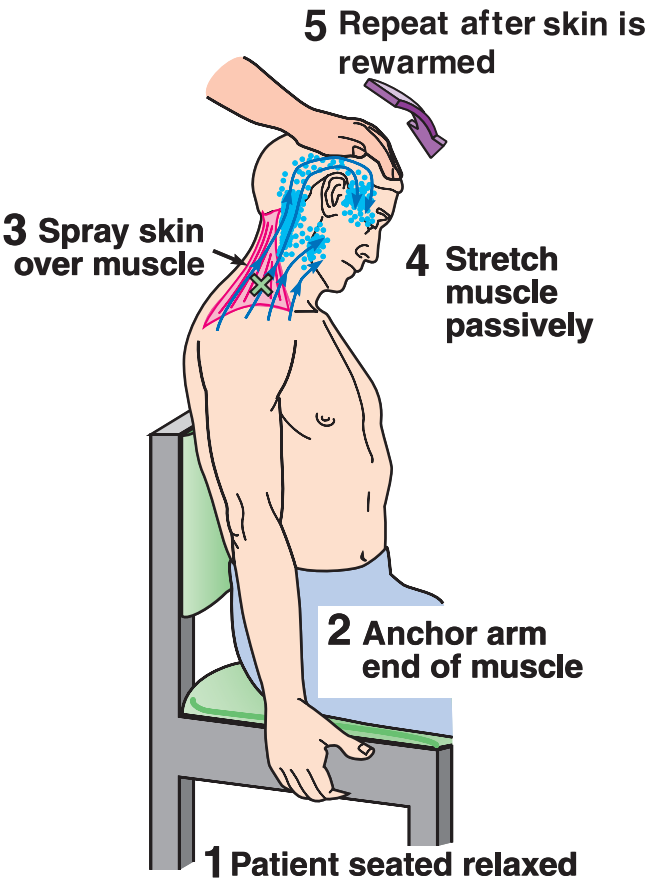

FIGURE 17-24 Sequence of steps when spraying and stretching any muscle to treat MF TrPs, here applied to the upper trapezius. **1.** Patient supported in a comfortable relaxed position. **2.** One end of the muscle is anchored. **3.** Skin sprayed with three to four parallel sweeps of vapocoolant over the length of the muscle and into the referred pain site. (arrows). **4.** Immediately after completing the spray pattern, the patient is asked to inhale deeply, then exhale as gentle pressure is applied to stretch the muscle during exhalation. **5.** Steps 3 and 4 should be repeated only after the skin is rewarmed. Hot packs and several cycles of full active range of motion follow. (Reproduced with permission from Travell JG, Simon DG, et al, 1999.[49])

occlusal adjustments, *are contraindicated*.[218] This is because, despite possible transient relief, the pain is likely to recur with equal or greater intensity.

The current treatment of choice is the use of *tricyclic antidepressant agents* such as *amitriptyline or imipramine* alone or, if there is a burning quality to the pain, in combination with a *phenothiazine*. Patients may need reassurance that *the pain is real and not psychogenic* but that invasive procedures will not help.

If a patient complains of associated gingival hyperesthesia, the use of topical agents such as topical anesthetic or capsaicin has been shown to be beneficial.[219] Unwanted stimulation of the area can be reduced by construction of an acrylic stent that can also be used to help apply any topical medication.

Burning Mouth Syndrome

Burning mouth syndrome (BMS) is an intraoral pain disorder that most commonly affects postmenopausal women. When it primarily affects the tongue, it is referred to as burning tongue or glossodynia.

Symptoms. Patients complain of intraoral burning, the tip and sides of the tongue being the most common sites, followed by the palate. Dry mouth, thirst, taste and sleep disturbances, headaches, and other pain complaints are frequently associated symptoms. Onset is often related to a dental procedure. The intensity of pain is quantitatively similar to that of toothache, although the quality is burning rather than pulsing, aching, or throbbing. The pain increases as the day progresses and tends to peak in the early evening. Patients with BMS may complain of spontaneous tastes or "taste phantoms" (dysgeusia).[224]

Etiology. There are many obvious oral and systemic conditions that are associated with mucogingival and glossal pains. These include candidiasis, geographic tongue, allergies to dental materials, denture dysfunction, xerostomia, various anemias and vitamin deficiencies (iron, vitamin B_{12}, or folic acid), diabetes mellitus, several dermatological disorders (lupus, lichen planus, erythema multiforme), human immunodeficiency virus, or systemic medications (either directly or indirectly through resultant xerostomia). However, in BMS, controlled studies have documented that the oral mucosa appears normal, and no obvious organic cause can be identified.[222]

Research supports a theory that damage to or dysfunction of the chorda tympani branch of CN VII contributes to the dysguesia and pain of burning mouth.[223] The special sense of taste from the tongue is mediated by the chorda tympani (anterior two thirds) and CN IX (posterior one-third). The chorda tympani innervates the fungiform papillae of the tongue, which, in turn, are surrounded by pain fibers from cranial nerve V. The chorda tympani normally inhibits both cranial nerve V (pain fibers) and cranial nerve IX (taste). Damage or partial deafferentation of the chorda tympani appears to release inhibition of both cranial nerves V and IX, producing both pain and taste phantoms, respectively.[224]

BMS is not to be confused with postmenopausal oral discomfort that has a burning component. In the latter condition, estrogen replacement therapy is effective about half of the time.[225] Psychological factors, although present in some of this population, do not appear to be etiological.[226]

Examination. The patient with BMS generally has a negative intraoral examination.[222] Obvious tissue irritating causes such as denture soreness, rough crowns or teeth, and other causes of tissue irritation such as candidiasis or true vitamin deficiencies must, of course, be ruled out.

Tests. Candidiasis should be tested for, even if the mucosa looks normal. *Local anesthetic rinse* will decrease the pain of other conditions such as geographical tongue or candidiasis but increase the pain of burning mouth. This increase in pain

is thought to be due to further loss of **A**β fiber inhibition. Sedimentation rate may be mildly elevated, and, in view of the higher incidence of immunological abnormalities, rheumatological evaluation should be considered.

Treatment. The management of BMS is still not satisfactory. Topical clonazepam (sucking on clonazeoam lozenges), benzodiazepines and low dose tricyclic antidepressants have some success.[222]

CONCLUSION

In conclusion, it is a time-consuming and difficult task to understand and manage people suffering unusual or chronic pain. Many multidisciplinary pain clinics employ orofacial pain dentists; physicians; psychologists; nurses; physical, occupational, and myotherapists; and other health professionals to coordinate patient care and to provide a comprehensive healthy environment for rehabilitation. Chronic pain management, including orofacial pain, is a growing specialty in health care. It takes years of training and experience to gain adequate insight into these complex cases.

Several dental schools across the United States now offer CODA accredited 2-year postgraduate orofacial pain programs to train interested dentists in the field of orofacial pain. Dentists with competence in this field and who have passed a certifying examination can be identified by contacting the American Board of Orofacial Pain or the equivalent Board or certifying agency in other countries. Orofacial pain dentists, armed with knowledge, interest, curiosity, and patience, and, finally, the instruments of diagnosis, enjoy the most rewarding experience—identifying the source of pain and relieving the suffering of a fellow human being. The orofacial pain dentist, oriented toward looking at the patient as a whole person, can gain much satisfaction from arriving at a correct diagnosis and saving teeth or preventing unnecessary surgery.

The complex and personal nature of good diagnosis cannot be overemphasized. This is the area in which the dentist is most likely, on a professional basis, to earn the respect and friendship of colleagues, both dental and medical, as well as patients.

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Chapter 18

Management of Pain, Fear, and Anxiety in the Endodontic Patient

Stanley F. Malamed

Pain, fear, and anxiety are not uniquely endodontic problems; they are, however, far more significant in endodontics because of the commonplace nature of the patients' underlying problem: pain. "Fear of dentists" consistently appears in surveys of common fears, along with fear of heights, flying, mice, and public speaking.[1] Common dental fears include fear of the unknown, fear of pain, and, perhaps the most common, fear of the "shot."

Numerous studies have assessed the incidence of dental fear (odontophobia, dentophobia) in the general population, determining that between 10% and 30% of the adult population suffers from moderately severe to extreme odontophobia.[2–4] Chanpong and colleagues,[4] in a survey of Canadian adults (n = 1101), found 7.6% stating they had "missed, cancelled, or avoided a dental appointment because of fear or anxiety." In response to the question "How would you assess your feelings toward having dental treatment done?" 5.5% assessed themselves as either "very afraid" (2.0%) or "terrified" (3.5%). In this "high fear" group, 49.2% had missed, cancelled, or avoided a dental appointment because of fear or anxiety compared with only 5.2% of the "low or no fear" group. Enkling and colleagues[5] reported that in 67% of odontophobic patients a prior painful dental or medical experience was the primary cause of their fear, followed by fear of needles (33%).

Dental fear is real and it hurts. For the patient, it is palpable. For the doctor and office staff, it stands as a barrier to the delivery of quality dental care. Within the realm of endodontics, many patients requiring root canal treatment do so as an end result of their extreme odontophobia. The first endodontic appointment, during which access will be gained and pulpal tissues removed, provides the greatest challenge to the endodontist, confronted with a patient who is in acute pain and having possibly been in chronic pain for several months, and who is fearful, not just of the local anesthetic injection but of the idea of "root canal work" itself.

Pain and fear are a potent combination, capable of provoking some of the most catastrophic situations imaginable in the dental office environment. Surveying the incidence of medical emergencies in the dental environment, Malamed[6] found that 54.9% occurred during the administration of the local anesthetic with an additional 22.0% occurring during dental treatment. When a medical emergency arose during dental treatment, 65.8% occurred during pulpal extirpation (26.9%) or tooth extraction (38.9%).[7] Over three-quarters of medical emergencies seen in dentistry are stress-related. Potentially stress-related medical emergencies include syncope, hyperventilation, the "epinephrine reaction," bronchospasm, seizures, angina pectoris, myocardial infarction, and cardiac arrest.

RECOGNITION OF FEAR

Recognition of dental fear should not be left until the patient is seated in the dental chair. Quite often the receptionist is asked revealing questions by patients, such as "Is the doctor gentle?" "Does the doctor give good shots?" Patients in the reception area may converse amongst themselves, discussing their upcoming treatment, perhaps in lurid ways. This invaluable information must be relayed to chair-side personnel who can now act to prevent a "problem" from developing.

Once seated in the dental chair, the patient's fears usually become more obvious. Fearful patients simply do not "look" comfortable. Legs remain crossed and fingers clutch the armrest of the dental chair, the so-called "white-knuckle syndrome." The patient closely watches everything, not wanting to be "snuck up on" by the doctor. Responses to questions are unusually prompt; speech is rapid. Perspiration may be observed on the patient's forehead, upper lip, and perhaps underarms.

If any of the previous is noted, it is important for the doctor to confront the patients, asking them "if anything about the upcoming procedure bothers them" Once the patient admits to having fears and once the fears are out in the open, the "problem" should be manageable.

Management problems occurring during the local anesthetic administration can be almost entirely prevented by taking a patient's "feelings" about receiving "shots" into consideration. Most people do not relish the thought of receiving intraoral local anesthetic injections as demonstrated by the high incidence of adverse reactions occurring at this time. Fifty-five percent of all medical emergencies reported by Malamed[6] were fainting, and over 54% of all emergencies occurred during administration of the local anesthetic.[7] Syncope accounted for 61.1% of dental office medical emergencies reported during a 1-year period in New Zealand.[8]

Fainting during injection is preventable by following a few simple steps aimed at making all local anesthetic injections as comfortable (atraumatic) as possible: (1) placing the patient about to receive an intraoral injection into a supine position *prior* to injection, (2) slow administration of the local anesthetic solution, and (3) use of sedation, if warranted, prior to the administration of the local anesthetic.

MANAGEMENT OF FEAR OF DENTISTRY

The concept behind the successful use of sedation is that fearful patients are overly focused on everything that happens around them, and to them, in the dental office environment. Sedation is about distraction—taking the patients mind away from what is happening in their mouth during the dental appointment. Distraction may occur with or without the use of drugs. Music, dark glasses, movies are nondrug means of distracting the fearful patient. Administration of a central nervous system (CNS)-depressant drug lessens the patients' awareness, taking their mind away from thinking about the dental treatment. They no longer over-respond to stimulation. They no longer care about the procedure and, in effect, become more "usual" patients.

Definitions

Pharmacosedation occurs as a result of depression of the CNS. Although various levels of CNS depression will be discussed, one is in fact dealing with a continuum, from the earliest manifestations of a drug's action—*minimal sedation*, through the controlled loss of consciousness—*general anesthesia*. Most jurisdictions in the United States require a licensed dentist to obtain a permit before being allowed to administer CNS-depressant drugs via various routes and to varying levels of CNS depression. A number of dental (and medical) organizations have published guidelines for the safe and effective use of these drugs.[9–11]

When describing, and defining, the various levels of CNS depression, the precise wording of the definition may vary slightly from country to country, state to state, province to province, and organization to organization, but the essence of the definitions is the same. The reader is strongly advised to adhere to those definitions that have been established in the jurisdiction in which he or she is licensed. Definitions, proposed by the American Society of Anesthesiologists, describe the degree of CNS depression ultimately achieved (Table 18-1).[12] The definitions that follow are taken from the American Dental Association's (ADA's) Guidelines for the Use of Sedation and General Anesthesia by Dentists.[11]

Minimal Sedation

Minimal sedation is defined as "a minimally depressed level of consciousness that retains the patient's ability to independently and continuously maintain an airway and respond appropriately to physical stimulation or verbal command, and that is produced by a pharmacologic or nonpharmacologic method or a combination thereof. Although cognitive function and coordination may be modestly impaired, ventilatory and cardiovascular functions are unaffected."[11–12] "In accord with this particular definition, the drug(s) and/or techniques used should carry a margin of safety wide enough to render unintended loss of consciousness unlikely. Furthermore, patients whose only response is a reflexive withdrawal from repeated painful stimuli would not be considered to be in a state of minimal sedation."[11–12]

"When the intent is minimal sedation for adults, the appropriate dosing of enteral drugs is no more than the maximum recommended dose of a single drug that can be prescribed for unmonitored home use."[11–12]

Nitrous oxide/oxygen (N_2O–O_2) inhalation sedation, when used in combination with other CNS-depressant drugs may produce minimal, moderate, or deep sedation or general anesthesia.

The following definitions apply to administration of ***minimal sedation***:

Maximum recommended therapeutic dose (MRTD): maximum FDA-recommended dose of a drug approved for unmonitored home use.

Incremental dosing: administration of multiple doses of a drug until a desired effect is reached, but not to exceed the MRTD.

Titration: administration of incremental doses of a drug until a desired effect is reached. Knowledge of each drug's time of onset, peak response, and duration of action is essential. Although the concept of titration of a drug to effect is critical, when the intent is minimal sedation, one must know whether the previous dose has taken full effect before administering an additional drug dose.

Moderate Sedation

Moderate sedation is "a drug-induced depression of consciousness during which patients respond purposefully to verbal

TABLE 18-1 ASA Chart of Categories

	Minimal Sedation (Anxiolysis)	Moderate Sedation/ Analgesia (Conscious Sedation)	Deep Sedation/Analgesia	General Anesthesia
Responsiveness	Normal response to verbal stimulation	Purposeful* response to verbal or tactile stimulation	Purposeful* response after repeated or painful stimulation	Unarousable, even with painful stimulus
Airway	Unaffected	No intervention required	Intervention may be required	Intervention often required
Spontaneous ventilation	Unaffected	Adequate	May be inadequate	Frequently inadequate
Cardiovascular function	Unaffected	Usually maintained	Usually maintained	May be impaired

*Source: American Society of Anesthesiologists Task Force on Sedation and Analgesia by Non-Anesthesiologists.[12]

commands, either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained."[11–12] "In accord with this particular definition, the drugs and/or techniques used should carry a margin of safety wide enough to render unintended loss of consciousness unlikely. Repeated dosing of an agent before the effects of previous dosing can be fully appreciated may result in a greater alteration of the state of consciousness than is the intent of the dentist."[11–12] "Furthermore, a patient whose only response is a reflexive withdrawal from a painful stimulus is not considered to be in a state of moderate sedation."[11–12]

Deep Sedation

Deep sedation is "a drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully following repeated or painful stimulation. The ability to independently maintain ventilatory function may be impaired. Patients may require assistance in maintaining a patent airway, and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained."[11–12]

General Anesthesia

General anesthesia is "a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation. The ability to independently maintain ventilatory function is often impaired. Patients often require assistance in maintaining a patent airway, and positive pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired."[11–12]

"Because sedation and general anesthesia are a continuum, it is not always possible to predict how an individual patient will respond. Hence, practitioners intending to produce a given level of sedation should be able to diagnose and manage the physiologic consequences (rescue) for patients whose level of sedation becomes deeper than initially intended."[12]

"For all levels of sedation, the practitioner must have the training, skills, and equipment to identify and manage such an occurrence until either assistance arrives (emergency medical service) or the patient returns to the intended level of sedation without airway or cardiovascular complications."[12]

REGULATION

Through the 1960s, upon receiving a dental degree and a license to practice dentistry, dentists in the United States were allowed to administer any form of anesthesia (from local anesthesia to sedation to general anesthesia). No prohibitions, except for common sense, existed. As no formal training had been received in these techniques (aside from local anesthesia) in dental school, most new doctors prudently avoided their use, managing fearful patients as best they could. Some, however, felt that they could easily administer anesthesia (in the broad sense) to their patients. Although some were successful, a large enough number became involved in serious untoward events, leading to death or severe neurologic damage, that governmental agencies (state dental boards and/or legislatures) began to seriously question whether dentists should be allowed to perform these techniques.

In the early 1970s, an Alaskan dentist had several deaths occur under halothane general anesthesia. As this dentist had little or no formal training in general anesthesia, Alaska became the first state to ban the administration of general anesthesia in the dental office. In 1974, in response to four patient deaths under general anesthesia in a short span of time in the office of an untrained dentist, Ohio became the first state to limit the use of general anesthesia to dentists who could prove adequate training, either through an oral surgery program or a 1-year anesthesiology residency.[13,14]

As of April 2015, all 50 states in the United States have enacted regulation governing the administration of general anesthesia and deep sedation in dental offices.[15] Deep sedation, by virtue of the fact that the patient's ventilatory status and ability to maintain an airway may be impaired, requires a level of training equal to that of general anesthesia. Education and training in general anesthesia in the United States requires a minimum of a 3-year residency in anesthesiology.[16]

Some dentists, untrained and now unable to administer general anesthesia, began administering CNS-depressant drugs parenterally, either intramuscularly or intravenously, as these techniques had not yet been regulated. Not surprisingly, a number of deaths occurred, as well as other serious morbidities over the ensuing years. And also, not surprisingly, legislative bodies began to regulate parenteral sedation. All 50 states in the United States now regulate the administration of parenteral moderate sedation.[17] (Intranasal [IN] sedation, a relatively new approach to CNS-depressant drug administration used primarily in pediatric dentistry, is classified as a parenteral route of drug administration.)

The oral route, the least effective and least controllable common mode of drug delivery, had always enjoyed a somewhat limited use in dentistry. As other routes of drug delivery and levels of sedation encountered increased scrutiny and regulation, interest burgeoned in this, as yet, unregulated mode of drug administration.

Orally administered drugs had always been an important management technique within the specialty practice of pediatric dentistry, with chloral hydrate, hydroxyzine, and promethazine forming a triad of oft-used drugs. In 1985, the American Academy of Pediatric Dentistry developed guidelines for the use of sedation by pediatric dentists who received adequate clinical and didactic experience in this technique during their residencies. These guidelines have been reevaluated several times with the most recent version accepted and published in 2008.[18]

Unfortunately, some untrained nonpediatric dentists (e.g., general dentists), now unable to administer drugs parenterally, began using oral sedative drugs to achieve more profound levels of CNS depression, with predictable results: death and severe morbidity (e.g., brain damage). Legislation to require permits for oral moderate sedation (OMS) in children appeared in the late 1990s. Enacted in 2000, California's legislation requires a

permit for OMS in patients less than 13 years of age.[19] At present, 11 states in the United States require advanced education and awarding of a permit for a licensed dentist to administer oral conscious sedation (OCS) to a pediatric patient.[17]

OMS for adult dental patients had an increase in popularity in the mid-1990s, that continues today. Although many oral drugs are available, one drug, triazolam (Halcion®), has proven the most popular.[20,21]

Now, for the first time, state dental boards have acted proactively rather than reactively as in the past, enacting legislation mandating continuing dental education (CDE) and a permit to administer OMS to the adult or pediatric dental patient. As of April 2015, 19 states in the United States have requirements for CDE and a permit for adult OCS.[17]

One route of drug administration has yet to be mentioned: inhalation. In contrast to the oral route, the inhalation route represents *the* most controllable technique of drug administration. In dentistry in the United States, the combination of N_2O and O_2 is available and used, with varying frequency, in approximately 35% of dental offices.[22] The ADA, in concert with manufacturers of inhalation sedation units, mandated the inclusion of safety features into these machines. The goal of these safety features is to make it close to impossible the administration of levels of O_2 less than 21% (ambient air) to a patient.[23] Deaths and serious morbidity associated with N_2O–O_2 inhalation sedation in dentistry have not occurred in recent years due primarily to the addition of these safety features and the requirement of the ADA's Commission on Dental Accreditation that all graduating dental students be trained to proficiency in inhalation sedation.[24] As of April 2015, only a handful of states require a permit for the use of inhalation sedation.

Sedation: Nondrug Techniques (Iatrosedation)

Management of a patient's dental fear should begin as soon as the patient enters the dental office. The environment, the ambiance of the office, establishes a mood either of quiet relaxation or of a hurried, frenetic pace. The dental staff should be alert to any telltale signs of dental fear and, if noted, report it to the dentist immediately. "Forewarned is forearmed." Relaxation of a patient by the doctor's behavior has been termed *iatrosedation*, a term formulated by Dr. Nathan Friedman.[25] The word is derived from the Greek prefix *iatro*, "pertaining to the doctor," and *sedation*, "the relief of anxiety."

The concept on which iatrosedation is based is rather basic: that the behavior of the doctor and staff has a profound influence on the behavior of the patient. Other terms applied to this concept include suggestion, chairside/bedside manner, and the "laying on of hands." The underlying premise of all these techniques is similar: that one can use nonchemical means to aid in relaxing the patient. For a more in-depth discussion of nondrug techniques of sedation, the reader is referred to *Sedation: a guide to patient management*.[26] Examples of iatrosedative techniques include hypnosis, acupuncture, audioanalgesia, and biofeedback.

Iatrosedation forms the building block for all sedation techniques requiring drug administration. Simply put, a patient who remains fearful, perhaps with distrust of the doctor, is less likely to "allow" their CNS-depressant drug to work. Although true for all routes of drug administration, it is especially relevant with the oral and inhalation routes.

The doctor who establishes a bond of trust with their patients will have them sit back in the dental chair and "let the drug work." Conversely, where the doctor–patient relationship is strained or uncomfortable, patients are much less likely to surrender to the clinical effects of the drug, not wanting to "lose control" of the situation. The level defined as minimal sedation is highly unlikely to succeed in this situation. Moderate sedation would have a somewhat greater success rate but still with a significant, perhaps unacceptably high, failure rate (Table 18-2).

Sedation: Drug Techniques (Pharamacosedation)

In dentistry, CNS-depressant drugs are administered by four routes: commonly by oral and inhalation, and less commonly by intravenous (IV) and intramuscular (IM). IN drug administration is a relatively recent addition to this armamentarium, primarily employed to provide moderate sedation in children.[27]

The following is a brief overview of these routes of CNS-depressant drug administration in dentistry. It is not meant to substitute for a complete course in pharmacology or in the safe and effective technique of drug administration. The doctor wishing to administer drugs by any of these routes should check with their regulatory boards for specific education and permit requirements for each technique or level of sedation.

Inhalation Sedation (N_2O–O_2)

Inhalation sedation with N_2O–O_2 represents the most controllable technique of sedation available and should represent, in this author's opinion, the "starter technique" for sedation in dentistry. Inhalation sedation possesses a number of compelling clinical properties that serve to increase its success rate and its safety, including (1) rapid onset of action (~20–30 s); (2) a level

TABLE 18-2 Efficacy of Sedation By Routes of Drug Administration

Technique route	Titrate to effect	Rapid reversal	Expected success rate (%)
Intravenous	Yes	Yes*	90
Inhalation	Yes	Yes	80
Intramuscular	No	No	67
Intranasal	No	No	67
Oral (adult)	No	No	50–60
Oral (child)	No	No	Older: 50–60 Younger: 35–40
General anesthesia†	Yes	Yes#	100

*Opioids. benzodiazepines.
†General anesthesia is *not* a sedation technique. It is included for comparative purposes only.
#Dependent on route of administration and drug.

of CNS depression that can rapidly be increased, if necessary; (3) the level of CNS depression that can be rapidly decreased, if needed—a significant factor in increasing the safety of inhalation sedation; (4) complete recovery following the delivery of 100% O_2 at the conclusion of the procedure, permitting almost all N_2O–O_2 patients to be discharged from the dental office unescorted, and with no prohibitions on their postinhalation sedation activities. No other route of drug administration offers this significant advantage. Because of its rapid onset, inhalation sedation with N_2O–O_2 may be titrated. The ability to titrate increases both the success and safety of the technique.

In order for the dose of any drug to be titrated, it must enter into the cardiovascular system rapidly. When possible, titration allows the doctor to individualize the dosage of the drug for each patient, negating the so-called "bell-shaped" or "normal distribution" curve. Approximately 68% of persons respond "normally" to the "usual" dose of a drug (normoresponders). Fourteen percent will under-react to this usual dose (hyporesponders), while 14% will overreact to the same dose (hyperresponders).

The technique of inhalation sedation with N_2O–O_2 possesses very few significant disadvantages. One factor, common to it and several other techniques, is that of patient cooperation. The patient breathes the gasses through a small mask placed on the nose, the nasal hood. Uncooperative patients, usually odontophobic younger children, or any patient who is claustrophobic, might not allow the nasal hood to be placed, condemning the inhalation route to failure. Persons unable to breathe through their nose, for whatever reason, will be unable to receive inhalation sedation in a dental office. A recommended technique of administration of N_2O–O_2 is outlined in (Table 18-3).

Inhalation sedation with N_2O–O_2 may be employed to provide minimum to moderate sedation. It may also be administered, where permitted by state regulation, in conjunction with drugs administered by other routes, to supplement the CNS depression they provide. N_2O–O_2 when used in combination with sedative agents may produce minimal, moderate, or deep sedation or general anesthesia.[11]

The use of N_2O–O_2 inhalation sedation in endodontics should become more common given the positive attributes of the technique. A common complaint from endodontists is that the nasal hood "is in the way" (Figure 18-1). Once experience is gained in the technique, this ceases to be a concern. Inhalation sedation with N_2O–O_2 has a success rate of approximately 80% in adult patients.

TABLE 18-3 Technique of Inhalation Sedation–A Review

1. Prior to placing nasal hood, start a flow of 5–6 LPM (liters per minute) of O_2.
2. Have patient assist in proper placement and securing of the nasal hood.
3. Determine if patient can "breathe comfortably" with 100% oxygen. ("Is the flow volume adequate?"). Increase the flow, if necessary.
4. Start titration of N_2O by increasing its flow to 1 LPM, decreasing the O_2 flow by 1 LPM.
5. After 1 min, determine what, if any, signs and symptoms the patient may be experiencing.
6. If needed, increase N_2O by 0.5 LPM, decreasing O_2, 0.5 LPM.
7. Repeat steps 5 and 6 until the patient reaches the desired level of sedation.
8. Administer local anesthesia as would be done if the patient were not receiving N_2O–O_2.
9. At the conclusion of the procedure, increase the O_2 flow to the level determined in step 3 and return the N_2O to O LPM.
10. Permit the patient to breathe 100% O_2, for not less than 3–5 min before considering removal of the nasal hood.
11. Assess recovery from sedation. If considered recovered, remove nasal hood before terminating the flow of O_2.
12. Permit the patient to leave dental chair. Have a staff person close to the patient so as to prevent any possible injury when standing, due to postural hypotension.
13. Document treatment in the patient's chart.

Oral Moderate Sedation

The oral route is the least controllable route of drug administration. A number of factors work against an orally administered drug being effective, including a slow onset of action (~1 h for most drugs); erratic absorption of the drug from the gastrointestinal (GI) tract; and, for some drugs, a significant hepatic first-pass effect. For these reasons, titration of the drug to clinical effect is not possible, eliminating the most important safety factor in drug administration. Once administered, the level of CNS depression reached by oral drugs is not easily increased (providing deeper sedation) or decreased (lighter sedation).

The duration of CNS depression from orally administered drugs greatly exceeds the typical length of the dental visit so these patients will always require an escort on being discharged from the office. This escort need be a responsible adult who has a vested interest in the health and safety of the patient.

The only advantages associated with the oral route of drug administration are that it is easier for both the doctor and the patient.

Having just described a technique that seems quite mediocre compared with other routes of administration, it must be stated that there is a legitimate place for orally administered CNS depressants in dentistry.

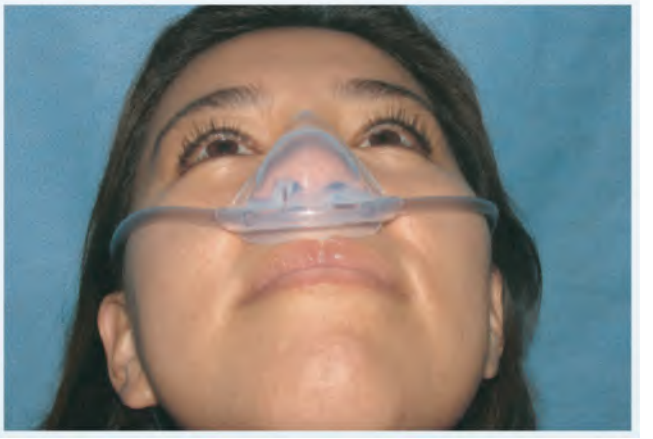


FIGURE 18-1 N_2O–O_2 nasal hood—Porter Silhouette Mask (Courtesy of Porter Instrument, Philadelphia, PA, www.porterinstrument.com/silhouette).

Odontophobic patients, especially when faced with the fear of root canal treatment, frequently require management with CNS depressants (1) the night prior to the planned appointment, in order for the patient to experience a restful night's sleep, and (2) in the morning, 1 h prior to the scheduled dental visit, to assist them in overcoming any last minute increase in their anxiety.

In the absence of other sedation techniques in a dental office, the oral route may also be used for intraoperative sedation. However, owing to the lack of control maintained over this technique by the doctor, the goal in administering oral CNS depressants should be limited to minimal to moderate CNS depression.

A number of excellent oral preparations are currently available. Table 18-4 presents some commonly used drugs and their recommended dosages.[28,29] When used alone, and in recommended dosages, orally administered drugs will have a success rate of approximately 50%–60% in adult patients. This success rate is even lower in younger children.

Intravenous Moderate Sedation

CNS-depressant drugs administered intravenously in the dorsum of the hand reach the brain in approximately 20–30 s. With this rapid onset, IV drug administration allows patients to be titrated to a desired level of CNS depression, thereby increasing control over the effect of the administered drug and, ultimately, the safety of the technique. Other advantages of IV drug administration include the ability to rapidly increase the level of sedation, if needed, and the reversibility of the most commonly administered IV drugs (benzodiazepines and opioids).

Disadvantages associated with intravenously administered CNS depressants include the requirement of fasting prior to the procedure, Nil Per Os (NPO status); an inability to quickly lessen the level of CNS depression; an inability to reverse the clinical actions of some drugs; and prolonged clinical recovery with an attendant need for an adult escort for the patient when discharged from the dental office.

Venipuncture is a learned skill and, though normally quite easy to achieve, represents the most difficult part of the entire IV sedation procedure. Once venous access is obtained, titration to the desired level of CNS depression is normally accomplished easily.

Many CNS-depressant drugs are available for IV administration, but those most often employed are the benzodiazepines, midazolam (formerly Versed®), and diazepam (formerly Valium®).

State requirements for a permit to employ IV moderate sedation (also termed "parenteral sedation") most often mandate 60 h of didactics and approximately 20 cases of IV sedation administered by the doctor in a supervised environment.[17] Variation may exist from jurisdiction to jurisdiction, so it is strongly advised that specific state or provincial dental board regulations be consulted.

Basic IV sedation technique has evolved over the past four decades. The combination of pentobarbital (Nembutal), meperidine (Demerol), and scopolamine represented the original IV technique introduced in the 1950s by Dr. Neils Björn Jörgensen.[30] As described by Jörgensen, the drugs were injected directly into the vein via the syringe that was then removed.[31] The Jörgensen technique provided a duration of CNS depression of approximately 2 h, a function of the barbiturate, a pentobarbital. With the introduction in the early 1960s of the benzodiazepine diazepam, a shorter duration of CNS depression became possible.[32] An additional benefit of diazepam was a brief period (approximately 10 min) of retrograde amnesia, permitting potentially traumatic procedures to be done with a likelihood of the patient not having any recall. For the patient this meant, in essence, that the fearful event (e.g., local anesthetic injection) never happened.

TABLE 18-4 Oral CNS-Depressants Used in Dentistry

Generic Name	Proprietary Name Availability (mg)		Usual Dental Dosage
Benzodiazepines			
Alprazolam	Niravam, Xanax	Tab: 0.25, 0.5, 1.0, 2.0	0.25–0.5 (max 4 mg/day)
Diazepam		Tab: 2, 5, 10	2–10 mg bid-qid
Flurazepam	Dalmane	Cap: 15, 30	15–30 mg at bedtime
Lorazepam	Ativan	05, 1.0, 70	2–3 mg/day given bid-tid
Midazaolam		Syr: 2 mg/mL	Pediatrics: 0.25–1.0 mg/kg single dose
Oxazepam	Serax	Cap: 10, 15, 30 Tab: 15	Adults (anxiety): mild to moderate: severe: 15–30 tid-qid
Triazolam	Halcion	Tab: 0.125, 0.15	0.25 qhs, max 0.5
Miscellaneous, nonbenzodiazepine anxiolytics, sedatives			
Eszopiclone	Lunesta	Tab: 1.0, 2.0, 3.0	Initial: 2 mg qhs
Zaleplon	Sonata	Cap: 5, 10	Insomnia: 10 qhs
Zolpidem	Ambien	Tab: 5, 10	Adult: usual 10 mg qhs
Miscellaneous sedative-hypnotic			
Hydroxyzine HCl	Atarax	Syr: 10 mg/5 mL Tab: 25, 50, 100	Adults (Sedation): 50–100 mg
Hydroxyzine pamoate	Vistaril	Cap: 25, 50, 100 Sus: 25 mg/5 mL	

Abbreviations: Cap, Capsule; Sus, Suspension; Syr, Syrup; Tab, Tablet.
Sources: (1) ADA/PDR Guide to Accepted Dental Therapeutics.[26] (2) www.ePocrates.com[27]

IV moderate sedation with a benzodiazepine has become the most popular technique in dentistry. IV benzodiazepine sedation meets the needs of contemporary dental practice, that is, sedation for approximately 1 h. Introduced in the United States in 1986, midazolam (Versed®) has now supplanted diazepam (Valium®) as the most used IV benzodiazepine in the area of 1-h IV moderate sedation. (As an aside, neither proprietary name, Versed or Valium exists any longer. The drugs are available solely as generics, midazolam and diazepam) Midazolam, unlike diazepam, is water soluble, thus capable of being administered intramuscularly and intranasally (see following discussions of these techniques). The amnestic properties of midazolam are considerably greater than diazepam's, providing lack of recall for most, if not all, of the dental appointment. Either drug, with one titrating dose, will provide the doctor with approximately 1 h of working time.

Direct injection of a drug into a vein, followed by removal of the syringe at the start of the dental procedure, gave way to the continuous IV infusion, in which venous access is maintained for the duration of the procedure. Initially, winged needles, also known as "scalp vein" and "butterfly needles," were used (Figure 18-2). Easy to insert, the winged needle has the disturbing propensity to perforate the vein during the dental procedure, leading to loss of venous access and formation of a hematoma. In recent years, the indwelling catheter has replaced the 'butterfly needle' as the preferred device. Once inserted and secured in a vein, the 3" plastic catheter is unlikely to accidentally become dislodged (Figure 18-3).

Opioids, specifically the short-acting fentanyl, have also been used in IV moderate sedation. When the primary goal of IV drug administration is management of fear, there is usually little or no need for opioid administration. Indeed, in the vast majority of successful IV sedation cases, the only drug(s) administered are either midazolam and/or diazepam. However, in situations in which surgery or other dental procedures that may prove painful are planned, or in which successful pain control may not be easily achieved with local anesthesia alone, opioid administration may be justified.

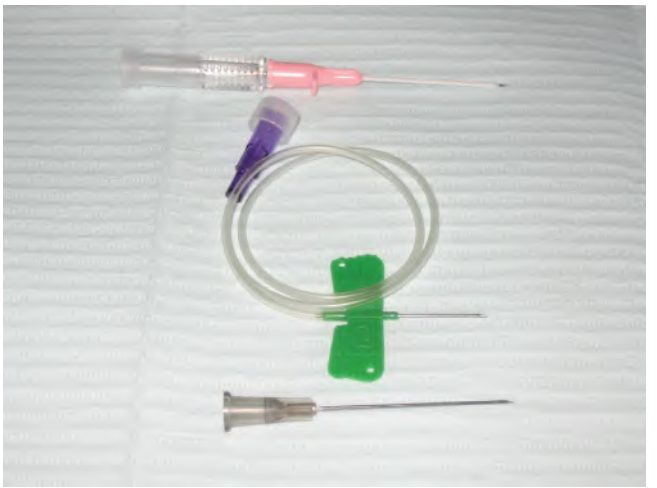


FIGURE 18-2 Intravenous needles.

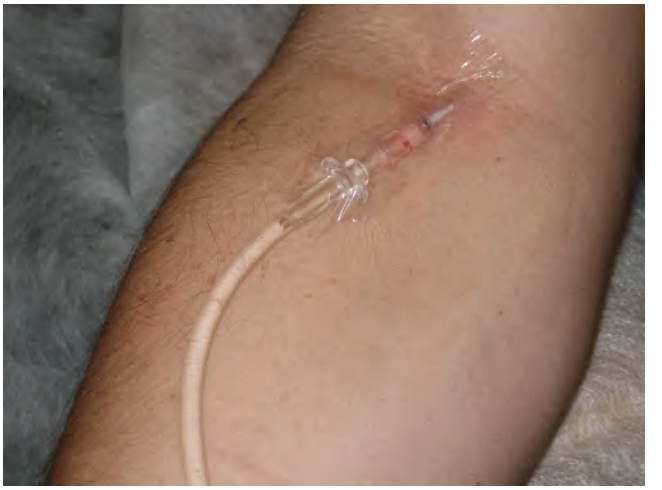


FIGURE 18-3 Indwelling catheter.

IV moderate sedation, administered carefully titrated to clinical effect, has a success rate of 90%. The IV route can be used to achieve moderate and deep sedation, as well as general anesthesia.

Intramuscular

IM, like IV, is a parenteral route of administration, in which the drug bypasses the GI tract, being absorbed directly into the systemic (venous) circulation. The hepatic first-pass effect is negated, leading to more reliable absorption and more rapid onset of action (~10–20 min) than with enterally administered drugs. However, titration is not possible, as the onset is not rapid enough, thus denying the IM route a major safety benefit and thereby limiting its indications for use in sedation in dentistry.

Doses of IM drugs are primarily determined on a weight (e.g. mg/kg) basis. Given the appropriate dosage, approximately 70% of patients should be CNS depressed to the desired level of sedation. However, another 15% will likely be under-sedated with that same dose, whereas the remaining 15% are CNS depressed to a level beyond that which is being sought. This latter group, hyperresponders to the drug, may be CNS depressed to a level beyond which the doctor is able to safely manage them. For example, an IM drug is administered to a patient to achieve a level of moderate sedation. However, the resultant level of CNS depression is deep sedation. At this level, patients are unable to adequately maintain their airway without assistance (e.g., head-tilt, chin-lift). Dental care would, of course, need to be delayed, as the doctor is required to "rescue" patients from this unintended level of CNS depression. Airway management and, possibly, ventilatory assistance are continued until the level of CNS depression lightens, a process that could require several hours.

Along with the inability to titrate IM drugs, it is also not possible to rapidly lessen or deepen the level of sedation. Reversal of intramuscularly administered drugs, if possible, by intravenously administered flumazenil or naloxone, would be

somewhat successful for a period of time. However, as a reservoir of the IM drug exists within the muscle into which it was injected previously, the drug will continue to be absorbed over several hours leading to the likelihood of a rebound sedation effect occurring as the action of the intravenously administered reversal agent diminishes. IM administration of the reversal agent is generally not recommended as its onset, 10–20 min, is too slow to provide any immediate relief in the event of over-sedation. Additionally, the degree of the intramuscularly administered reversal agent's effectiveness would be less than that seen following its administration IV.

IM drugs are not as controllable as intravenously administered drugs. Sedation should, therefore, be limited to a moderate level, with the doctor trained to recognize and manage (e.g., rescue) the patients should they inadvertently enter deep sedation. Patients receiving IM drugs for sedation must also maintain NPO status prior to sedation.

Recovery from IM drugs is prolonged; thus, the patient must have an adult escort when being discharged from the office. This being said, it would seem that IM drugs have few indications in dentistry. However, used judiciously by the doctor trained in parenteral moderate sedation (IV, IM, IN), the technique can be employed with a likelihood of success of about 67%. Midazolam is the most commonly employed IM CNS-depressant drug in moderate sedation.

Intranasal

The IN administration of CNS-depressant drugs is relatively new within medicine and dentistry, though it has been used for many years by drug abusers (e.g., cocaine) and for the administration of nasal decongestants.

Nasal mucosa is highly vascular; therefore, absorption of drugs instilled into the nares is more rapid than for other routes of administration. Lam and colleagues[27] compared the efficacy, in children, of IN midazolam with that of its IM administration, finding the level of sedation to be comparable. IN midazolam has also been adopted in emergency medicine in the management of acute seizures in pediatric patients.[33] Primosch[34] compared nasal drops versus nasal spray as a technique of IN drug administration and found that although the effectiveness of moderate sedation was not influenced by the method of nasal administration, spray administration produced significantly less aversive behavior than administering drops in 2- to 3-year-old dental patients of similar behavioral characteristics.

Most studies and clinical usage of the IN route of drug administration appear in pediatrics, there being few reports of the use of this technique, and of its effectiveness, in adults, thereby limiting its utility in most endodontic cases.

The advantages of IN drug administration include its more rapid onset of action (compared with enteral routes) and the lack of need for injection and the accompanying trauma, both psychological and physical, associated with it.

Disadvantages of IN drug administration are similar to those of IM. IN drugs cannot be titrated. Dosages are based on weight (mg/kg). Along with the inability to titrate IN drugs, it is also not possible to rapidly lessen or deepen the level of the resultant CNS depression. Reversal of intranasally administered drugs, if possible, by intravenously administered flumazenil or naloxone, would be somewhat successful. IM or IN administration of a reversal agent is generally not recommended as its onset, 10–20 min, is too slow to provide any immediate relief in the event of over-sedation. Additionally, the efficacy of an IM or IN reversal agent would be less than that seen with its IV administration.

IN drugs are not as controllable as intravenously administered drugs. The level of CNS depression sought should, therefore, be limited to moderate, with the doctor trained to recognize and manage (e.g., rescue) the patients should they enter into deep sedation. Patients receiving IN drugs for sedation must also maintain NPO status prior to sedation.

Recovery from IN drugs is prolonged, thus the requirement for an adult escort for the patient when discharged from the office.

Utilization of the techniques of moderate sedation enable the endodontist to manage the dental fears of the overwhelming majority of odontophobic patients. Table 18-5 illustrates the possible uses of the techniques of moderate sedation in endodontic practice. Unfortunately, not all fears can be successfully managed with moderate sedation techniques. For these few patients, general anesthesia is required.

General Anesthesia

General anesthesia is the controlled loss of consciousness, the final step in our continuum of CNS depression that started with minimal sedation. The success rates of the sedation

TABLE 18-5 Techniques of Moderate Sedation–Summary

	Preoperative		Perioperative	Ability to titrate	CNS depression level when used alone	CNS depression level used in combination
	Night prior to treatment	Morning of treatment				
Oral sedation	Yes	Yes	Yes	No	Min, mod	Min, mod, deep, GA
Inhalation			Yes	Yes	Mod	Mod, deep, GA
IV			Yes	Yes	Mod, deep, GA	Mod, deep, GA
IM/IN			Yes	No	Mod, deep, GA	Mod, deep, GA

Abbreviations: Deep, deep sedation; GA, general anesthesia, Min, minimal sedation; Mod, moderate sedation.

techniques described previously, along with the amnestic properties of some of the drugs employed, specifically the benzodiazepines midazolam and triazolam, has decreased the need for general anesthesia as a method of managing significant numbers of odontophobic patients.

All 50 states in the United States regulate the use of general anesthesia in dentistry.[15] A residency in anesthesiology of 3-years duration represents the current educational requirement. As of April 2015, 10 residency programs in anesthesiology for dentists were accredited by the American Dental Board of Anesthesiology.[35]

Doctors who have completed accredited residencies becoming dentist anesthesiologists provide anesthesia services for other dentists either (1) by establishing a free-standing outpatient surgical center, in which the endodontist (or any other dentist) brings the odontophobic patient to the surgical center to carry out the scheduled procedures while the patient receives general anesthesia provided by the dentist anesthesiologist, or (2) by traveling to the office of the dentist who will be managing the dental needs of the fearful patient, wherein dentist anesthesiologists bring along all of their anesthesia drugs, equipment, and expertise.

Once the dentist anesthesiologist has induced general anesthesia, the dentist can then proceed with the endodontic procedure on a now more nearly "ideal" patient.

Management of Perioperative Pain in the Endodontic Patient

Peter Milgrom wrote, "Deal with the fear first, then pain will be a minor problem."[36] The prevention of treatment-related pain occurring during the extirpation of pulpal tissues is a not uncommon problem. In a survey of 121 dentists, 31% stated they had encountered a problem "often" or "sometimes" when treating mandibular premolars, while 55% had the same difficulties with mandibular molars in all clinical situations.[37] Molars were the only maxillary teeth proving difficult to anesthetize (16%).[37]

Asked about difficulties obtaining adequate anesthesia with various types of dental disease, the majority indicated frequent difficulty when treating exacerbated chronic pulpitis (69%) and acute pulpitis (74%).[37]

When fear of dentistry is added to this clinical situation, the likelihood of obtaining the level of clinical anesthesia required to painlessly enter into the pulp chamber and extirpate pulpal tissues is markedly diminished.

Ignoring a patient's fear only exacerbates this situation as the patient will, remain overly alert throughout the procedure, responding to even the most usually innocuous of stimuli (e.g., sound, vibration, pressure) as though they were painful.

Pain Control in Endodontics the Local Anesthetic Drugs

Local anesthetics are the safest and most effective drugs in medicine for the prevention and management of pain. Local anesthetics are also the only drugs that actually prevent nociceptive impulses from reaching the brain where they are interpreted as pain.

Five local anesthetics are employed by the dental profession: articaine HCl, bupivacaine HCl, lidocaine HCl, mepivacaine HCl, and prilocaine HCl.*

Table 18-1 lists local anesthetic formulations currently available in North America (April 2015) based on their expected duration of pulpal anesthesia (in the nonpulpally-involved tooth). In dentistry in the United States, lidocaine (49%) and articaine (35%) represent approximately 85% of local anesthetic usage (December 2014).[38]

"Plain" local anesthetic formulations, mepivacaine 3% or prilocaine 4%, are unlikely to provide either the depth or duration of pulpal anesthesia necessary to successfully (e.g., painlessly) extirpate the pulp of an infected tooth, especially the mandibular molar.[39] As all injectable local anesthetics are inherently vasodilators (arterioles, capillaries), nonvasoconstrictor-containing formulations produce an increase in blood flow at the site of drug deposition. Increased blood flow leads to three potentially negative outcomes: (1) less profound anesthesia, a result of a smaller volume of local anesthetic entering into the nerve; (2) a shorter duration of action, as the local anesthetic diffuses out of the nerve more quickly; and (3) a higher local anesthetic blood level, potentially increasing the risk of overdose when large doses are administered.

Inclusion of a vasoconstrictor, epinephrine, or levonordefrin (in the United States), in the anesthetic solution negates these actions by decreasing blood flow at the site of drug administration. This allows a greater amount of local anesthetic to diffuse into the nerve where it remains for a longer time, resulting in an increased depth of anesthesia; longer duration of pulpal anesthesia; and a decreased local anesthetic blood level (e.g., decreased risk of overdose).

Concentrations of epinephrine required to provide these beneficial effects are minimal, usually 1:100,000 (articaine, lidocaine), or 1:200,000 (articaine, bupivacaine, prilocaine). Use of epinephrine 1:50,000 (lidocaine) should be limited solely to the provision of localized areas of hemostasis during surgical procedures involving soft tissue. Only a few drops of the solution need be infiltrated directly into the surgical site to produce this action.

Lidocaine 2% with Epinephrine 1:100,000 or 1:50,000

This drug will provide duration of pulpal anesthesia of approximately 60 min in the nonpulpally involved tooth, with duration of soft tissue anesthesia ranging from 3 to 5 h. Onset of pulpal anesthesia is usually listed as developing in from 3 to 5 min following injection. As there is no clinical difference in the quality (depth, duration) of anesthesia between the two concentrations of epinephrine,

*All local anesthetics are used in their water-soluble acid salt form. Therefore the correct designation for all LAs should be: lidocaine HCl, mepivacaine HCl, and so on. For the sake of brevity they will be referred to simply by their name, for example, lidocaine, mepivacaine.

use of the 1:100,000 is recommended for pain control purposes. The only recommended use of 1:50,000 epinephrine is hemostasis. Both lidocaine formulations are appropriate for pulpal anesthesia when extirpating the pulp of an infected tooth, though the 1:100,000 formulation is still recommended.

Articaine 4% with Epinephrine 1:200,000 or 1:100,000

This drug is similar to lidocaine in both the onset and duration of (soft and pulpal) anesthesia. As there is no clinical difference in anesthetic onset, depth, or duration between the two articaine formulations, it is suggested that the 1:200,000 formulation be used for pain control purposes, though the author of this chapter has no qualms employing the 1:100,000 formulation of articaine for pain control. Both articaine formulations are appropriate for pulpal anesthesia when extirpating the pulp of an infected tooth.

Though scientific evidence of a superiority of articaine over the other commonly used local anesthetics is lacking,[40,41] anecdotal reports from endodontists indicate a perceived increased rate of successful pulpal anesthesia. However, there is evidence that administration of articaine by mandibular infiltration in adult patients results in more successful anesthesia used either as a sole injection or as a supplement to inferior alveolar nerve block (IANB) when compared with lidocaine.[42,43] The use of articaine by buccal infiltration in the mandible is discussed in the following technique section.

Mepivacaine 3% ("Plain") or Mepivacaine 2% with Levonordefrin 1:20,000

Mepivacaine 2% with levonordefrin is quite similar in its clinical characteristics to both lidocaine and articaine with epinephrine. Levonordefrin is used as a 1:20,000 concentration, providing clinical actions similar to epinephrine 1:100,000, though its cardiovascular effects are not as pronounced.[44] Mepivacaine 2% with levonordefrin is an appropriate local anesthetic when extirpating the pulp of an infected tooth.

Mepivacaine 3% ("plain") is a short-acting local anesthetic providing between 20 and 40 min of pulpal anesthesia. Infiltration of mepivacaine 3% will provide approximately 20 min of pulpal anesthesia but up to about 40 min when administered by nerve block. The depth of pulpal anesthesia is not as profound as the levonordefrin-containing solution and therefore is not commonly selected for administration when extirpating the pulp of an infected tooth.[39] However, the pH of 3% mepivacaine is higher (~6.5) than vasoconstrictor-containing formulations and many dentists take advantage of this by administering a fraction of a cartridge (e.g., about 1/4–1/3) prior to the administration of a vasoconstrictor-containing solution to make the injection more comfortable. The pH of a local anesthetic containing a vasoconstrictor is approximately 3.5.

Prilocaine 4% ("Plain") or Prilocaine 4% with Epinephrine 1:200,000

Prilocaine is available as both 4% "plain" formulation and 4% with 1:200,000 epinephrine. The epinephrine-containing formulation provides approximately 60 min of pulpal and from 3 to 5 h of soft tissue anesthesia. Prilocaine was the first local anesthetic formulation to have 1:200,000 epinephrine—still the least concentrated epinephrine solution available in dentistry in the United States. It is an appropriate formulation for pulpal anesthesia when extirpating the pulp of an infected tooth.

Prilocaine 4% "plain," like mepivacaine "plain' provides a relatively short duration of not as profound pulpal anesthesia. It is, therefore, less likely to provide pulpal anesthesia profound enough to permit painless access to, and extirpation of, an infected pulp. Also like mepivacaine "plain," the duration of pulpal anesthesia observed with prilocaine 4% plain differs significantly when administered by infiltration (~10–20 min) or nerve block (NB) (up to 60 min).

Both formulations of prilocaine 4% have been associated with a perceived increased risk of paresthesia when administered by IANB.[45]

Bupivacaine 0.5% with Epinephrine 1:200,000

Bupivacaine 0.5% with epinephrine 1:200,000 is a long-duration formulation when administered by nerve block. Though providing a longer duration of pulpal anesthesia by infiltration than any of the other local anesthetic formulations, it is following NB where bupivacaine stands out. Pulpal anesthesia of up to 6 h duration has been seen in some clinical trials following IANB.[46] One negative to the use of the drug as a primary agent for pain control is its prolonged time of onset of anesthesia (6–10 min). However, the primary use of bupivacaine in dentistry has been as an integral part of the protocol for the prevention of postsurgical pain[47] (Table 18-2).

Table 18-3 summarizes the currently available local anesthetic formulations as related to their onset, duration of soft tissue and pulpal anesthesia, and recommended maximum dosage.

BUFFERED LOCAL ANESTHETIC SOLUTIONS

A vasoconstrictor is added to injectable local anesthetics to increase both the depth and duration of pain control as well to increase their safety (by retarding their diffusion into the cardiovascular system, resulting in lower anesthetic blood levels thereby minimizing the risk of local anesthetic overdose). Vasoconstrictor-containing local anesthetic solutions are highly acidic, with a pH of approximately 3.5. "Plain" local anesthetic solutions have a pH of approximately 6.5.

More acidic local anesthetic solutions have a slower onset of action compared to "plain" drugs as a result of a smaller percentage of the un-ionized form (RN) of the local

anesthetic present in the solution. The RN form of the local anesthetic is lipid-soluble—able to diffuse through the lipid-rich nerve membrane into the nerve to then block sodium (Na^+) channels, producing anesthesia. In acidic solutions hydrogen ions (H^+) are present. The more acidic the solution the greater the number of H^+ present. Some H^+ attach to RN forming RNH^+ an ionic form that is unable to cross the nerve membrane. With lidocaine, at a pH of 3.5, 0.004% of the ions present in solution are RN. (Table 18-4). Once injected, the body slowly buffers the local anesthesia (LA) solution toward the physiologic pH of 7.35–7.45, a process that may require up to 45 min following IANB.[48] At a pH of 7.4, 24.03% of lidocaine is present in the RN-form, able to penetrate the nerve membrane.

This process can now be accelerated through buffering (e.g., alkalinizing) the dental local anesthetic solution immediately before its administration with a stabilized sodium bicarbonate solution.[48] The process of buffering a dental local anesthetic cartridge takes between 3 and 5 s.[49]

Time for onset of pulpal anesthesia was compared between lidocaine 2% with epinephrine 1:100,000 (pH 3.5) and buffered lidocaine 2% with epinephrine 1:100,000 in a prospective, randomized, double-blinded trial. Onset of pulpal anesthesia (assessed by electric pulp tester) was 6 min 37 s for the "traditional" local anesthetic solution and 1 min 51 s for the buffered lidocaine solution.[50] Seventy-one percent of the buffered local anesthetics achieved pulpal anesthesia in less than 2 min, only 12% of the traditional anesthetics achieved this.

The injections of buffered lidocaine were more comfortable (VAS† of 0.6) compared to the unbuffered lidocaine injections (VAS = 2.3). Anecdotal reports from dentists, particularly endodontists, indicate that there is an increased efficacy of buffered lidocaine when anesthetizing "hot" mandibular molars, compared with unbuffered LA solutions.

The United States Food and Drug Administration has approved local anesthetic buffering only for lidocaine, (as of April 2015), however all other dental local anesthetics have been buffered with success comparable to that found with lidocaine.

Buffered local anesthetic solutions appear to have an important place in the pain management armamentarium of endodontists.

Pain Control in Endodontics-Maxillary Technique

The basic injection technique employed for maxillary anesthesia, by most dentists, is the ***infiltration*** (supraperiosteal). The buccal cortical plate of bone in most adult patients is thin and porous enough for local anesthetic solution deposited at the apex of a tooth to diffuse through it and block nerve conduction. Infiltration anesthesia requires deposition of approximately 0.6 mL of solution at, or above, the apex of the tooth to be treated. A 27-gauge short dental needle is recommended.

The most difficult teeth to anesthetize in the maxilla are molars, especially following infiltration anesthesia. If the palatal root of the first or second maxillary molar extends ("flares") more toward the palate than "normal," infiltration of a solution of lidocaine, mepivacaine, prilocaine, or bupivacaine may not diffuse far enough medially, failing to anesthetize the palatal root, causing the patient to experience pain when that area of the tooth is treated. Infiltration of articaine will, in most cases, provide anesthesia of the flared palatal root, evidence of its greater lipid-solubility and ability to diffuse through soft and hard tissues better than other local anesthetics.

Where maxillary infiltration is unsuccessful (e.g., thicker than normal cortical bone; presence of apical infection, flared palatal root of molar), NB anesthesia is suggested. Maxillary nerve blocks include the anterior superior alveolar (ASA) NB; posterior superior alveolar (PSA) NB; anterior middle superior alveolar (AMSA) NB; and the second division (V_2) NB.

Anterior Superior Alveolar (ASA) NB

This technique is often (mistakenly) termed the "infraorbital" NB. (The infraorbital nerve is a terminal branch of the maxillary division of the trigeminal nerve, and provides anesthesia only to the soft tissues of the lower eyelid, lateral border of the nose, and upper lip). The ASA nerve provides anesthesia to the maxillary incisors, canine and, in most patients, the premolars.

Following negative aspiration, a volume of 0.9–1.2 mL of anesthetic solution is deposited outside the infraorbital foramen (completing the infraorbital NB). The most significant part of the technique is as follows: the application of digital pressure over the infraorbital foramen for 1–2 min, forcing the local anesthetic into the foramen, blocking the ASA nerve. A 27-gauge short needle is recommended.

Posterior Superior Alveolar (PSA) NB

A 27-gauge short needle is inserted into the buccal fold adjacent to the second maxillary molar. The syringe is oriented in an "inward, upward, and backward" direction and the needle advanced 16 mm. The needle tip should now be located just posterior and medial to the maxillary tuberosity, in the pterygomaxillary space, where the PSA nerves are found. Following negative aspiration a volume of 0.9 mL of anesthetic is administered.

Anterior Middle Superior Alveolar (AMSA) NB

Computer-controlled local anesthetic delivery systems have enabled local anesthetic injections to be delivered more comfortably.[51] As a result of their studies, the AMSA NB was developed. The AMSA NB provides pulpal anesthesia to the maxillary incisors, canine, and premolars; their supporting buccal soft tissues and bone; as well as soft tissue anesthesia of the entire palate on the side of injection. No extraoral anesthesia occurs (e.g., lip, anterior face).[52,53]

†VAS—visual analog scale. "0" "I felt nothing"; "10" "the worst pain ever experienced." VAS readings of 3 or lower are considered "comfortable."

A 27-gauge short needle is recommended. Needle insertion is on the hard palate about halfway along an imaginary line connecting the midpalatal suture to the free gingival margin between the first and second maxillary premolars.[54] A volume of 1.4–1.8 mL is slowly administered following negative aspiration.

The AMSA NB would be indicated for use when soft tissues in the maxillary buccal fold of the tooth to be treated show evidence of infection.

Maxillary (V_2) NB

The V_2 NB is indicated primarily when anesthesia of the entire hemimaxilla is required—a rare event in endodontics. However, when other techniques—infiltration, nerve block, intraosseous (IO)—have failed or are contraindicated, the V_2 block should be considered. Two approaches are described—a "high tuberosity' approach and the "greater palatine canal" approach. The "high tuberosity" approach is easier to administer but is associated with a higher positive aspiration rate and risk of hematoma, while the "greater palatine canal" is more difficult to master but is associated with fewer complications.[55] Both approaches to the V_2 NB are described in detail in dental local anesthesia texts.[55] Table 18-6 summarizes drug administration techniques.

Pain Control in Endodontics—Mandibular Technique

It is in the mandible where most significant problems in achieving effective pain control arise. Thirty-one percent of doctors (37 of 121 doctors surveyed) stated they had difficulty achieving pain control "often" or "sometimes" treating mandibular premolars while 55% (67 of 121) mentioned the

TABLE 18-6 Comparison of Routes of Drug Administration

Route of Drug Administration	Onset of Action	Titrate Yes–No	Advantages	Disadvantages
Inhalation	Rapid (~20 s)	Yes	Patient need not be NPO; rapid onset; titration; rapidly increase or decrease level of CNS depression; full recovery in most patients; no prohibitions of postoperative functions	Patient cooperation required; ineffective if unable to breathe through nose
Oral	Slow (~1 h for maximal clinical effect)	No	Easy for patient; easy for doctor	Patient cooperation needed; slow onset; inability to titrate; erratic absorption from GI tract; significant hepatic 1st-pass effect for some drugs; no control over ultimate level of CNS depression; inability to quickly increase or decrease level of CNS depression; inability to reverse CNS depression; prolonged recovery; requirement for escort for patient on leaving dental office
Parenteral: Intramuscular	Intermediate (10–20 min)	No	More reliable absorption than oral; minimal patient cooperation required	Patient must be NPO; potential needle injury; relatively slow onset; inability to titrate; no control over ultimate level of CNS depression; inability to quickly increase or decrease level of CNS depression; inability to reverse CNS depression; prolonged recovery; requirement for escort for patient on leaving dental office
Parenteral: Intravenous	Rapid (~20 s)	Yes	Rapid onset; titration; rapidly increase level of CNS depression; most drugs reversible	Venipuncture is learned technique; patient must be NPO; inability to quickly decrease level of CNS depression; prolonged recovery; requirement for escort for patient on leaving dental office
Parenteral: Intranasal	Intermediate (10–20 min)	No	More reliable absorption than oral; minimal patient cooperation required	Unpleasant (bitter) taste if liquid enters oral cavity; may irritate nasal mucosa; inability to titrate; no control over ultimate level of CNS depression; inability to quickly increase or decrease level of CNS depression; inability to reverse CNS depression; prolonged recovery; requirement for escort for patient on leaving dental office

same for mandibular molars, in all treatment circumstances, not solely endodontically-involved teeth.[37]

Incisive NB

Anesthesia of the premolars, canine, and incisors can easily be achieved in most clinical situations using the incisive NB(often incorrectly termed the "mental" nerve block).

A 27-gauge short needle is recommended. The mental foramen is located based on a well angulated radiograph or by palpation in the buccal fold. It is most often located at or below the apex of the second mandibular premolar. The needle is inserted into the lowest portion of the buccal fold at about the first premolar and then advanced until the needle tip lies outside the mental foramen (the syringe should be kept below the patient's line-of-sight for psychological reasons). Following negative aspiration 0.6–0.9 mL of local anesthetic is deposited slowly. This completes the mental NB. As is the case with the previously discussed ASA NB, the most important aspect of this technique is the application of finger pressure over the site of local anesthetic deposition for minimally 1 min, preferably 2, forcing local anesthetic into the mental foramen, blocking the incisive nerve at that location. All teeth anterior to this location will be pulpally anesthetized.

The incisive NB will provide clinically adequate pulpal anesthesia in essentially all clinical situations, leaving us to consider those "darned" mandibular molars.

Inferior Alveolar NB

The IANB is the traditional technique employed by dentists worldwide when seeking to achieve mandibular anesthesia. Yet this technique has a relatively low success rate overall, and even more so in pulpally involved teeth. In situations of exacerbated chronic pulpitis, 69% (84 of 121) had anesthetic difficulties (almost always, often or sometimes) with 74% mentioning similar problems with acute pulpitis.[37]

There are many approaches to the IANB; the most commonly used involving placing the barrel of the syringe in the corner of the patient's mouth on the contralateral side. The appropriate intraoral landmark is located and the needle (a 25- or 27-gauge long needle is strongly recommended‡) is advanced until bone is gently contacted on the lingual aspect of the mandibular ramus. Following two negative aspirations, a volume of 1.5 mL of local anesthetic is deposited slowly. The syringe is withdrawn and the needle reinserted into the soft tissue in the buccal fold just distal to the last mandibular molar to anesthetize the buccal nerve. Following negative aspiration the remaining 0.3 mL of anesthetic solution is deposited. This buccal NB provides anesthesia to the buccal soft tissues distal to the mental foramen.

Given the significant failure rate of the IANB, other approaches to mandibular anesthesia have been developed including the Gow-Gates mandibular NB; the Akinosi-Vazirani (or Vazirani-Akinosi) closed-mouth mandibular NB, the periodontal ligament (PDL) injection, IO anesthesia; and the administration of articaine by buccal infiltration in the mandible either as a sole injection or as a supplement to a previously administered IANB.

Gow-Gates Mandibular NB

In 1973 an article describing a "high" technique of mandibular anesthesia was published.[58] The technique, using extraoral landmarks, described depositing local anesthetic solution at the lateral aspect of the neck of the mandibular condyle. This technique now known as the "Gow-Gates technique," has become popular. Simply put, the most common reason for an experienced dentist to miss the IANB is deposition of the local anesthetic *below* the mandibular foramen. The nerve is "gone," having disappeared into mandibular bone that is too thick to permit local anesthetic diffusion to occur. We have "missed." In most situations the doctor goes back and administers a second IANB at a site just slightly higher (5–10 mm) than the initial tissue puncture, and profound anesthesia develops. We have learned, through trial and error, that this patient's mandibular foramen is "higher" than the average patient's. "A higher injection is better."

Dr. Gow-Gates took this concept of "higher is better" to an extreme, depositing local anesthetic at the neck of the mandibular condyle. The mandibular division of the trigeminal nerve (V_3) has just exited foramen ovale and, with the patient's mouth opened wide, V_3 is lying close to the mandibular condyle. Using a 25- or 27-gauge long dental needle the depth of soft tissue penetration to bone is approximately 25 mm. The Gow-Gates mandibular NB is described more fully in textbooks of local anesthesia and videos.[59,60] If bone is not contacted, local anesthetic should not be administered. Once mastered, the Gow-Gates technique has a higher success rate than the IANB.[61]

Akinosi-Vazirani, or Vazirani-Akinosi, Closed Mouth Mandibular NB

In clinical situations where a patient has a unilateral trismus (spasm of the muscles of mastication) and is unable to open their mouth, neither the IANB nor Gow-Gates techniques can be administered. If this patient presents in pain, requiring endodontic treatment on a mandibular molar, this closed-mouth mandibular block technique can be employed with a rather high success rate for pain control, but with an even higher success rate at blocking the motor portion of V_3.[62,63] Blocking the motor portion of V_3 allows the patient to open their mouth. If pain control has not also occurred, then other techniques (IANB, Gow-Gates) can now be employed.

‡ All-too-often dentists will use a 30-gauge short needle for the IANB in adult patients. This author strongly recommends ***NOT*** doing so. Though rare, dental needles can break. If a needle is inserted into soft tissue to its entire length (to its hub), when it breaks (breaking *always* occurs at the hub) the needle fragment is "lost." Retrieval is usually difficult and is fraught with significant complications.[56,57] In a review of broken needle reports Malamed et al found all involved short needles with 95% being 30-gauge. Of 86 reports of broken needles, 92% occurred with IANB.[56]

A 25- or 27-gauge long dental needle is recommended. With the patient's teeth gently in contact, the barrel of the syringe is placed adjacent to the maxillary buccal fold. The needle is inserted into the soft tissue on the lingual aspect of the mandibular ramus at a site directly across from the maxillary tuberosity. The height of needle insertion is that of the mucogingival junction of the last maxillary molar. With the needle bevel facing ***away*** from bone the needle is advanced 25 mm. This should place it in the vicinity of V_3. As no bone is contacted (the needle is advanced parallel to the mandibular ramus) this depth of needle penetration is only an approximation. If unsuccessful, a second attempt is made with insertion being either at a greater depth or lesser depth, as determined by the doctor. The Akinosi-Vazirani technique deposits local anesthetic at a position midway between that of the IANB (lowest) and Gow-Gates (highest). Table 18-5 summarizes mandibular injection techniques.

Pain Control in Endodontics—Alternative Techniques

The injection techniques described previously will provide effective pain control in virtually all clinical situations. However there will be occasions when successful pain control remains elusive. This is particularly likely to occur with mandibular molars. The techniques described in the following may be used in either the maxillary or mandibular arches but have been most often used in the latter, as mandibular molars are the most difficult teeth to fully anesthetize.

Periodontal Ligament Injection

The PDL injection, also known as the PDL or intraligamentary injection, can be used as a sole injection or as a supplement to a partially successful NB or infiltration.[64] In endodontics, the PDL is most commonly used to supplement a partially successful IANB where a small, isolated area of the tooth remains sensitive during treatment, while the remainder of the tooth is insensitive. In this example, the mesial aspect of a mandibular first molar remains sensitive during treatment. A 27-gauge short needle is recommended. The bevel of the needle is placed into the periodontal space on the mesial aspect of the first molar, either the mesiobuccal or mesiolingual. With the bevel facing the root of the tooth the needle is advanced into the PDL space until firm resistance is encountered. A volume of 0.2 mL of local anesthetic is deposited slowly. Successful PDL injection is predicted on the following: (1) significant resistance to the drugs' deposition; (2) ischemia of the soft tissues in the area (this will occur whether or not the anesthetic solution contains a vasoconstrictor). The onset of anesthesia is virtually instantaneous. However the duration of pulpal anesthesia is impossible to determine because of the small volume of local anesthetic administered. If necessary, additional anesthetic may be administered during the procedure.

The PDL injection should not be administered into an area where clinical or radiographic evidence of infection is present. Additionally the PDL injection may not be as effective in the presence of periapical infection.

The PDL injection is a variation of IO anesthesia (see following discussion).

Intraosseous Injection

IO anesthesia has been demonstrated to be a valuable adjunct to achieving pain control in difficult clinical situations.[65–67] The thickness of the cortical plate of bone in the adult mandible, of most patients, precludes successful administration of infiltration anesthesia. The mental foramen, usually situated apical to the second premolar, permits local anesthetic access to the incisive nerve, blocking nerve impulses originating anterior to this location (e.g., incisive NB).

Creating a small perforation in the cortical plate of bone distal to the tooth to be treated permits a short 27-gauge dental needle to be painlessly inserted. Local anesthetic solution is slowly administered. For "hot" mandibular teeth it is recommended that one cartridge (1.8 mL) be administered.[68]

Because of the vascularity of this area it is not uncommon for patients to experience palpitations whenever a vasoconstrictor-containing local anesthetic is used. As vasoconstrictor-containing local anesthetics provide a longer duration and more profound anesthesia than "plain" local anesthetics, it is recommended that a 1:200,000 epinephrine concentration be selected.[69,70]

Using a local anesthetic containing epinephrine should provide pulpal anesthesia of approximately 30 min duration while a "plain" solution should provide approximately 15 min of not as profound anesthesia.

The IO procedure is relatively comfortable. Local infiltration of the soft tissue at the site of the injection is recommended. Perforation of the interproximal bone is almost always entirely atraumatic.

The technique is described in greater detail in local anesthesia textbooks.[71]

Intrapulpal Injection

Prior to the introduction of IO anesthesia, buffered local anesthetics and articaine (see the following), achieving anesthesia profound enough to permit entry into the pulp chamber of infected mandibular (and on rare occasion, maxillary) teeth was problematic. More specifically, this problem occurred with mandibular molars because few alternative techniques were available with which the doctor could obtain profound anesthesia.

Deposition of local anesthetic directly into the coronal portion of the pulp chamber of a pulpally involved tooth provides effective anesthesia for pulpal extirpation and instrumentation where other techniques have failed. The intrapulpal injection may be used on any tooth when difficulty in providing profound pain control exists, but from a practical view it is necessary most commonly on mandibular molars.[72,73] Intrapulpal injection produces pain control both by the pharmacological action of the local anesthetic and

applied pressure. This technique may be used once the pulp chamber is exposed, either surgically or pathologically.

The needle is placed into the perforation in the roof of the pulp chamber (preferably a small opening) and a volume of 0.2–0.3 mL of local anesthetic is injected. Unfortunately, there will be a brief moment of pain experienced by the patient as the drug is administered.

Fortunately, today, with the availability of multiple anesthetic options, the need for intrapulpal injection has significantly decreased.

Articaine by Buccal Infiltration

Articaine 4% with epinephrine 1:100,000 or 1:200,000 has become the second most used local anesthetic in dentistry in the United States despite having only been introduced in June 2000,[38] and the fact that most well-designed clinical trials have concluded that articaine is "as safe and effective as lidocaine" (the drug to which it is most often compared).[74,75]

Following its introduction, numerous anecdotal reports were received from dentists claiming they no longer needed to administer the IANB to work in the adult mandible painlessly. They stated that mandibular infiltration with articaine HCl was uniformly successful. These claims were initially met with skepticism. However over the past 9 years, a number of well-designed clinical trials comparing infiltration in the adult mandible of articaine HCl 4% with epinephrine 1:100,000 to lidocaine 2% with epinephrine 1:100,000 or 1:80,000 clearly demonstrated articaine's superiority when administered as a sole injection for pulpal anesthesia.[76–78]

Of greater clinical significance for endodontics is the buccal infiltration of articaine as a supplement to theIANB, and other block techniques, that may be less than fully effective in blocking nerve conduction in "hot" mandibular molars. Kanaa et al.[79] administered IANBs (lidocaine 2%, epinephrine 1:80,000) to subjects on two occasions. Subjects then received either a buccal infiltration of articaine 4% with epinephrine 1:100,000 (2.0 mL) by the first molar or a "dummy" injection at the same site. The first molar, first premolar, and lateral incisor were tested using electric pulp tester (EPT) for 45 min. Significant increases in success were noted with articaine infiltration for the first molar and lateral incisor ($p = <0.001$) (Table 18-7) At the conclusion of the study (45 min) there was no indication of decreasing anesthetic efficacy in any of the three teeth tested[79] (Figure 18-4).

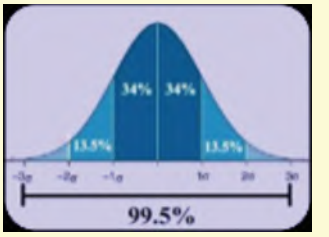
Greater success would be seen in "real life" clinical situations by supplementing an IANB with articaine buccal infiltration (0.9–1.2 mL) at the apex of the tooth to be treated. (In the clinical trial articaine was infiltrated by the first mandibular molar).

Suggested Local Anesthetic Technique Sequences for Maxillary and Mandibular Endodontically-Involved Teeth

It is this author's suggestion that the following sequences be considered when seeking painless entry into a "vital" pulp chamber. Both maxillary and mandibular sequences begin with the assumption that "all is good."(Tables 18-8–18-13).

TABLE 18-7 Local Anesthetics by Expected Duration of Pulpal Anesthesia

- Short-duration (~30 min)
- Lidocaine 2%, Mepivacaine 3%, Prilocaine 4%
- Intermediate-duration (~60 min)
- Articaine 4%, Lidocaine 2%, Mepivacaine 2%, Prilocaine or 4% (all with vasoconstrictor)
- Long-duration (>90 min)
- Bupivacaine 0.5% (with vasoconstrictor)



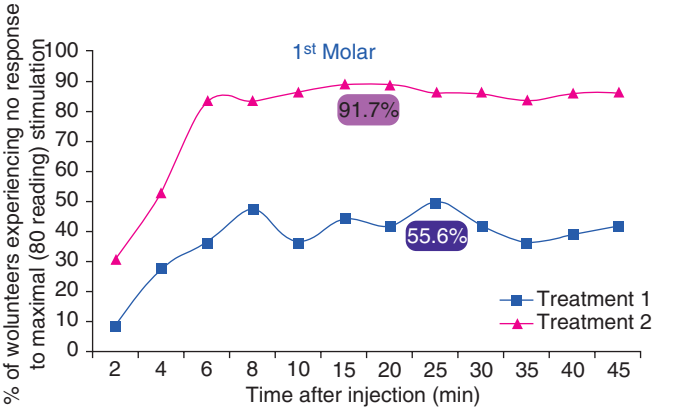


FIGURE 18-4 Duration of pulpal anesthesia for mandibular first molar following IANB with lidocaine 2% epinephrine 1:80,000 (blue line) versus IANB with lidocaine 2% epinephrine 1:80,000 with articaine 4% with epinephrine 1:100,000 buccal infiltration (pink line).[43]

TABLE 18-8 Suggested Pain Management Protocol

Pre-surgical NSAID po 1 h prior to appointment
- Ibuprofen 600 mg

LA of choice for surgery
- Articaine, Lidocaine, Mepivacaine, Prilocaine

Long-acting LA at end of surgery just prior to discharge of patient
- Bupivacaine

NSAID on timed basis (q4,6,8h) for xx days
- Ibuprofen 600 mg qid

Postsurgical telephone call early evening

Maxillary teeth:

1. Infiltration
   a. Preferably with buffered lidocaine or articaine
2. Appropriate NB (ASA, PSA, AMSA)
   a. Supplement NB with buffered articaine buccal infiltration (1.0 mL)
3. Consider sedation
   a. Inhalation sedation with N_2O–O_2 preferred

If necessary:

4. PDL injection

TABLE 18-9 Comparison of Available Local Anesthetic Formulations

Drug %	Vasoconstrictor	Mg LA/Cartridge	Onset (Pulpal)	Duration (Pulpal)	Duration (Soft Tissue)	MRD (FDA) mg/kg (Absolute Max)
Lidocaine 2%	Epi 1:50,000 Epi 1:100,000	36	3–5 min	60 min	3–5 h	7.0 (500)
Articaine 4%	Epi 1:100,000 Epi 1:200,000	72	3–5 min	60 min	3–5 h	7.0 (none listed) = USA 7.0 (500) = Canada
Mepivacaine 3%	None	54	3–5 min	20 min (infiltration) 40 min (NB)	2–3 h	6.6 (400)
Mepivacaine 2%	Levo 1:20,000 = USA Epi 1:100,000 = Canada	36	3–5 min	60 min	3–5 h	6.6 (400)
Prilocaine 4%	None	72	3–5 min	10–20 min (infiltration) 40–60 min (NB)	2–4 h	8.0 (600) = USA 8.0 (500) = Canada
	Epi 1:200,000	72	3–5 min	60 min	3–6 h	8.0 (600) = USA 8.0 (500) = Canada
Bupivacaine 0.5%	Epi 1:200,000	9	6–10 min	90 min (infiltration) up to 360 min (NB)	4–12 h	None listed (90) = USA 2.0 (90) = Canada

Abbreviations: Canadian MRDs from Health Canada; Epi, epinephrine; FDA = US Food & Drug Administration; Levo, levonordefrin; MRD, maximum recommended dose;

TABLE 18-10 Percentage of Un-ionized (RN) Local Anesthetics at Various pH Values

% Un-ionized (RN) LA				
pH	**Lidocaine pKa 7.9**	**Articaine pKa 7.8**	**Mepivacaine pKa 7.6**	**Bupivacaine pKA 8.1**
7.4 (body pH)	24.03	28.47	38.69	16.63
6.5 (plain)	3.83	4.77	7.36	2.45
3.5 (with epi)	0.004	0.005	0.008	0.003

TABLE 18-11 Summary of Maxillary Injection Techniques

Technique	Pulpal Anesthesia of …	Recommended Volume of Local Anesthetic (mL)
Infiltration (supraperiosteal)	Single maxillary tooth	0.6
Anterior superior alveolar NB	Incisors, canine, premolars	0.9–1.2
Posterior superior alveolar NB	Molars	0.9–1.8
Anterior middle superior alveolar NB	Incisors, canine, premolars	1.4–1.8
Maxillary (V_2) NB	Incisors, canine, premolars, molars	1.8

TABLE 18-12 Summary of Mandibular Injection Techniques

Technique	Pulpal Anesthesia of …	Recommended Volume of Local Anesthetic (mL)
Articaine infiltration	Single maxillary tooth	0.9–1.2
Incisive NB	Incisors, canine, premolars	0.6–0.9
Inferior alveolar NB	Incisors, canine, premolars, molars	1.5
Gow-Gates mandibular NB	Incisors, canine, premolars, molars	1.8
Akinosi-Vazirani (closed mouth) mandibular NB	Incisors, canine, premolars, molars	1.8

TABLE 18-13 Percentage of Successful IANB: Lidocaine Versus Lidocaine and Articaine Infiltration[43]

Anesthesia Success >2 Consecutive 80/80 Readings	IA + A-Caine Infiltration	IA + A-Caine Infiltration	IA + Dummy Infiltration	IA + Dummy Infiltration	McNemar Test *P*-Value
	Failure *n* (%)	Success *n* (%)	Failure *n* (%)	Success *n* (%)	
1st molar	3 (8.3)	33 (91.7)	16 (44.4)	20 (55.6)	<0.001
Premolars	4 (11.1)	32 (88.9)	12 (33.3)	24 (66.7)	0.021
Lateral incisors	8 (22.2)	28 (77.8)	29 (80.6)	7 (19.4)	<0.001

5. IO (rarely necessary in maxilla)
6. Intrapulpal (rarely necessary in maxilla)

Mandibular teeth:

1. Block technique: IANB, Gow-Gates NB, Akinosi-Vazirani NB
   a. Preferably with buffered lidocaine or articaine
2. Supplement NB with buffered articaine infiltration in buccal fold of tooth to be treated (1.0 mL)
3. Consider sedation
   a. Inhalation sedation with N_2O–O_2 preferred

If necessary:

4. PDL injection
5. Intraosseous
6. Intrapulpal

SUMMARY

Dental pain, anxiety, and fear exist and are a fact of life, especially in the specialty of endodontics. The primary obstacles to successful endodontic treatment are the presence of dual problems: pain and fear. To ignore fear, in the best of circumstances, will complicate dental treatment unnecessarily, as the patient remains an unwilling participant throughout the procedure. Fear lowers the pain reaction threshold, with odontophobic patients being unable to sit still during their treatment, adding to the frustration of the treating doctor. Fear increases catecholamine release into the cardiovascular system with a resultant rise in the incidence and severity of medical emergency situations.

The recognition of fear, and its management, removes a primary obstacle to the delivery of health care. Once fear is eliminated, clinically adequate pain control is normally accomplished quite readily through administration of local anesthetics, the safest and most effective drugs in medicine for preventing and managing pain.

The "hot" tooth, especially a mandibular molar, can also present a significant barrier to the delivery of painless dental care. Fortunately, today there are many options for the dentist to choose from. New local anesthetics (e.g., articaine); buffered local anesthetic solutions; newer techniques (e.g., Gow-Gates NB; Vazirani-Akinosi NB); and articaine administered by buccal infiltration in the mandible, all work to increase the likelihood of endodontic treatment being delivered painlessly.

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Chapter 19

Preparation for Treatment

C. John Munce, Terrell F. Pannkuk, Adham A. Azim, Rajiv G. Patel, Ziv Simon

CASE PRESENTATION AND TREATMENT PLANNING

Proper dentist–patient communication is an important aspect of any dental treatment. Thorough case presentation and truthful, complete answers to all of a patient's questions, fosters a warm and trusting relationship. This results in better patient compliance and a lower level of anxiety during treatment.[1] Patients presenting for endodontic treatment usually have many questions involving the nature of the procedure, the amount of pain expected during and after treatment, as well as the success rate and long-term prognosis of treatment.

The determination as to whether or not a tooth requires endodontic treatment is established during the initial examination. With the aid of an intraoral camera, radiography, cone-beam computed tomography (CBCT), and other imaging tools (see Chapter 9), the case can be fully presented to the patient. These high-tech diagnostic devices allow the dentist to thoroughly explain the reasons for the patient's current discomfort or condition, the need for intervention and the expected treatment outcome. The examination should not be limited to endodontic evaluation alone, but should include periodontal and restorative considerations as well (see Chapter 8).

Once the necessity for endodontic treatment has been established, nonsurgical root canal treatment or retreatment will most often be the recommended procedures. Occasionally, surgical intervention, such as apicoectomy or intentional replantation, may be advised to address unresolved periapical pathosis, obstructed canals, or other conditions not able to be addressed nonsurgically. The clinician should also be able to determine if further treatment might be required to ensure a good, long-term prognosis. Procedures such as surgical crown lengthening or orthodontic extrusion might be necessary to facilitate tooth isolation and subsequent restoration. In conjunction with endodontic treatment, guided bone regeneration may be required to restore the periodontal attachment and contribute to the longevity of the tooth.[2]

The clinician should be familiar with advanced restorative and periodontal procedures. Many teeth previously determined to be nonrestorable or considered likely to have a poor treatment outcome, can currently be saved with a higher degree of predictability (Figure 19-1). Decision-making should involve the entire team of collaborating clinicians, including the endodontist, periodontist, orthodontist, and restorative dentist as well as the patient. In the absence of severe pain or swelling requiring emergency endodontic intervention, treatment planning should be completed prior to initiating treatment. It is essential to collect all relevant diagnostic information prior to starting a course of treatment (Figure 19-2). In some instances, no treatment is advised and extraction is recommended (Figure 19-3).

Teeth requiring nonsurgical endodontic intervention may often present with an existing full coverage restoration. In cases where poor margins exist, the restoration should be removed and the restorability should be determined. If the margins are adequate, the clinician may attempt to maintain the restoration providing it does not interfere with access and visibility. The decision to remove or maintain an adequate full coverage restoration rests largely with the clinician and should be determined on a case-by-case basis.

Patients are often concerned about the time and number of appointments required to complete root canal therapy. The current evidence suggests that endodontic treatment may be completed in single (when indicated) or multiple visits with similar success rates and post-operative complications.[3–5] With advancements in endodontic armamentarium, completion of endodontic treatment in a single visit has gained popularity. Multiple visits, on the other hand, may be advantageous for many case types, including retreatment, calcified canals, broken instruments, post removal, cases with tortuous canals, and patient management cases to name a few. Multiple visits, however, are indicated in cases of severe symptoms, persistent canal exudate, resorption or when further canal disinfection is required through intracanal medication.[6–9] Inexperienced clinicians should anticipate extra time requirements for root canal treatment. In all cases, the length of any given treatment visit should not extend beyond the tolerance level of either the patient nor the dentist. When limits have been reached, intracanal medication should be placed (when appropriate), the tooth temporized and another appointment scheduled to continue the treatment.

INFORMED CONSENT

Endodontic treatment, like all dental treatment, must be preceded by informed consent. The dentist must thoroughly explain the proposed treatment, the benefits, the risks, and alternative treatments, including the option of no treatment at all.[10,11] Informed consent is a legal concept developed via case law and is enforceable through judicial actions. The prudent clinician will be careful to "inform before he or she performs."[12]

Informed consent is the patient's agreement to a proposed course of treatment, based on having received clear and understandable information about the potential benefits and risks of the proposed treatment. The dentist should ensure that the

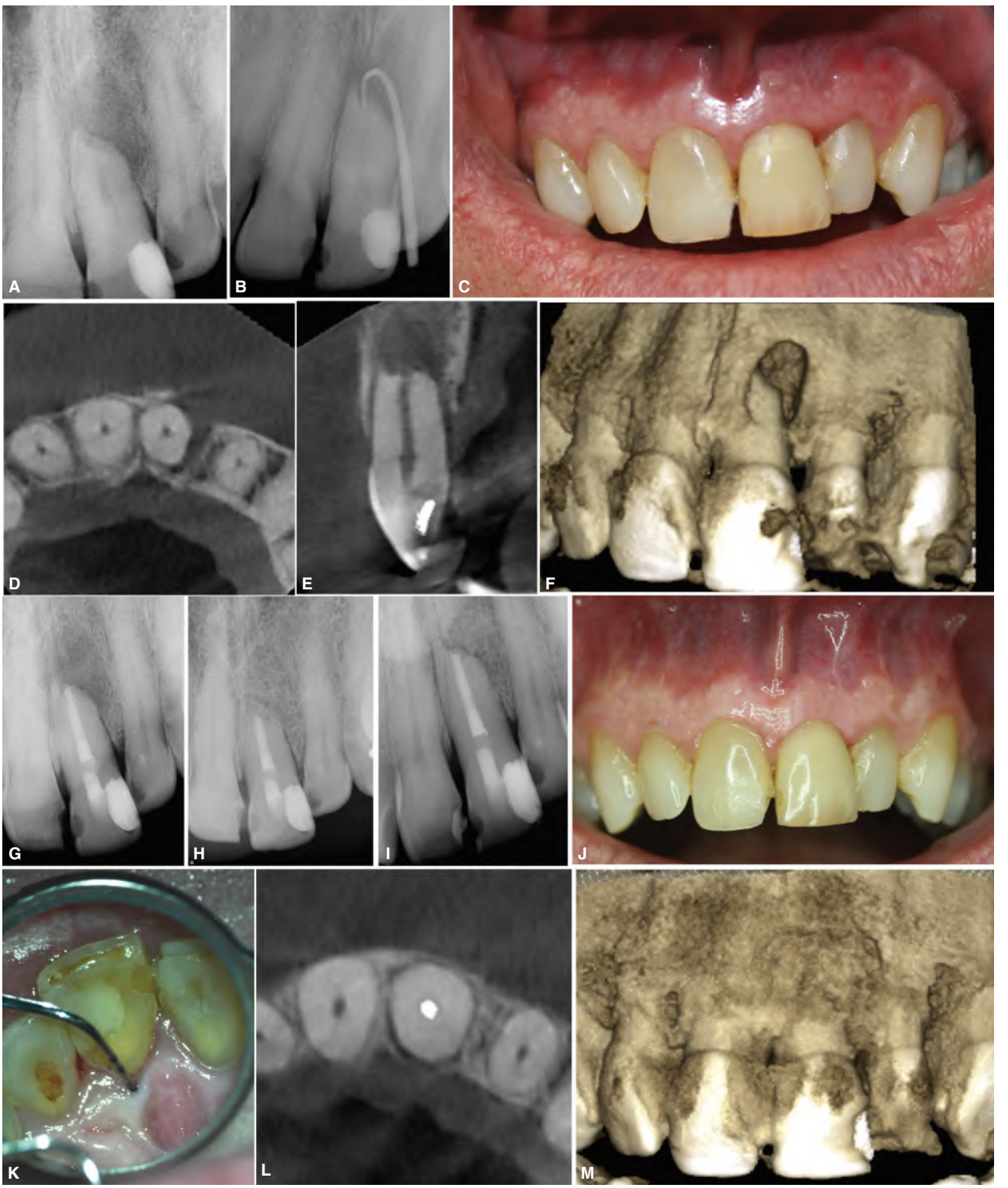

FIGURE 19-1 ***A–C***, Periapical radiograph of a maxillary central incisor with large periapical lesion and a sinus tract tracing to the apex that shows apical resorption. This tooth was associated with grade II mobility and probing to the apex on the palatal surface. ***D–F***, CBCT images showing loss of the buccal plate of bone and the interproximal bone support. In the sagittal view, the tooth appears to have a poor crown–root ratio. ***G,H***, Periapical radiographs following root canal treatment using MTA as an apical barrier. Patient then underwent surgical intervention to remove the periapical granuloma, and guided tissue regeneration (GTR) using allograft bone material and resorbable membrane was performed. ***I–K***, Six-month follow-up showing complete healing of the surgical site with no mobility and normal probing (3 mm) 360°. ***L,M***, Follow-up CBCT showing bone filling the entire osseous defect. (Courtesy of Dr. Adham A. Azim, Buffalo, NY.)

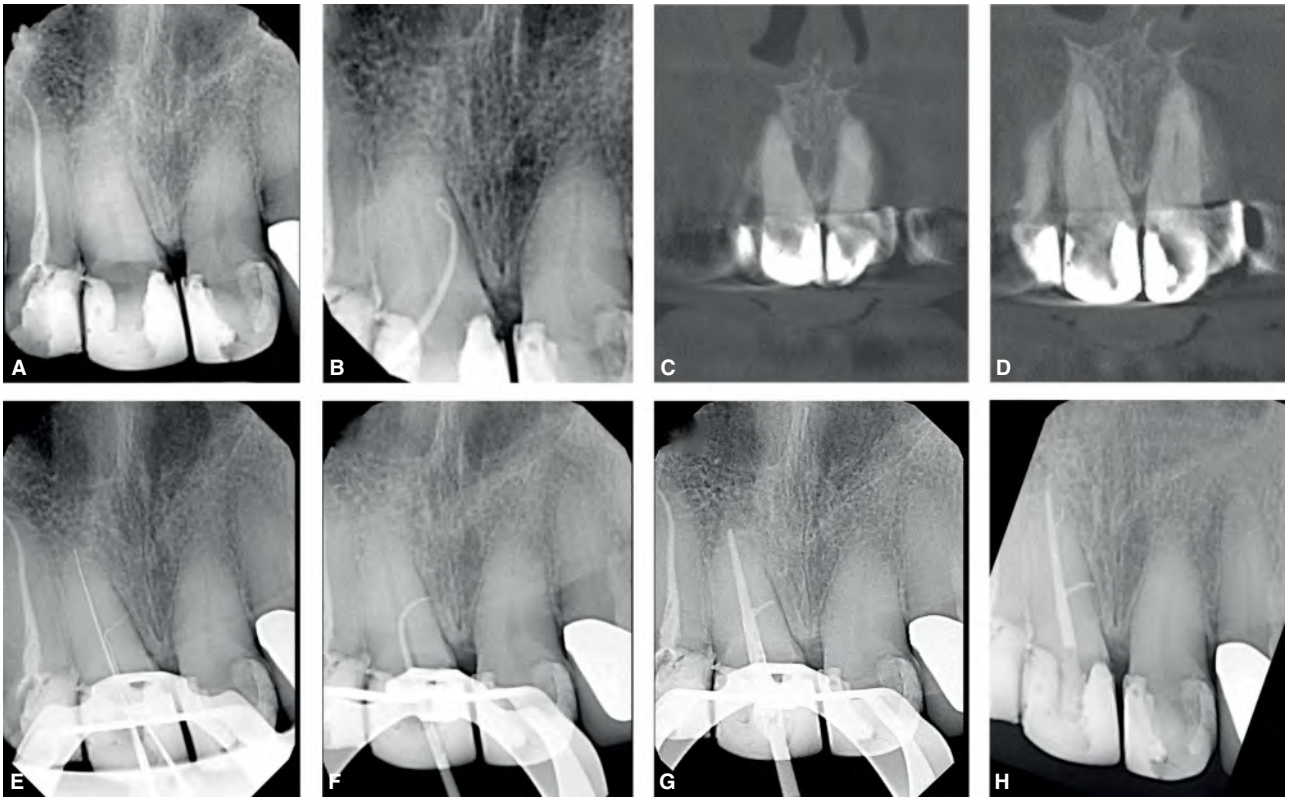

FIGURE 19-2 ***A,B***, Preoperative radiograph showing the maxillary right central incisor with healthy periapical tissue and a midroot sinus tract that traces to the mesial surface of the root. ***C,D***, Coronal view CBCT images reveal a lateral lesion and an associated lateral canal. ***E–H***, Based on CBCT measurements, the lateral canal was negotiated with hand files, then enlarged up to size 30/04, using rotary files, and filled using warm vertical condensation of gutta-percha. (Courtesy of Dr. Adham A. Azim, Buffalo, NY.)

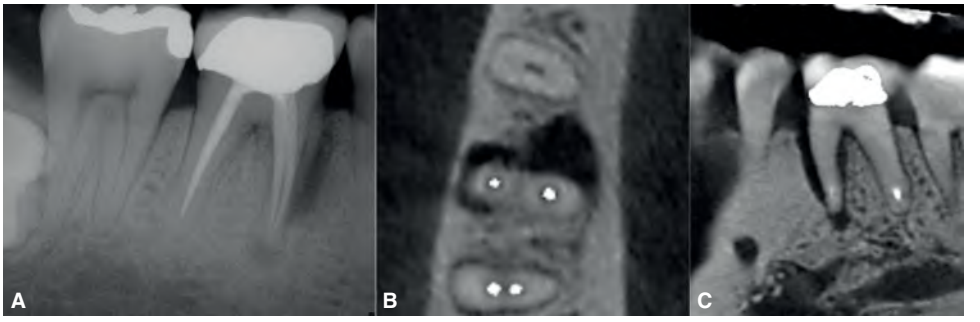

FIGURE 19-3 ***A***, Periapical radiograph of the mandibular right first molar showing prior endodontic treatment and a lateral lesion extending the full length of the mesial aspect of the mesial root, contiguous with the apical lesion on the mesial. ***B***, CBCT axial view shows the lesion extending from the buccal surface to the lingual surface of the mesial root. ***C***, CBCT sagittal view shows a bone loss pattern typical of vertical root fracture, extending the full length of both the mesial and furcal aspects of the mesial root. Extraction was advised. (Courtesy of Dr. Adham A. Azim, Buffalo, NY.)

information is provided to the patient in a language and terms that the patient understands. If necessary, an interpreter should be used or written information in the patient's native language is provided. While a statutory definition of "informed consent" may or may not exist in a particular state, there is extensive case law that defines the process, and it is well understood within the medical and legal communities to mean that a patient must "receive sufficient information to make a meaningful decision" regarding his or her own health care.[10]

Informed consent involves the process of the practitioner's explanation of the treatment, its potential benefits and risks, and the patient's understanding of that explanation. The informed consent discussion should be documented in the patient's treatment record. Although it is not required to use a written informed consent form, the American Association of Endodontists provides a sample form that can be used (Figure 19-4). Patient treatment refusal should be documented in the patient's record. Dentists should be aware of

SAMPLE STATEMENT OF CONSENT FOR ENDODONTIC TREATMENT

1. I hereby authorize Dr. _____ and any other agents or employees of _____ and such assistants as may be selected by any of them to treat the condition(s) described below:

   _____

   _____

2. The procedure(s) necessary to treat the condition(s) have been explained to me, and I understand the nature of the procedure(s) to be: _____

   _____

3. The prognosis for this(these) procedure(s) was described as: _____

   _____

4. I have been informed of possible alternative methods of treatment including no treatment at all.

5. The doctor has explained to me that there are certain inherent and potential risks in any treatment plan or procedure. I understand that the following may be inherent or potential risks for the treatment I will receive:
   swelling; sensitivity; bleeding; pain; infection; numbness and/or tingling sensation in the lip, tongue, chin, gums, cheeks, and teeth, which is transient but on infrequent occasions may be permanent; reactions to injections; changes in occlusion (biting); jaw muscle cramps and spasm; temporomandibular joint difficulty; loosening of teeth, crowns or bridges; referred pain to ear, neck and head; delayed healing; sinus perforations; treatment failure; complications resulting from the use of dental instruments (broken instruments—perforation of tooth, root, sinus), medications, anesthetics and injections; discoloration of the face; reactions to medications causing drowsiness and lack of coordination; and antibiotics may inhibit the effectiveness of birth control pills.

6. It has been explained to me and I understand that a perfect result is not guaranteed or warranted and cannot be guaranteed or warranted.

7. I have been given the opportunity to question the doctor concerning the nature of treatment, the inherent risks of the treatment, and the alternatives to this treatment.

8. This consent form does not encompass the entire discussion I had with the doctor regarding the proposed treatment.

Patient's Signature _____ Date/Time _____

Doctor's Signature _____ Date/Time _____

Witness' Signature _____ Date/Time _____

FIGURE 19-4 Sample informed consent form by the American Association of Endodontists.

country or state laws and whether informed refusals must be set forth in writing.

To ensure adequate disclosure, so the patient can be fully informed, the dentist should draw upon clinical judgment and experience, as well as current literature and research. The standard is whether a reasonable patient would be able to understand and whether a reasonable patient would have consented to the treatment if adequately informed of the significant risks.

TREATMENT TEAM COMMUNICATION

Computer technology has transformed the traditional "post-card" single tooth endodontic treatment report showing two radiographs taken before and after endodontic therapy into a comprehensive real-time information exchange of ongoing treatment planning and execution details. A digital charting system, with coordinated file storage, can simplify the

organization of patient's records containing text, image, and even video files. The data can then be conveniently retrieved and organized into an e-mail, containing multiple attachments providing all collaborating clinicians with the details of a planned and treated case. A networked computer system, with a main server, basic computer literacy, and technical support, are mandatory for a modern dental practice.

The advantage of digital communication is clear, with the patient receiving efficient interdisciplinary treatment unobstructed by delays. Comprehensive treatment planning is less prone to confusion, misinterpretation, and potential catastrophic errors.

Ideal technological tools, for ideal treatment team communication, include the following:

1. Networked computer system
2. Digital charting software that may include an integrated or separate image organizer
3. Digital radiography
4. Flatbed scanner (reflective and film capability)
5. Dental operating microscope with still and video camera attachment
6. Redundant backup systems in addition to the local network computer system (cloud, web-based, secondary external hard drives rotated off-site)
7. Online meeting and conferencing software

A strategically designed office may create a seamless gathering of data from chair-side to the front office, that is then converted and stored into organized electronic files, enabling efficient communication to the collaborating clinicians, oftentimes even before the patient walks out of the office (Figure 19-5).

FIGURE 19-5 With a convenient location of the computer screen and keyboard, the creation and storage of digital records can occur contemporaneously and photographic images can be easily captured via a camera mounted on the microscope. (Courtesy of Dr. Terrell F. Pannkuk, Santa Barbara, CA.)

The numerous technological advantages of modern data collection and communication come with many significant risks as well. Computer hacking, viruses, and identity theft are universal concerns that have alarmingly become common place. The Congressional Research Service reported that in 2012, 12.6 million Americans were victims of identity theft, and the average fraud victim incurred $365 in costs as a result.[13]

The risks of a security breach are not only a direct expense to the practitioner, but can also create liability should the patient's private information be stolen. Health Insurance Portability and Accountability Act (HIPAA) was created in the United States in 1996. A specific privacy rule was enacted in 2003 that dramatically changed health care practice and the release of patient information. The disclosure of Protected Health Information (PHI) has been strictly enforced and regulated such that reasonable steps to safeguard privacy are expected at all times.

Patient information exchange via e-mail must be limited to the patient's approved treatment team, and it is prudent to include an e-mail tag such as the following to prevent the accidental dissemination of private information:

> Confidentiality Notice: The information contained in and transmitted with this communication is strictly confidential, is intended only for the use of the intended recipient, and is the property of [insert name of dentist]. If you are not the intended recipient, you are hereby notified that any use of the information contained in or transmitted with the communication or dissemination, distribution, or copying of this communication is strictly prohibited by law. If you have received this communication in error, please immediately return this communication to the sender and delete the original message and any copy of it in your possession.

In summary, a referred consultation with a specialist is a request for an advanced perspective of the diagnosis and an advanced treatment service. Treatment team communication should be an interdisciplinary educational interaction that provides enhanced patient-centered care. The majority of endodontic procedures are performed by nonspecialists. This has significantly increased the complexity of cases referred to endodontic specialists. Retreatment of iatrogenically compromised teeth, as well as treatment of patients having special management problems, has often become the typical case referred to an endodontist. The endodontic specialty, and the dental profession as a whole, will greatly benefit by the clinical practitioner having an interdisciplinary focus, and seeking to learn and share from each patient interaction.

PREMEDICATION WITH ANTIBIOTICS

In the past, patients presenting for endodontic treatment were often prescribed prophylactic antibiotics to prevent possible pain and swelling (flare-up). In a survey of the American Association of Endodontists in 2002, 85% of participants reported that they prescribed a pretreatment

loading dose of antibiotics.[14] However, subsequent studies have shown that prophylactic antibiotic prescription has no effect on endodontic flare-up.[15,16]

Patients with certain medical conditions may require antibiotic premedication prior to endodontic treatment. For example, patients with risk of developing infective endocarditis (IE) should receive antibiotic premedication to prevent blood borne microorganisms from flourishing in the blood stream and lodging on shunts or prostheses.[17,18] In 2007, the American Heart Association (AHA) established significant updates in the guidelines for prophylactic antibiotics regimens to their prior 1997 guidelines.[19] The reason for the revision was that endocarditis is much more likely to result from frequent exposure to random bacteremias associated with daily activities than from bacteremia caused by a dental procedure.[20] In addition, the revised AHA guidelines were based on evidence that the risks of antibiotic-associated adverse effects exceeded the benefits.[21,22] The AHA now recommends that prophylactic antibiotics be limited to only those patients with high risk of developing IE, such as patients with the following conditions[19]:

1. Prosthetic cardiac valve or prosthetic material used for cardiac valve repair
2. A history of IE
3. A cardiac transplant that develops cardiac valvulopathy
4. Unrepaired cyanotic congenital heart disease, including palliative shunts and conduits
5. A completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first 6 months after the procedure
6. Any repaired congenital heart defect with residual defect at the site or adjacent to the site of a prosthetic patch or a prosthetic device (that inhibits endothelialization)

Except for the conditions listed previously, prophylactic antibiotics are no longer recommended for any other form of congenital heart disease.[19] For patients with the select conditions, prophylaxis is reasonable because endothelialization of a prosthetic material occurs within 6 months after the procedure.

The AHA has developed a standard antibiotic regimen for patients at high risk of developing IE, with alternative regimens for patients with allergies or when oral administration is not possible. The standard antibiotic regimen for all dental procedures is currently amoxicillin since it is better absorbed by the gastrointestinal tract and maintains a higher serum level than penicillin. The recommended regimens and dosages are listed in Table 19-1.[19]

For patients at high risk of developing IE, it is recommended that the patient take prophylactic antibiotics 1 h prior to dental treatment. This allows the antibiotics to reach adequate levels in the blood stream. If the patient forgets to take the prophylactic dose, the recommended dosage may be administered up to 2 h after the procedure. In cases where a patient is on an antibiotic regimen prior to treatment, the dentist may prescribe a different class of antibiotics than the class the patient is currently using. (For example, if the patient is currently on a regimen of clindamycin, the dentist should select amoxicillin for prophylaxis.)

In 2003, an advisory statement was drafted by the American Dental Association (ADA) and the American Academy of Orthopedic Surgeons (AAOS) stating that there was insufficient evidence supporting prophylactic antibiotic prescription for patients with total joint replacement.[23] Due to the lack of evidence, a regimen similar to patients with high risk of IE was recommended for patients with total joint replacement for the first 2 years after surgery. In 2014, the ADA Council on Scientific Affairs assembled an expert panel to update and clarify the clinical recommendations for premedications to patients with total joint replacement receiving dental treatment. No association between dental procedures and prosthetic joint infections was found in the updated systemic review and the panel concluded that there is no need to prescribe prophylactic antibiotics before dental procedures for patients with prosthetic joint replacements.[24] Since it is impossible to make recommendations for all patients, the ADA encourages dental professional to take this recommendation into account, consult with the patient's orthopedic surgeon when needed, and consider the patient's needs and preferences

TABLE 19-1 Recommended Antibiotic Regimen and Dosage for Patients Requiring Premedication before Dental Treatment

	Antibiotic regimen	Dosage 30–60 min prior to treatment		Administration
		Adults	Children	
Standard dosage	Amoxicillin,	2 g	50 mg/kg	Oral
	Cefazolin	1 g	50 mg/kg	
Allergy to penicillin	Clinadmicin	600 mg	20 mg/kg	Oral
	Azithromycin/clarithromycin	500 mg	15 mg/kg	
	Cephalexin	2 g	50 mg/kg	
Inability to administer orally	Cefazolin or ceftriaxone	1 g	50 mg/kg	IM or IV
	Ampicillin	2 g	50 mg/kg	
Allergy + inability to administer orally	Clinadmicin	600 mg	20 mg/kg	IM or IV
	Cefazolin/ceftriaxone	1 g	50 mg/kg	

Abbreviation: IM = intramuscular; IV = intravenous; kg = kilogram; mg = milligram.

when planning treatment. Clinicians should also exercise their clinical judgment in determining whether premedication for a patient is appropriate.

It is important to note that an antibiotic regimen should be prescribed in conjunction with proper endodontic treatment when there are systemic signs and symptoms of infection or a progressive/persistent spread of infection. For more details, see Chapter 30.

ANTI-ANXIETY REGIMEN

Patients presenting for endodontic treatment are often anxious about the procedure. There is often an unrealistic assumption that the procedure will be painful. Such misinformation can result in making it difficult for the patient to be relaxed during treatment. Although a majority of patients are able to control their fear, some fail to do so. Clinicians should attempt to fully explain the dental procedures prior to treatment and discuss possible minor discomforts during or after the treatment. Explanation of the treatment steps before initiating the procedure has been shown to reduce the patient's anxiety level.[1] In cases where patient's fear cannot be controlled, antianxiety protocol ranging from nitrous oxide to conscious sedation can be administered. For more details, see Chapter 18. The selection of the appropriate antianxiety medication should be based on each individual patient and his or her ability to control his or her behavior and anxiety during the treatment appointment.

PAIN MEDICATION

It can be challenging to completely anesthetize patients presenting for endodontic treatment with inflamed pulp or periapical tissue. The low pH environment resulting from the inflamed pulp or periapical tissue may affect the efficiency of local anesthetics and result in anesthetic failure.[25,26] Prescribing low dose of oral ketamine (10 mg) before endodontic treatment has been shown to enhance the effect of inferior alveolar nerve block in treatment of mandibular molars with irreversible pulpitis.[27] Other studies have shown that preoperative administration of ibuprofen or other nonsteroidal anti-inflammatory drugs (NSAID) 1 h prior to the local anesthesia injection is an effective method for achieving a profound anesthesia during endodontic treatment in teeth with inflamed pulp tissue.[28]

Postoperative pain may be anticipated following endodontic therapy.[29,30] Irritation of the periapical area may be expected due to extrusion of debris, irrigation, and root filling material during treatment. Studies have shown that prophylactic administration of pain medication may reduce postoperative pain.[31–33] Although postoperative pain may be mild or even absent following endodontic therapy,[34] clinicians often prescribe prophylactic pain medication to avoid possible moderate or severe pain after endodontic treatment.

THE DENTAL OPERATING MICROSCOPE

The Romans, in the first century, noted that objects viewed through glass appeared larger. Thus began the historical timeline of discoveries and inventions that significantly enhanced human vision. In 1609 Galileo Galilei mounted a convex and concave lens and created the compound microscope.[35] A new world of health science was revealed by Anton van Leeuwenhoek "largely due not only to his method of grinding, but also to the skill with which he mounted his lense."[36] In his letters to the Royal Society, his controversial claim to have documented single-celled organisms in 1674 which he called *animalcules* put him at odds for a period with the intelligentsia of his day. In 1922, Nylen performed a surgical procedure on the eye, that marked the beginning of modern microsurgery.[37,38] A few decades later the microscope became the standard protocol for additional medical specialties including neurology as well as ophthalmology,[39,40] coinciding with the Carl Zeiss Company (Germany) introducing the first commercial binocular microscope in 1953.[41]

A physician and dentist introduced the first dental operating microscope in 1978.[42,43] This team of Apotheker and Jako worked with Chayes-Virginia to develop the "dentiscope" and offered the first clinical hands-on microscope courses at Harvard Dental School in 1982.[41,44] Interest in the first generation dental microscope was minimal and Chayes-Virginia discontinued production and sales by 1986.

The Commission on Dental Accreditation (CODA) of the ADA received a formal recommendation by the American Association of Endodontists (AAE) in 1995 to include microscope training in the Accreditation Standards for Advanced Specialty Education Programs in Endodontics that was agreed upon in 1996, and microscope training became mandatory in these programs in 1997. In spite of the mandatory microscope training for the endodontic specialty, a significant number of endodontic specialists in practice and great number of general dentists performing endodontic procedures do not use this important tool. Figure 19-5 demonstrates the use of a dental operating microscope during four-handed dentistry.

Benefits of the microscope used for endodontic procedures include:

1. Improved treatment of calcified canals[45,46]
2. Refinement of endodontic surgical procedures such as apicoectomy with root-end filling where the micropreparation includes microgrooves and allows a smaller discrete access plan[47]
3. Improved management of complex root anatomy during nonsurgical endodontic treatment[48–52]
4. Improvement of postsurgical healing due to less traumatic flap management and approximation of tissues during microsuturing to favor healing by primary intention.[39,53,54]
5. Elimination of obstructions from root canals[55–58]
6. Diagnosis of root fractures[59]
7. Internal repair of root perforations[60]

Endodontic treatment performed prior to the introduction of the microscope was limited to tactile awareness without a clear view of the operating field. Dental instruments were larger with a cumbersome design. These same larger instruments used with the modern dental operating microscope are ill-suited for the finer perspective offered with 10×–20× magnification and enhanced lighting allowing direct visualization and preparation of anatomical features existing deeper in the root. Microsurgical ultrasonic instruments, microburs, and microsurgical hand instruments have been introduced to accommodate the new "micro perspective." A reduction in the frequency of surgical endodontic procedures has occurred as use of the microscope allows meticulous dismantling of posts and materials cemented and placed deep in the root. In one study, clinicians under and over the age of 40 were compared for visual acuity.[61] Visual acuity is measured as the angle between the eye and two distinct points or the identifying dimension of a recognized structure, which in the study was the E-optotype used in routine eye examinations. Dentists over 40 years of age were dependent on the microscope to inspect any location inside the root canal. It was found that dentists over the age of 40 could rarely detect the 0.06 mm width of a canal orifice without the orthograde illumination and magnification provided by a dental operating microscope.

The advent of the dental operating microscope has been described as a revolutionary advancement in diagnosis and execution of endodontic therapy.[62] The AAE has stated that microsurgery offers distinct advantages over traditional endodontic surgery.[63]

The following are common microscope options:

Microscope mountings:

1. Floor stand
2. Lateral wall mount
3. Ceiling mount

Lighting options:

1. Halide
2. Xenon
3. LED

Documentation accessories:

1. Still camera
   a. Video capture
   b. Digital "Point and shoot"
   c. SLR (Figure 19-6)
2. Video

The position of the patient and microscope can be adjusted to facilitate treatment in different areas of the mouth and the following guidelines determine position:

1. Vertical posture of the dentist and assistant in their respective chairs set to match positioning parameters.
2. The clinician's arms should hang relaxed and straight.

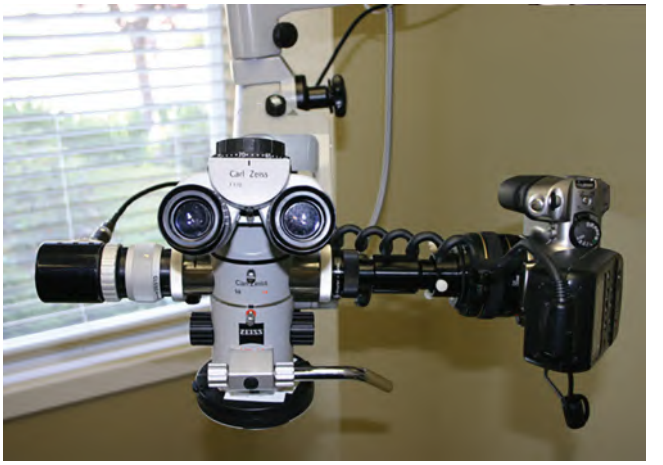

FIGURE 19-6 A beam splitter on the microscope allows the addition of adapters to accommodate video and SLR photography. (Courtesy of Dr. Terrell F. Pannkuk, Santa Barbara, CA.)

3. The head of the patient requires a sufficient angle and arching of the neck to accommodate visibility and access to posterior teeth when working in the 11–12-o'clock position. Occasional operator rotation to the 9-o'clock position is required for oral examination and use of hand files without contortion of the shoulders and arms.
4. Lower to middle magnification steps are used during most treatment procedures and the highest step magnification setting is used primarily for photography.
5. Indirect vision with a mirror is required for maxillary teeth and is often more comfortable than treating mandibular teeth in patients having limited jaw opening.

In summary, the complexities of the root canal system are illuminated, magnified, and better treated by a dentist utilizing the advantage of a dental operating microscope. The AAE published information highlighting the specific advantages of practicing endodontics with the microscope.[47] The advent of numerous microscope choices and features adapted to dental practice have shown microscopic dental technique to prolong a dentist's practice career and provide higher quality treatment.

LOUPES

Although the dental operating microscope is the preferred method for enhanced visualization during endodontic procedures, many practitioners report effective use of loupes. Some practitioners use the microscope for some aspects of a procedure and loupes for other aspects depending upon the desired amount of magnification. When utilizing loupes, high-intensity illumination is critical in order to elaborate the field and to reduce shadowing.

Innovations are being made for loupes as well as attached headlamps. There are adjustable magnification levels ranging from 3× to 5× (Orascoptic, Middleton, WI, U.S.A.). Higher levels of magnification with expanded field are available, including 4.5×, 6×, and 8× (Designs for Vision,

Ronkonkoma, NY, U.S.A.) and 5.5×, 6.5×, and 8× (SurgiTel, Ann Arbor, MI, U.S.A.). The higher the magnification, the smaller the depth and width of the field of vision. However, because endodontic procedures are generally performed within a confined space, the higher powers are recommended in order to visualize fine detail and deep canal morphology. Headlamps may be attached to the frames or on a separate headband and may consist of xenon or LED lights.

The visual acuity of loupes decreases as the working distance increases. Therefore, tall practitioners may need higher magnification. The loupes should feel comfortable, the neck should not be strained, and the visual field angulation should be adjusted to the operator's specific circumstance and preference. Many manufacturers also provide prescription within the lens portion surrounding in-the-lens telescopes.

For educational purposes and communication among collaborating clinicians, sophisticated high-magnification loupes (up to 8× or greater by special order) with power-matched video camera and coaxial high-intensity illumination offer not only excellent intraoral procedural capture, but also the unique ability for the clinician to capture at will, by a simple turn of the head, steps of a technique distant from the oral cavity such as custom fabrication or modification of clinical implements or instruments and procedures performed by the endodontic assistant. There are a number of loupe-mounted video systems available, including NanoCam (Designs for Vision, Ronkonkoma, NY, U.S.A.), HD LoupeCam (VizVOCUS, Scottsdale, AZ, U.S.A.), Surgicam Pro (SurgiTel, Ann Arbor, MI, U.S.A.) and through-the-lens Alos camera (High Q, Lexington, KY, U.S.A.).

ENDODONTIC SETUP

All instruments necessary for endodontic treatment should be conveniently available in the operatory to both the assistant and the dentist, and they should be neatly arranged for procedural efficiency. Various specialized instrument holders and setups are available (Figure 19-7), and each office should develop the setup that is most efficient and convenient. Typically, the endodontic tray setup will include the following:

- Injection syringe and anesthetic
- Mirror, periodontal probe and endodontic explorer
- Dental dam, dental dam frame, clamp forceps and dental dam punch
- Clamp(s)
- Floss for passing the dam through proximal contacts and for ligating the clamp
- High-speed and slow-speed handpieces and burs
- Locking cotton plier for general purposes and for bur and rotary instrument insertion (Figure 19-8)
- Canal-irrigation syringes
- Endodontic spoon excavator
- Mixing spatula and mixing pad or glass slab
- Obturation instruments

FIGURE 19-7 Endodontic instrument kit. (Courtesy of HuFriedy, Chicago, IL.)

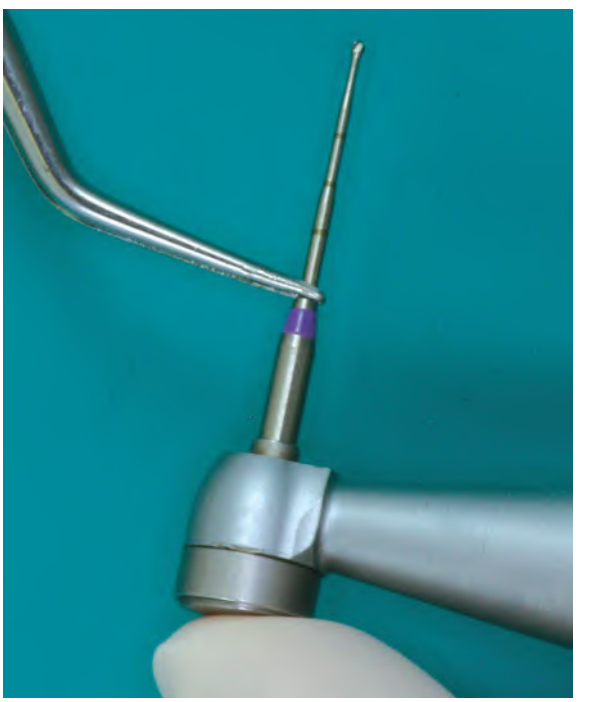

FIGURE 19-8 Various endodontic rotary instruments, including the Munce Discovery Burs as demonstrated here, Gates Glidden burs and rotary files, can be conveniently "dropped" into a latch-type slow-speed handpiece with a locking cotton plier while spinning the rotor via the foot control.

- Canal cleaning and shaping instruments that can be arranged on a disposable sponge (or another disposable setting) in a carrier such as the Endoring (Jordco, Beaverton, OR, U.S.A.) (Figure 19-9)

FIGURE 19-9 The Endoring allows for efficient setup and access to hand and rotary files. (Courtesy of Jordco, Beaverton, OR.)

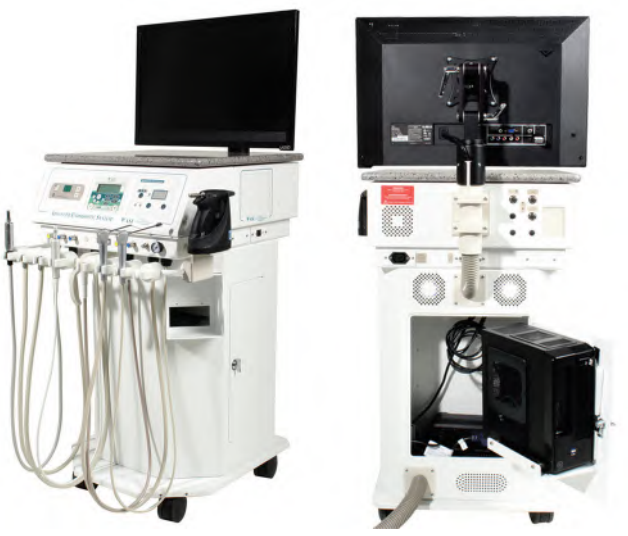

FIGURE 19-10 Views of the front and back of the ASI Advanced Endodontic System® Designer Series Ergo iTech. (Courtesy of ASI, Englewood, CO.)

Conveniently located and readily accessible should be various broken instrument retrieval devices, crown removal devices, mineral trioxide aggregate (MTA) (or alternative), various irrigants, sealer, gutta-percha, Dycal, Cavit, posts, and build-up materials as well as supporting instrumentaria.

A delivery cart system, specifically configured for endodontics, can contribute to proper ergonomics and efficiency in the operatory. The Ergo iTech™ (Figure 19-10) provides a unique solution for diverse endodontic instrumentation. Dentists can designate their specific choice of instruments and instrument locations on the cart, each with operation from a single foot control. Accessories can be integrated such as instrument trays, a monitor mount, and an internal CPU shelf.

TOOTH ISOLATION

Dental dam isolation has been used for over 150 years, developed by Dr. Stanford C. Barnum in 1864.[64] Since then it has evolved to include multiple shapes and types, all with a common aim, to isolate the tooth structure during dental procedures. Dental dam isolation is the optimal method of endodontic isolation. Numerous advantages include[65–69]:

1. Protection of the patient from swallowing or aspiration of endodontic instruments, irrigants or medicaments.
2. Creation of an aseptic operating field and elimination of salivary contamination of the root canal system and instruments.
3. Reduction in the microbial content of air turbine aerosols produced during dental procedures and thus a reduction of cross-contamination risk in the dental practice.
4. Creation of a more efficient and favorable working environment by minimizing rinsing and patient conversations.
5. Improved visibility by providing a dry field and minimizing mirror fogging.
6. Retraction and protection of the tongue, lip and other soft tissues.

Recent studies have shown a significant improvement in the survival and outcome of endodontically treated teeth when dental dam was used during root canal therapy and post placement.[70,71] Although it may be inconvenient for a clinician to use with certain patients (mouth breathers, patients with exaggerated gag reflex, severely anxious patients), inconvenience is not an acceptable excuse for avoiding dental dam placement. Root canal treatment without dental dam isolation may place doctors under litigation risks if the patient swallows or aspirates an endodontic instrument (Figure 19-11).[65,72] Previous publications state that "routine placement of dental dam is the standard of care,"[73] and it is a mandatory procedure for limiting cross-contamination, infection of the root canal system, and potential systemic infection.[65–69,74] In some clinical situations (calcified pulp chambers, calcified canals, crowned teeth), locating the canal(s) is challenging and may require orientation of the external root surface using a periodontal probe. In such cases, and depending on the tooth location, placement of dental dam can be delayed until canals are located to avoid excessive damage to the remaining tooth structure or possible root perforation and its consequent complications. Once the canals are located, the dental dam should be placed immediately. No endodontic file should be inserted into a canal without dental dam isolation.

Dental Dam Components

The dental dam system consists of three main components: a dental dam sheet, frame, and a clamp. The dental dam sheets are autoclavable and come in different thicknesses (thin, medium, heavy, extra heavy, and special heavy), colors (ranging from light yellow to gray), sizes (5 x 5 and 6 x 6 in) and

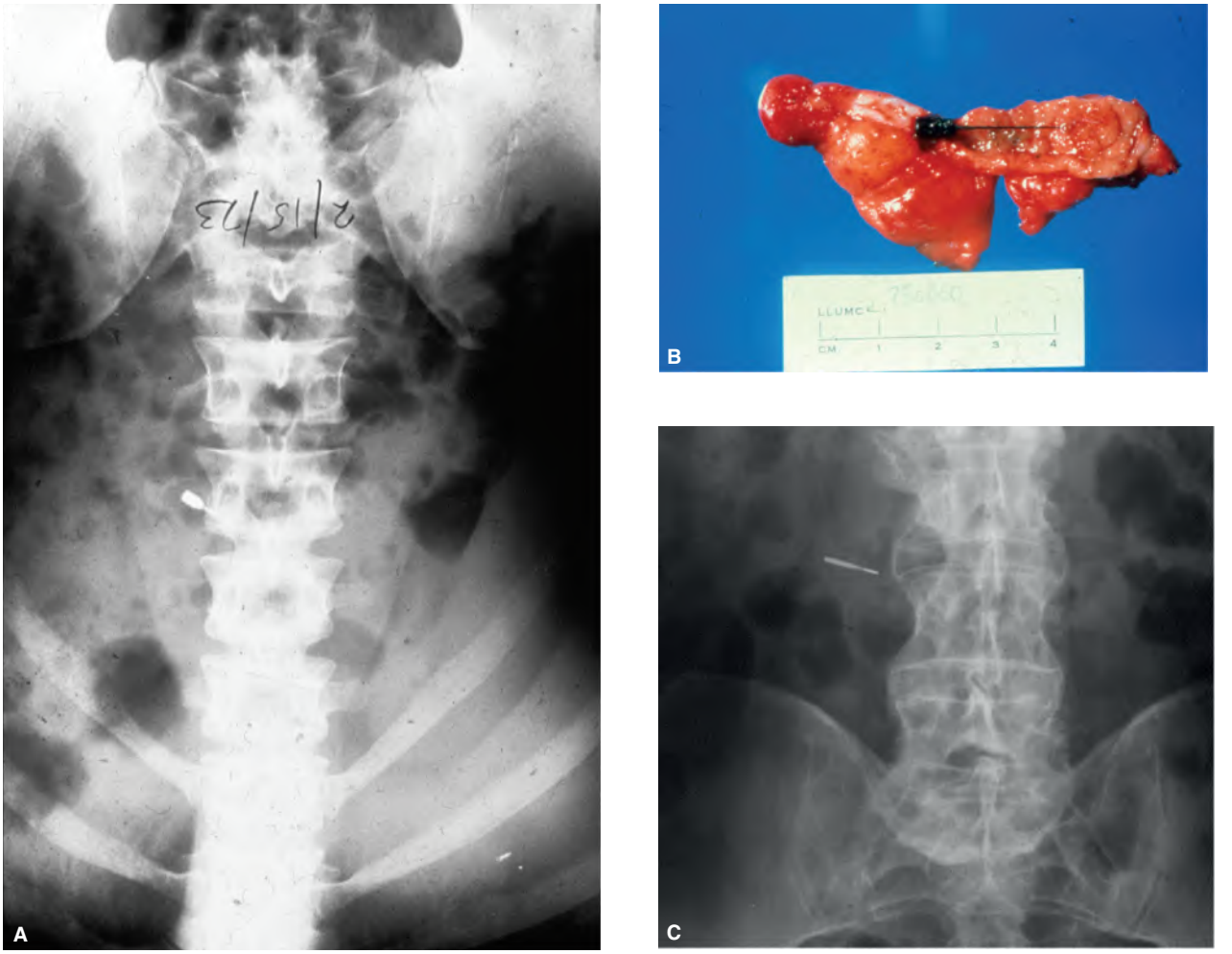

FIGURE 19-11 ***A***, Swallowed endodontic file ended up in the appendix resulting in acute appendicitis. ***B***, Specimen shows file in the appendix removed by appendectomy. Use of dental dam would have prevented this complication. ***C***, Dental burs can sometimes disengage from headpieces and be swallowed, as shown here. This is also preventable with dental dam. (***A*** and ***B*** reproduced with permission from Thomsen LC, Appleton SS, Engstrom HI. Appendicitis induced by an endodontic file. *Gen Dent* 1989:37–50.)

materials (latex and nonlatex). The commonly used dental dam sheet in endodontics is the medium thickness due to its higher resistance to tear compared to the thin sheets and its easier manipulation compared to the heavier variants. The selection of the dental dam color depends primarily on the clinician's preference. Darker color sheets will provide better contrast, improved visibility, and higher quality images and videos during photographing and recording. Lighter colors, on the other hand, create a brighter field, which may be useful if the illumination is compromised. The sheet also comes in a nonlatex form for patients with latex allergy. The nonlatex dental dam, however, comes in only one size- 6" × 6", and one thickness—medium.

The function of the dental dam frame (Figure 19-12) is to retract the sheet during procedures and to allow visibility and access to the tooth. Frames are available in either metal or plastic. Although metal frames are more durable, they require removal before taking radiographs. This may affect the stability of the dam and can compromise isolation. Plastic frames, on the other hand, offer certain advantages during endodontic procedures. They are radiolucent and thus do not require removal prior to taking radiographs. Plastic frames are available in different shapes and forms including fixed and hinged. They are also available as disposable frames. The plastic hinged frame allows the dam to be folded to one side for taking radiographs necessary during endodontic treatment.

The function of the clamp is to retain the dental dam and frame on to the target tooth. In case of multiple tooth isolation, the clamp is always placed on the most posterior tooth to be isolated. The clamp is formed of two beaks with a connecting bow (Figure 19-13). The beaks can be apically inclined or serrated (tiger clamps) to allow better engagement of teeth with compromised structure. The beaks of the clamp are positioned

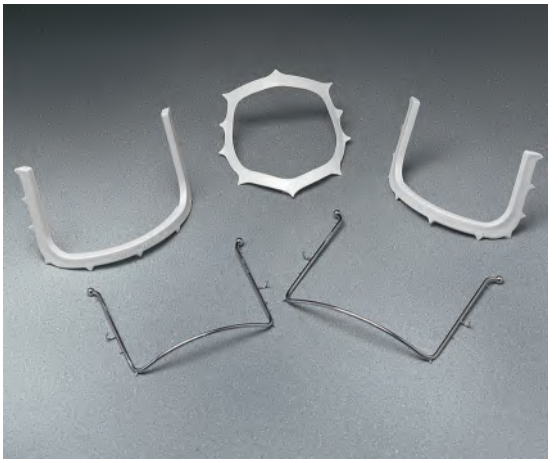

FIGURE 19-12 Images of different shapes of metal and plastic dental dam frames.

FIGURE 19-13 Image of a Hu-Friedy tiger clamp. 1. Central wing; 2. anterior wing; 3. bow; 4. beaks; 5.tines; 6. forceps hole; 7. anterior notch; 8. posterior notch.

on the buccal/labial and lingual/palatal aspects of the tooth below the height of contour using the clamp forceps. The clamp retains the sheet under the anterior and central wings or under the platform of the beaks if a wingless clamp is used.

The clamp should be stabilized prior to initiating treatment. Unstable clamps may jeopardize isolation and cause damage to the tooth structure and surrounding soft tissue during treatment. The stability can be checked by rocking the clamp using both thumbs.

Clamps should routinely be ligated with a lengthy section of dental floss through both forceps holes with the floss draped outside the patient's mouth and secured to the patient bib or some other convenient item that cannot be swallowed or aspirated in the event the clamp becomes insecure or breaks. While there are a variety of ways to reduce the risk of aspiration or swallowing of an intact clamp, attaching the floss through both forceps holes (Figure 19-14) offers the added security of retrievability of both elements in the event of a fractured bow.

Clamps are available in variety of shapes and sizes attempting to match the tremendous variety of tooth forms, orientations, and degrees of debilitation. The general classifications are central and lateral incisor, canine, premolar, and molar clamps; and they are available both with and without wings (Table 19-2). Clamps with wings provide better retraction of soft tissue, less salivary leakage; and for placement of the clamp, dam, and frame as a single unit, winged clamps are required.

Dental Dam Holes

The required hole in the dental dam can be placed either by using the dental dam punch or scissors. The punch has a series of holes of varying sizes on a disk that can be rotated to select the appropriate size for the tooth to be isolated. A pair of iris scissors offers an alternative method to create a hole in the sheet. Folding the sheet into four quarters and cutting the corner will create a hole in the center of the dam. The size of the hole can be controlled by the size of the piece cut from the corner (Figure 19-15).

Dental Dam Placement Techniques

Stability of the dental dam system is necessary throughout the procedure. The dam must cover the entire mouth without encroaching on the patient's nose or eyes. Improper dental dam placement may result in leakage and contamination of the working field. Multiple techniques may be used to place the dental dam.

Single-Unit Method

A hole is punched in the dam, and the sheet is then stretched medium tight by attaching it at the four corners of the frame. The central wings of the clamp are inserted, one at a time, through the hole, and the entire system is carried as a unit to the patient's mouth using the forceps. The clamp is placed on the target tooth, and the dam is released from the wings of the clamp with a plastic instrument (Figure 19-16). This is a relatively safe method because the clamp is secured to the dental dam. It is convenient in the case of anterior teeth and premolars, and it is more challenging in the case of molars and teeth with structural compromise due to reduced visibility.

Clamp-First Insertion Method

The clamp is first placed on the tooth. The punched dental dam, mounted loosely onto the four corners of the frame, is then brought to the mouth, and the hole is stretched over the clamp. Floss is used to carry the margins of the dam through the proximal contacts to completely cuff the tooth. The dam is then stretched more fully onto the frame.[75] This is one of the least complicated placement methods because the target tooth is most easily visualized during clamp placement (Figure 19-17).

Multiple Isolation of Proximate Teeth Method

When root canal treatment is performed on multiple teeth that are proximate to each other, or when a tooth cannot retain a clamp due to insufficient structure (Figure 19-18), multiple holes can be placed in the dental dam to allow treatment of the target tooth/teeth. In the case of a single target tooth unable

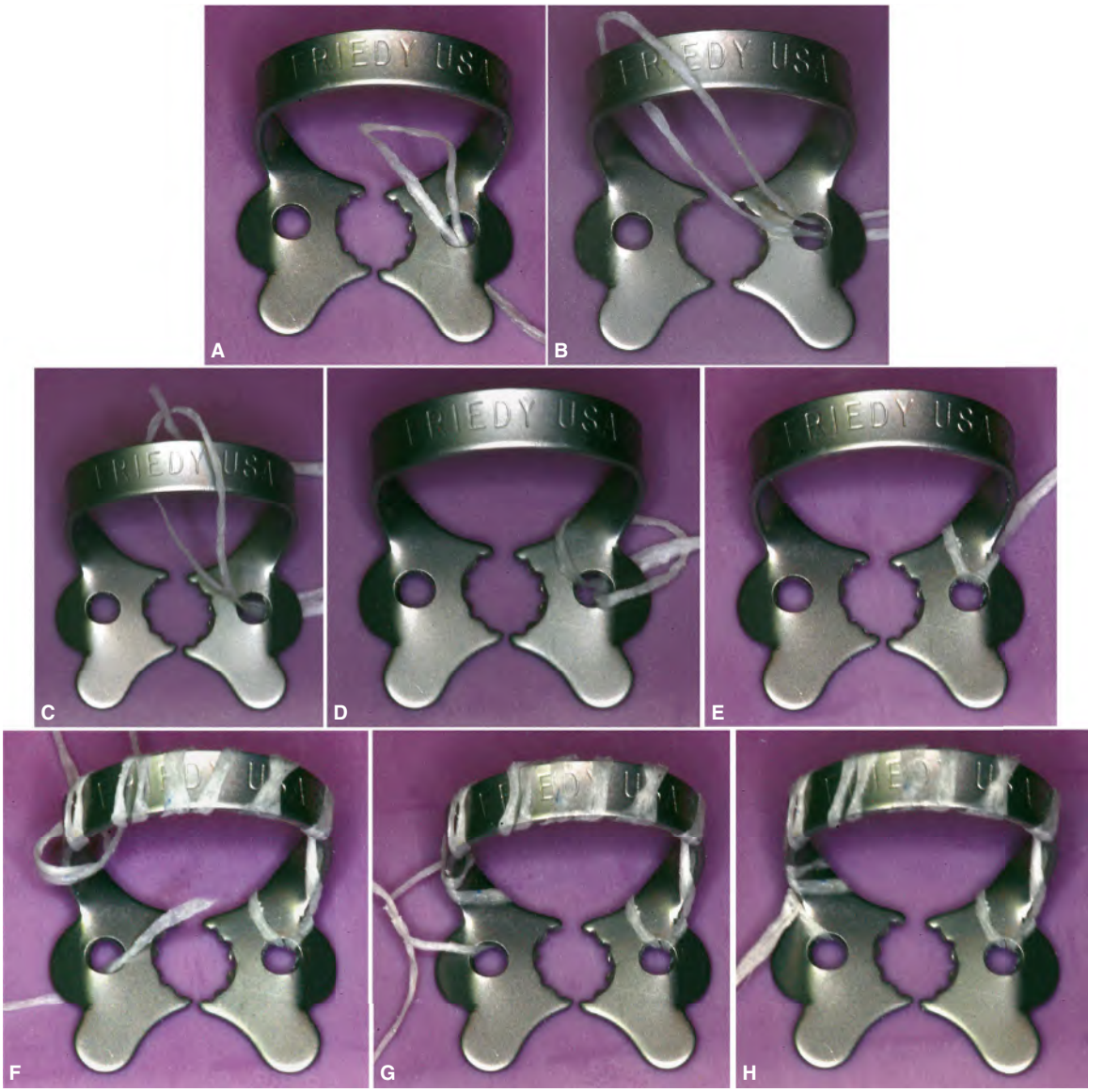

FIGURE 19-14 Clamp safety strap technique. If a dental dam clamp breaks at the bow, floss ligature on the bow or in only one of the forceps holes will be insufficient to prevent swallowing or aspiration of elements of the clamp. Floss ligation through both forceps holes will secure both elements of a broken clamp as well as an intact clamp should it launch from the tooth. ***A***, Insert the loop of a 20-inch section of floss through what will be the lingual forceps hole of the clamp; ***B***, loop it over the bow; ***C***, insert free ends of the floss through the loop from behind the bow; ***D,E***, draw the loop down tight against the elbow of the clamp; ***F***, take several wraps of the doubled floss around the bow and insert a single free end through what will be the buccal forceps hole; ***G,H***, tie a double knot with the other free end and tie the tail to the dental dam frame or the patient bib (i.e., tie to something the patient cannot aspirate or swallow in the event the clamp breaks or launches from the tooth). Done properly, the free ends of the floss will be on the buccal side of the clamp, and the floss in the forceps holes will be oriented such that it will not be impinged on by the beaks of the forceps which could weaken or break the floss. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

to retain a clamp, the clamp is placed on a tooth distal to the target tooth. In the case of multiple teeth to be treated, the clamp is placed on the most posterior target tooth if it can retain a clamp. If this tooth is excessively compromised, the clamp should be placed on a more posterior tooth capable of retaining a clamp. The most distal hole is then stretched over the clamp, and the remaining holes are drawn over each additional tooth to be exposed for the procedure. It can be helpful in certain cases to place a second clamp on the most anterior tooth to be exposed with the bow facing mesially. Ligation with dental floss may enhance isolation where necessary (Figure 19-19). In multiple tooth isolation of anterior teeth, clamps placed on teeth on opposite sides of the midline will be oriented normally, with bows facing distally (Figure 19-20).

Multiple Isolation of Distant Teeth Method

When root canal treatment is performed on multiple teeth that lie in different quadrants yet in the same arch (maxillary

or mandibular), a clamp may be placed on each tooth to be treated, and two holes can be punched on either side of the dam allowing simultaneous treatment of both teeth (Figure 19-21).

Split Dam Method

The split dam method has many practical applications. It is yet another useful isolation method when the target tooth cannot accommodate a clamp due to insufficient structure.

TABLE 19-2 Dental Dam Clamps Selection

	Clamps
Maxillary teeth	
Central incisor	6, 9, 209, 210, 212
Lateral incisor	6, 9, 209, 210, 00
Canine	6, 9, 210
Premolars	0, 2, 2A, W2A, 209
Molars	26, 26N, 27N
Mandibular teeth	
Incisor	6, 9, 210, 212
Canine	6, 9, 210
Premolars	0, 00, 2, W2A, 209
Molars	26N, 27N,, W3, W8A, 14, 14A

Two holes are punched in the dam separated by several millimeters depending on the clinical circumstance. The material between the holes is snipped with a pair of iris scissors creating the split dam (Figure 19-22). The clamp is placed on a more distal tooth and the sheet is then placed over the clamp and stretched to include the target tooth and at least the next anterior tooth (Figure 19-23).

The split dam method is also effective when treating teeth with calcified canals as it allows exposure of the external contours of the target tooth, providing orientation during deep exploration for the canal remnant. Additionally, the split dam can be a good choice when the target tooth has been restored with a porcelain crown as studies have shown that, even when properly stabilized, clamps can damage porcelain fused to metal crowns.[76,77]

When root canal treatment is performed on two distant teeth connected by a bridge, the split method can result in a large gap that may compromise isolation. In such cases, the margins of the split dam can be approximated from the lingual/palatal to the buccal/labial using suture material, piercing the sheet and passing under the bridge to be tied off on the facial. Any remaining exposed tissue can be blocked out using Oraseal or Liquiddam (Figure 19-24).

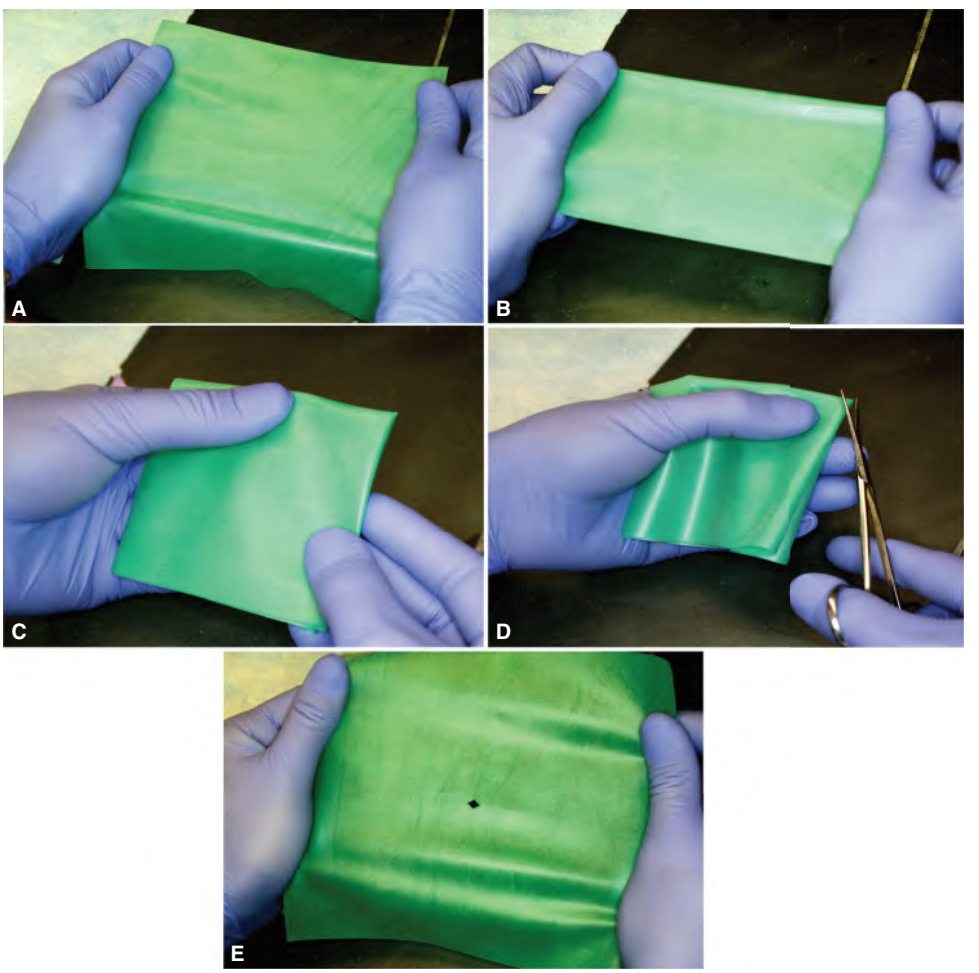

FIGURE 19-15 Different steps for creating a dental dam hole using scissors. ***A***, Dental dam sheet; ***B***, sheet folded in half; ***C***, sheet folded in half again; ***D***, cut hole at the folded corner; ***E***, hole created in the center of the dam.

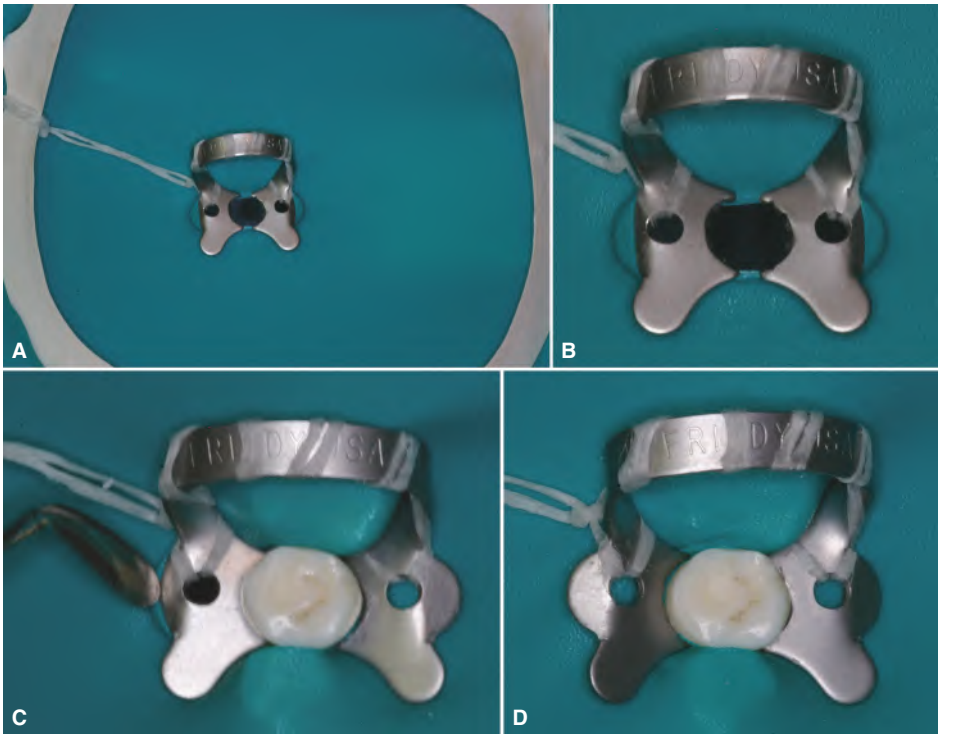

FIGURE 19-16 Single-unit dam placement technique. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

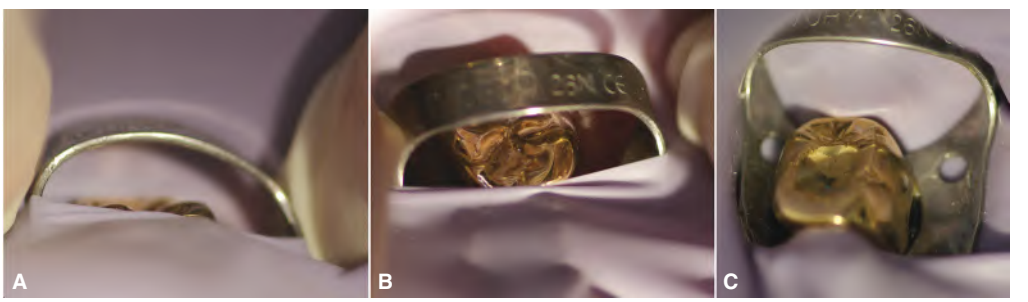

FIGURE 19-17 Clamp-first insertion method for dental dam placement. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

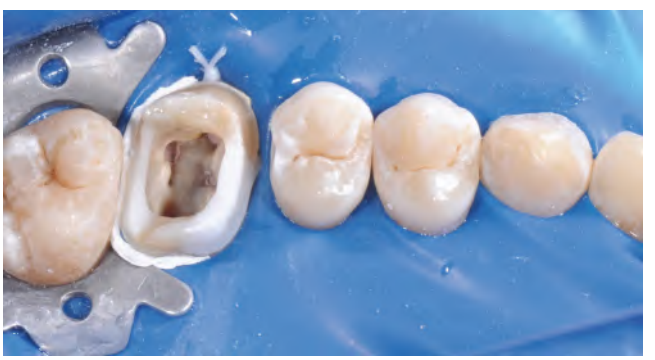

FIGURE 19-18 Multiple tooth isolation for the treatment of a maxillary first molar. (Courtesy of Dr. Maxim Belograd, Kremenchug, Ukraine.)

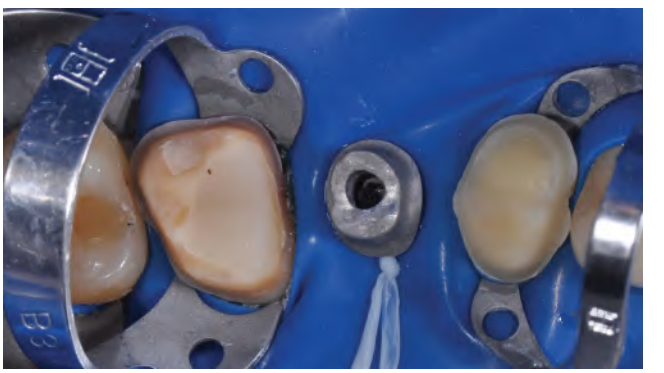

FIGURE 19-19 Multiple tooth isolation for treatment of a maxillary second premolar using clamps on the maxillary first and second molars and maxillary first premolar. (Courtesy of Dr. Maxim Belograd, Kremenchug, Ukraine.)

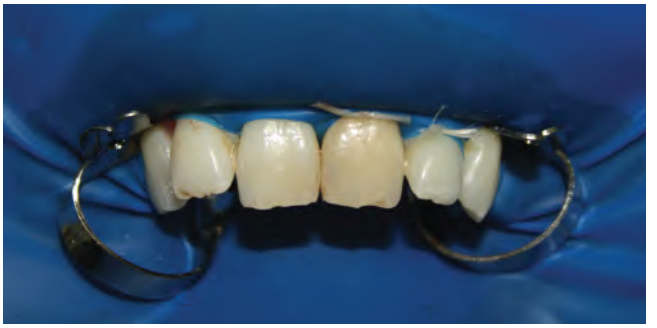

FIGURE 19-20 Multiple tooth isolation for treatment of maxillary central incisors using clamps on canines. (Courtesy of Dr. Adham A. Azim, Buffalo, NY.)

FIGURE 19-21 Isolation of mandibular canines using two clamps placed on target teeth. Two holes are then punched in the dental dam corresponding to the canines. The holes are then drawn over the clamps, one clamp at a time, and then stretched onto the dental dam frame. (Courtesy of Dr. Adham A. Azim, Buffalo, NY.)

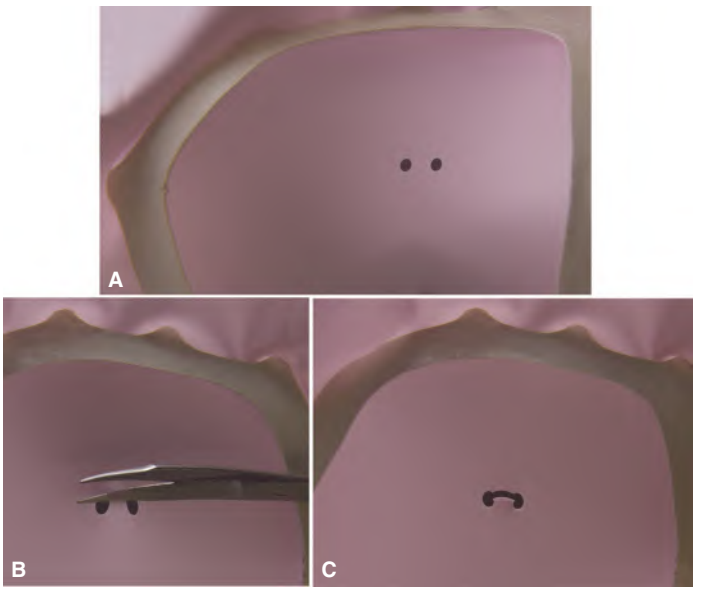

FIGURE 19-22 Preparation of a split dam. ***A***, Punch two holes in the dam a few millimeters apart as necessary to accommodate the teeth to be isolated; ***B***, snip the material between the two holes; ***C***, a split dam is created.

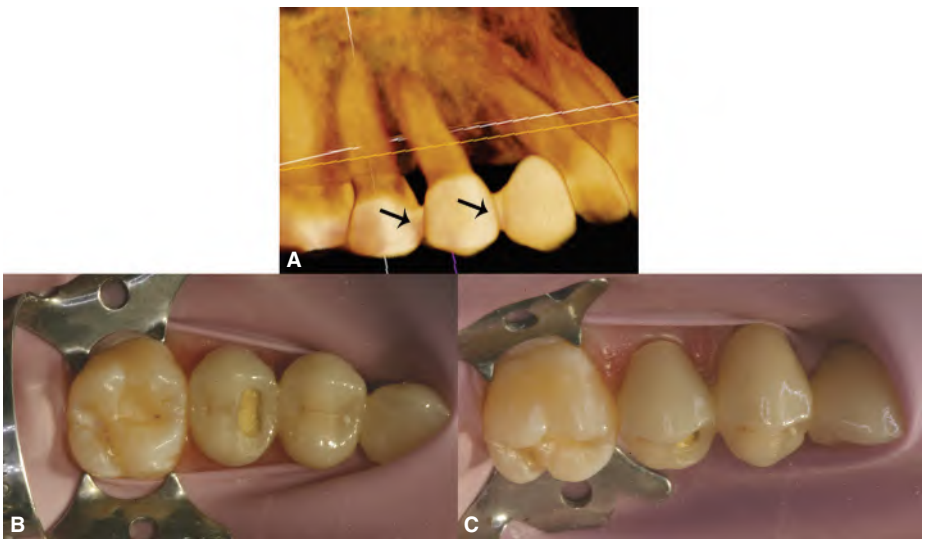

FIGURE 19-23 Case demonstrating use of the split dam technique to ***A***, isolate the maxillary 2nd premolar, part of a double abutted cantilevered bridge (arrows). ***B,C,*** the dam was stretched to include the cantilevered canine, sliding completely under the ridge-lap to cuff the mesial, buccal and palatal aspects of the first premolar. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

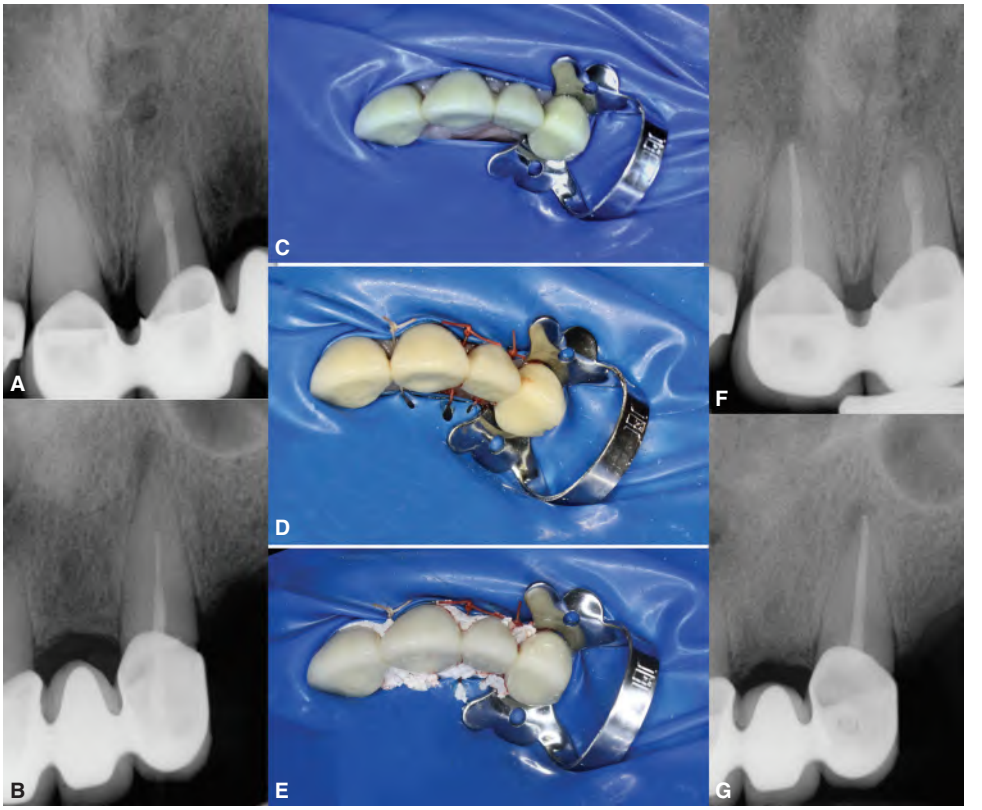

FIGURE 19-24 Split dam technique for multiple tooth isolation for treatment of the maxillary right central incisor (***A***), and the maxillary left canine (***B***). ***C***, Clamp is placed on the most distal tooth, the maxillary left canine and the single large hole in the dam is stretched to include the right central incisor. ***D***, The gap between the palatal and the labial is closed by approximating the margins of the dam using suture material. ***E***, Remaining minor spaces are then sealed with Oraseal (Ultradent). ***F,G***, Root canal treatment is completed on the maxillary right central incisor and the maxillary left canine. (Courtesy of Dr. Adham A. Azim, Buffalo, NY.)

OVERCOMING ISOLATION CHALLENGES

Endodontic isolation can be difficult when certain conditions exist, and chief among those conditions is dental caries. Despite worldwide decrease in the incidence of dental caries,[78] it continues to impact all age groups[79] and in many parts of the world, it is the primary reason cited for tooth extraction.[80]

Difficult isolation can be encountered when certain factors are present[81]:

1. Minimal supragingival coronal tooth structure
2. Extreme posterior positioning of the tooth
3. Severe root caries
4. Loose crown or bridge restorations
5. Edentulous areas posterior to the tooth to be treated
6. Tooth prepared for a crown with an "ice cream cone" form
7. Complicated patient management

Various methods can be used to assist in creating ideal endodontic isolation even in complex circumstances (Figure 19-25).

Clamp Modification

Dental dam clamps are manufactured in many different designs, shapes, and sizes attempting to accommodate many clinical circumstances and most tooth groups. Clinical circumstances, however, are infinite, and teeth develop in myriad shapes and sizes and emerge in every imaginable orientation. As a result, even a well-stocked inventory of clamps will not be able to meet every isolation challenge. Clamp modification can be an effective method of overcoming some of these challenges.[82–85] When planning clamp modification, it is important to avoid modifications that might adversely affect the structural integrity of the clamp. For example, avoid cuts to the bow of the clamp that could lead to clamp breakage under function. Likewise, when removing a wing, leave adequate metal around the outer rim of the forceps hole to prevent the hole from breaking open during clamp placement or removal. When altering the mesiodistal reach of clamps, equal amounts should be removed from both the mesial and distal aspects of the beak being modified so as not to skew the orientation of the clamp, unless skewing the clamp is the objective. In all cases, after modification, the cut surfaces must be polished with a Brownie Point, Brownie Wheel, or equivalent to remove metal burrs that can catch and tear the dental dam.

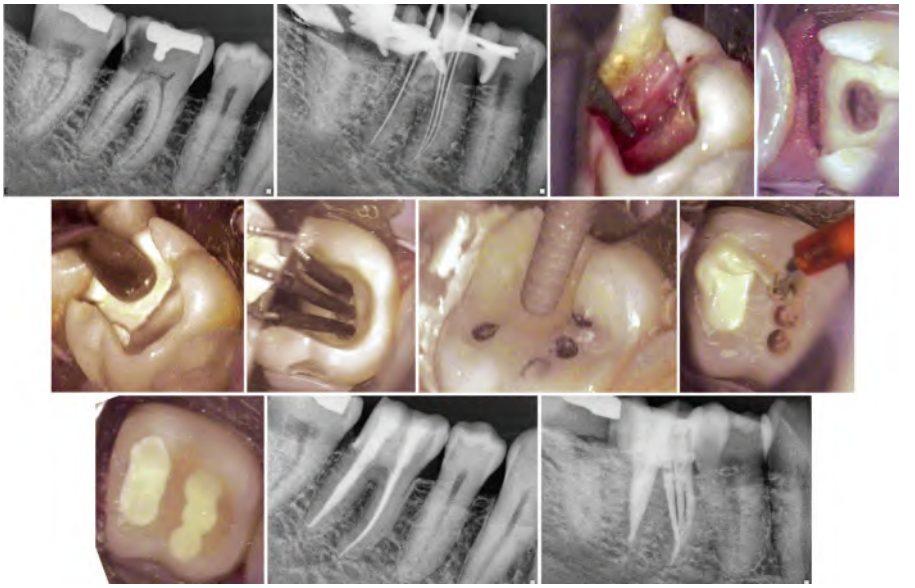

FIGURE 19-25 Challenging isolation resulting from severe coronal breakdown is managed by high-speed diamond tissue troughing, sculptable Cavit matrix barrier and bonded preendodontic build-up via Canal Projection. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

Reducing the Mesio-distal Reach of the Beaks

If the mesio-distal reach of the beaks of the clamp extends beyond the width of the neck of the tooth (Figure 19-26A,B), the dental dam may not invaginate properly to cuff the tooth and provide moisture-proof isolation. This reach can be effectively reduced by first scribing the amount of desired reduction onto the clamp with a fine-tipped indelible marker (Figure 19-26C), prior to removing equal amounts of metal from both the mesial and the distal aspects of the buccal beak, or the lingual beak or both with a high-speed bull-nosed diamond (Figure 19-26D). Any resultant burrs should be removed by polishing with a Brownie Wheel or equivalent (Figure 19-26E). Customizing a stock clamp by reduction of reach (Figure 19-26F) can provide precise cervical adaptation of clamp on both buccal and lingual aspects of the tooth (Figure 19-26G,H).

Removing Tines from the Beaks

If the beaks are tined, the mesio-distal reach can be reduced by removing equal numbers of tines from both the mesial and distal aspects of the buccal beak, or the lingual beak or both with a high-speed bull-nosed diamond (Figure 19-27A,B).

Removing a Wing

Removing a wing from a clamp can be helpful, particularly when working on either maxillary or mandibular second and third molars as the ramus can lie tight against the buccal aspect of these teeth. Also, when a tooth is extremely malposed or when there are exostoses or hyperplastic gingival tissues impinging on cervical aspects of the tooth, wing removal may be necessary to accommodate proper seating of the clamp. The wing can be sliced off with a high-speed fissure bur. Care must be taken not to overly thin the outer rim of the forceps hole to prevent it from breaking open during clamp placement or removal (Figure 19-27C–E).

Removing One Bow from a Butterfly Clamp and Modifying the Reach of the Beaks

A butterfly clamp can be converted into a standard single-bow clamp by removing one bow with a high-speed fissure bur (Figure 19-28A,B). Mandibular incisors present with the unique challenge of a very small mesio-distal width on both the buccal and lingual aspects at the neck of the tooth. Not only are there few clamps manufactured with small enough mesio-distal dimension to perfectly accommodate these teeth, but the pull of the dental dam against a standard nonbutterfly-type clamp with a tiny mesio-distal dimension tends to rotate the bow aspect of the clamp incisally. The symmetry of a butterfly-type clamp can help to offset this rotating effect, but most butterfly clamps lack the tiny mesio-distal dimension required for mandibular anterior teeth. This excessive reach can be effectively reduced by removing equal amounts of metal from both the mesial and the distal aspects of the buccal beak, or the lingual beak or both with a high-speed bull-nosed diamond. In combination with wing removal, this is an effective way to customize a clamp to accommodate small-necked teeth (Figure 19-28C,D)

Sharpening the Tines or the Smooth Beaks

Whether smooth or tined, the beaks of clamps can be effectively sharpened for improved retention by careful hollow-grinding with a high-speed football diamond (Figure 19-29).

Changing the Inclination of the Beaks

The inclination of the beaks can be modified by grasping the clamp at the neck of the bow with a large locking hemostat,

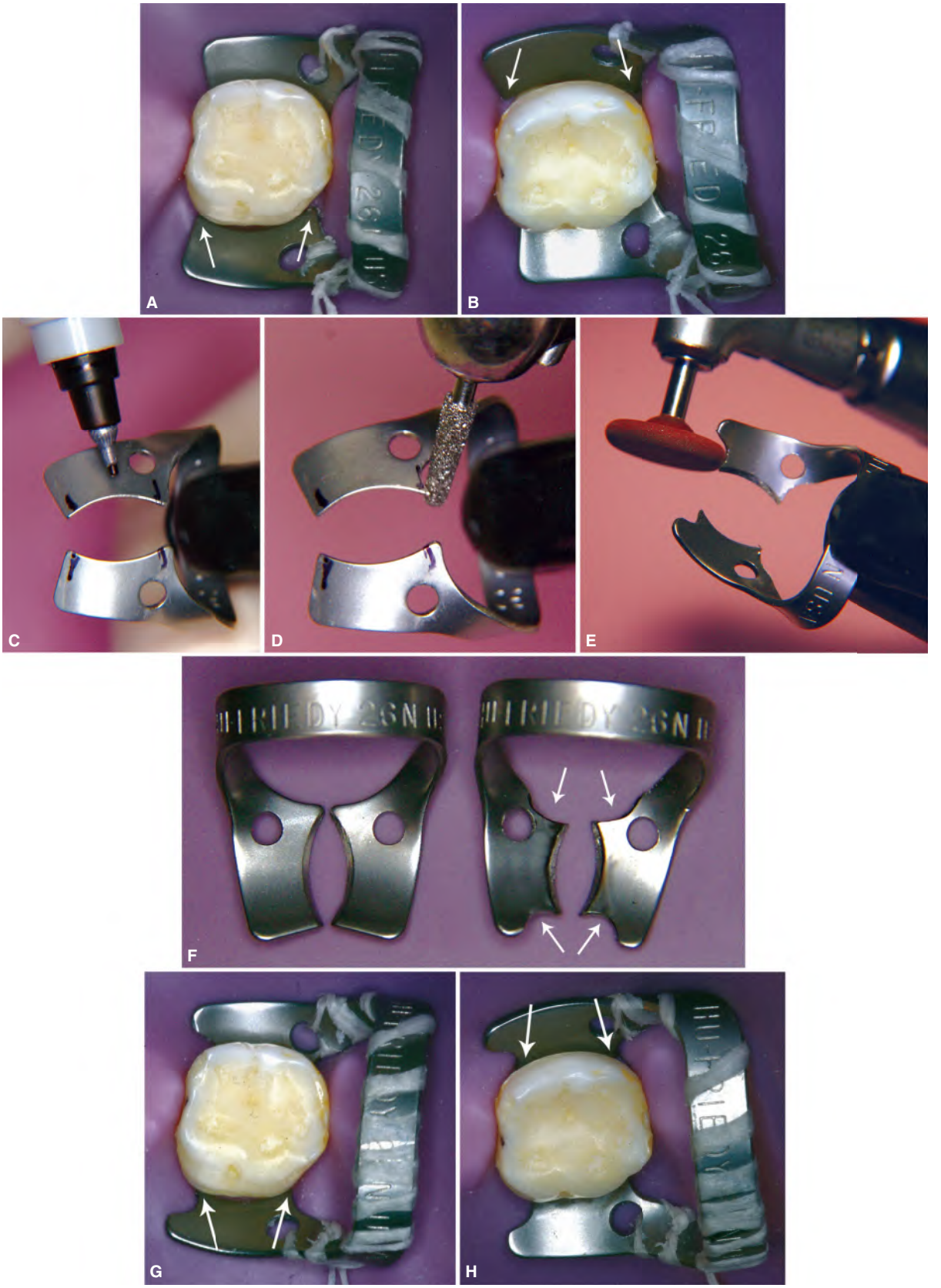

FIGURE 19-26 Reducing the mesio-distal reach of the beaks. See accompanying text for description of steps A through H. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

FIGURE 19-27 ***A***, Removal of tines and a wing from the clamp. ***B***, The tines being removed with a high-speed bull-nosed diamond. ***C***,***D***, Demonstrate the initiation of the cut for wing removal. ***E***, Modified clamp with tines removed from both buccal and lingual aspects and one wing removed. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

inserting an instrument with a beefy shaft through the forceps hole and bending the beak apically or coronally as required (Figure 19-30).

Flowable Tissue Block-out and Matrix Barrier Materials

Teeth requiring root canal treatment often present in broken-down condition, missing one or more cavity walls or reduced in preparation for a crown. The dental dam alone may not provide complete isolation of the operating field.

Commercially Available Block-Out Agents

There are a number of syringeable materials that have been specifically designed for the purpose of blocking leakage around the dental dam. These products include Kool-Dam (Pulpdent, Watertown, MA, U.S.A.), OraSeal Caulk and OpalDam (Ultradent, South Jordan, UT, U.S.A.) and (Figure 19-31).

Dycal

The low viscosity and flow of Dycal (Dentsply, York, PA, U.S.A.) is ideal for sealing exposed tissue and dental dam gaps (Figure 19-32). It can also be used when standard matrix band application is impractical. A "floating" sectional matrix secured with wedges and luted with Dycal allows placement of a core with ideal margins and contours in the most challenging tooth-defect situations. The setup for placement of Dycal is simple and can be available chair-side at any time during a root canal treatment procedure (Figure 19-33A).

Dycal/Cavit Matrix Barrier

Though multi-factorial, overcoming extreme endo-restorative isolation challenges resulting from significantly compromised coronal-radicular structure nearly always involves, as a first step, control of fluid contaminants migrating into the working field from adjacent deep sulcular tissues. Effective moisture-blocking placement of traditional band-type matrix

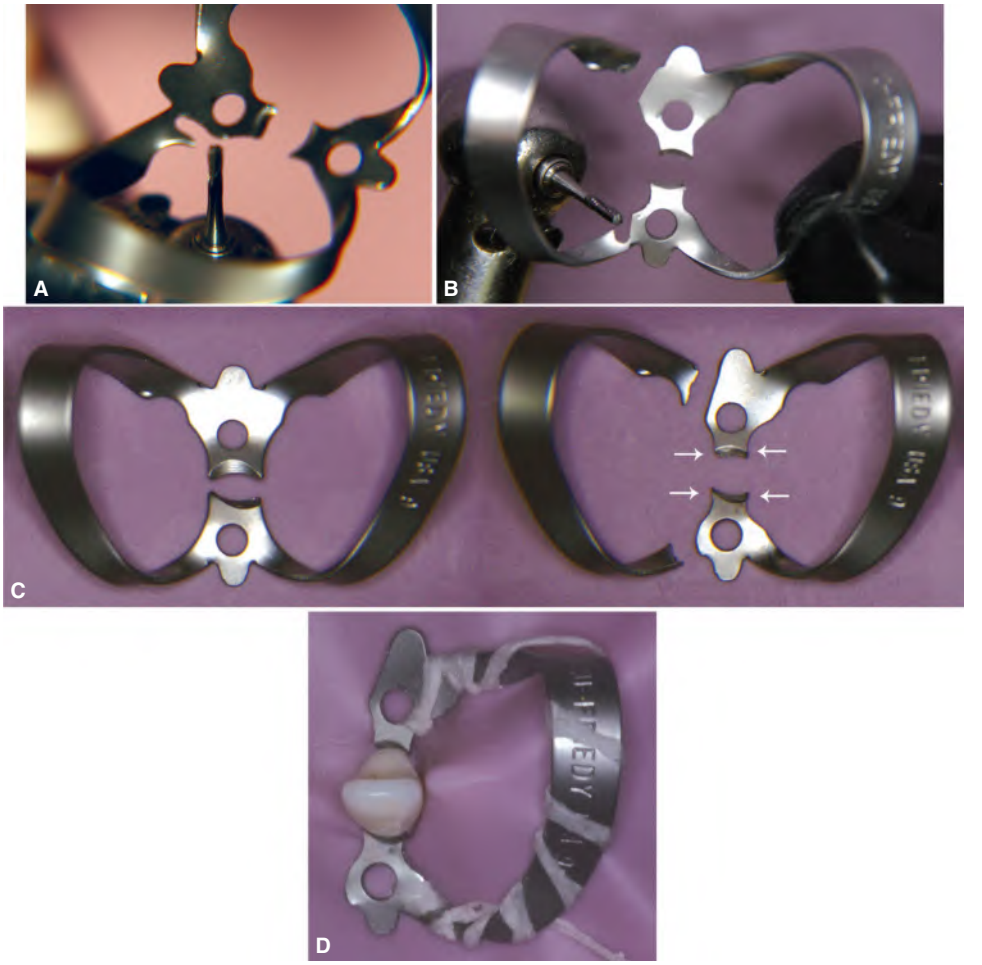

FIGURE 19-28 Modifying butterfly clamps. ***A,B***, Removal of one bow from a butterfly clamp. ***C***, Butterfly clamp before modification, reduction of the mesio-distal reach as well as one bow removed. ***D***, Modified clamp precisely seated on a mandibular incisor. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

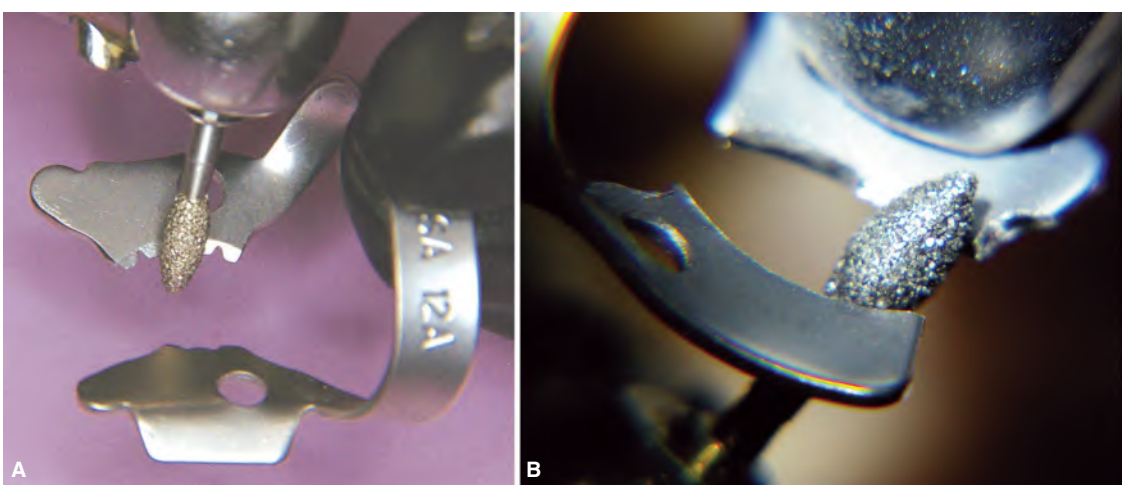

FIGURE 19-29 ***A***, Sharpening the tines. ***B***, Sharpening the smooth beaks. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

barriers, such as Toffelmire matrix bands, sectional matrices, and auto-matrix barriers, can be extremely difficult. Achieving extension of the band beyond the margin of the defect and wedging effectively when the defect is gaping and deep can be a formidable challenge. Durable blockage of the primary entry point for these contaminants over deep margins is an absolute requirement in the development of a preendodontic build-up with marginal integrity. While a flowable moisture-blocking barrier of Dycal can be practical in such cases, at times a more mousse-like barrier is preferred.

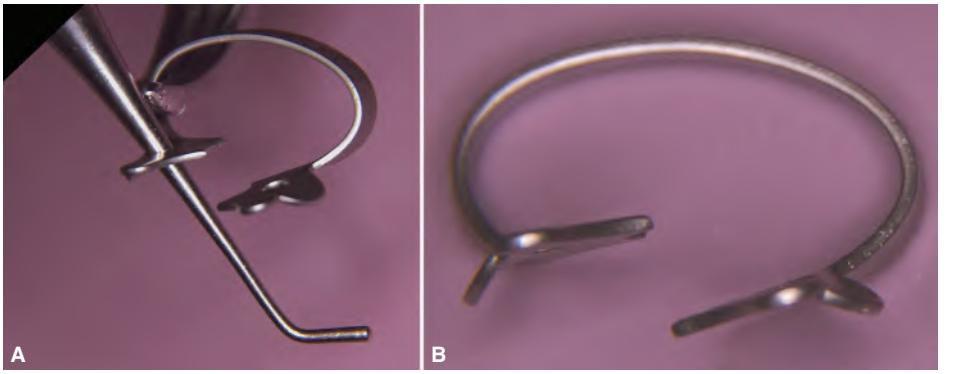

FIGURE 19-30 Changing the inclination of the beaks. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

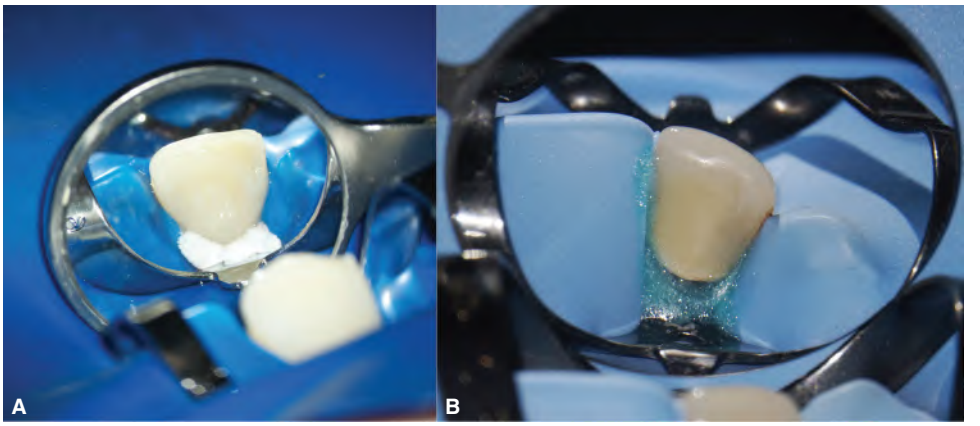

FIGURE 19-31 Isolation of maxillary incisors with the aid of flowable block-out materials such as ***A***, Oraseal Caulk and ***B***, OpalDam (Ultradent, South Jordan, UT) to seal out leakage. Figure 19-31B is (Courtesy of Dr. Carlos Aznar Portoles, Amsterdam, the Netherlands.)

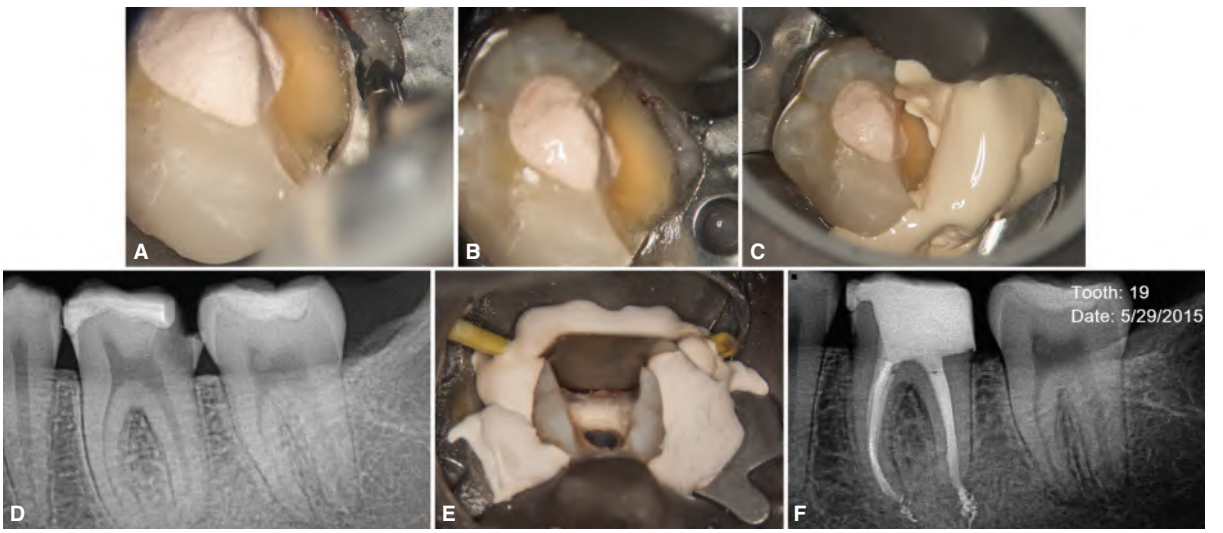

FIGURE 19-32 Dycal as a convenient flowable matrix barrier. ***A***, Cracked molar showing a fine flame tip composite finishing bur precisely cutting away the clamp metal along the margin of the fracture. ***B***, Completed clamp modification. ***C***, Injection of Dycal into the modified clamp space ensures an aseptic dental dam seal prior to endodontic access. ***D***, Preoperative radiograph showing extensive distal caries involving the pulp. ***E***, A "floating" sectional matrix held in position with two wedges and luted securely with Dycal, ***F***, This provides ideal isolation for endodontic procedures and/or for the final build-up. (Courtesy of Dr. Terrell F. Pannkuk, Santa Barbara, CA.)

FIGURE 19-33 ***A***, Dycal can be simply and efficiently mixed. A Centrix Needle Tube can be used to deliver the Dycal precisely into the areas it is needed to seal gaps and secure matrices after dental dam placement. ***B***, A 4:1 Dycal to Cavit blend provides a more mousse-like consistency. The Cavit portion should be drawn into one of the Dycal portions and mixed before initiating the setting process by drawing in the second Dycal portion.

The flowability of Dycal can be moderated by "cutting" it with a portion of Cavit. There are various formulations for Cavit, and readers may wish to bench-test a combination of different formulations to establish a preference. Consistent results can be achieved, however, using Dycal and Cavit G (3M Espe Dental, St. Paul, MN, U.S.A.) in a 4:1 ratio (4 Dycal to 1 Cavit) that renders a useful moisture-friendly consistency that will flow through either a green or orange AccuDose NeedleTube (Centrix, Shelton, CT, U.S.A.). The ratio may be varied to suit the clinician's preference or to compensate for the fact that Cavit tends to dry out over time. The 4:1 Dycal to Cavit ratio is determined by first dispensing onto a mixing pad or glass slab equal amounts of parts A and B of Dycal, and then a portion of Cavit equal to half of one of the Dycal portions. The Cavit should be drawn into one of the Dycal portions and mixed thoroughly before mixing in the second Dycal portion (Figure 19-33B). This sequence provides more ample working time as the setting process is not initiated until the second Dycal portion is mixed in.

Prior to mixing the Dycal and Cavit together, tissue troughing is often required to allow the flowable barrier to be placed beyond the level of the deep defect (Figure 19-34). Split dam is often required for these types of cases to facilitate troughing of the tissues beyond the deep defect without interference from interseptal segments of dental dam material. Infiltration of lidocaine with 1:50,000 parts epinephrine into crestal and papillary tissues can provide considerable hemostasis. One or two millimeters of axial wall subjacent to the defect should be exposed by troughing with a high-speed tapered diamond laser or via electrosurgery. If necessary, additional hemostasis can be achieved with the application of ferric sulfate-containing agents such as ViscoStat (Ultradent, South Jordan, UT, U.S.A.) or Quick-Stat FS (Vista Dental Products, Racine, WI, U.S.A.).

Dycal and Cavit are mixed and injected by NeedleTube into the gingival trough and over the exposed tissue. It can be helpful to swage the un-set hydrophilic material into the trough and against the adjacent tooth (if present) with a water- or alcohol-saturated cotton pellet; the latter providing greater acceleration of the setting process. The chamber can then be flooded with alcohol to continue the setting acceleration. The mature moisture-proof Dycal/Cavit barrier can be custom-trimmed from within the cavity to the exact deep cavity margin using various-sized slow speed round burs or a high-speed bull-nosed diamond. During this delicate cutback process, the deep margin is where the importance of the gingival troughing plays a key role. If the gingival troughing had not been performed, the flowable barrier would not have been able to be placed beyond the deep marginal defect. In that scenario, the attempt to custom-trim the barrier to the exact margin would perforate the barrier precisely at the critical deep margin, allowing blood, tissue fluids, and saliva to compromise the purpose of the barrier. Once the deep defect is completely isolated by this customized matrix barrier, the walls can be etched and primed, bonding agent can be applied, and a full preendodontic composite build-up or a *Half-Wall* (described in the Half-Wall section under "Pretreatment Build-up" below) can be placed. Prior to dismissal, the Dycal/Cavit is easily cracked away with an explorer.

Clamp Retention Methods

Bonded Composite Buttons

The absence of the typical anatomic height of contour on teeth that have been reduced by crown preparation may hinder clamp stability. Composite buttons can be bonded onto the cervical aspect of both the buccal/labial and/or lingual/palatal crown surfaces to act as an undercuts for clamp retention. A clamp with apically inclined beaks may provide improved engagement of tooth surfaces in select cases (Figure 19-35), and it can be a practical choice when composite "buttons" will be bonded after placement of the clamp.

Composite Donut

Despite the fact that other factors such as crown : root ratio, mobility, and periodontal condition may be within

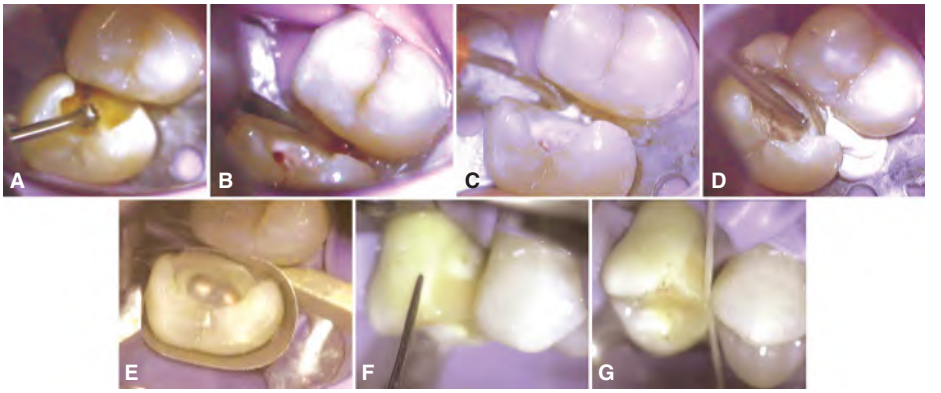

FIGURE 19-34 Dycal/Cavit Matrix Barrier and *Half-Wall*. After extensive caries removal, the prepared margin is deeply subgingival. The gingiva is troughed beyond the deep margin of the defect with a high-speed finger diamond or via laser or electrosurgery. The Dycal/Cavit blend is applied by first syringing a bead with a Centrix Needle Tube into the gingival trough and then flowing additional bulk over the exposed tissues and against the adjacent tooth (if present) to lock the material below the adjacent height of contour. An alcohol-saturated cotton pellet can be used to swage the unset blend into the desired shape to receive the flowable *Half-Wall* material later in the process. On setting of this mix, accelerated by flooding the cavity with alcohol, the durable moisture-proof barrier is custom-trimmed from inside the cavity to the exact margin of the deep defect with long-reaching stiff narrow shafted Munce Discovery Burs (CJM Engineering, Inc., Santa Barbara, CA) of various diameters. If composite will be the *Half-Wall* filling material of choice, as it was in this case, the cavity is etched and primed, and bonding agent is applied. A color-contrasting composite is injected to create a visibly distinct *Half-Wall* at the deep margin for convenience in subsequent crown preparation. Alternatively, compomer, glass ionomer, or a sandwich of glass ionomer and composite can be placed as the *Half-Wall* material. On polymerization of the *Half-Wall*, the flowable Dycal/Cavit matrix barrier is easily cracked away with an explorer. Root canal treatment is then completed, now under ideal endodontic dental dam isolation protocol. To incorporate the *Half-Wall* into the final build-up, the surface of the composite *Half-Wall* is freshened by sandblasting or by lightly brushing with a high speed diamond. A Toffelmire band or other band-type matrix barrier can be placed quite easily, now that there is a bonded *Half-Wall* where there had previously been only a gaping defect and aggressively wedged, creating a reliable gingival seal and facilitating the development of proximal contact (if desired/required as it was in this case). All available tooth structure is again etched and primed, and additional composite is bonded to the prepared tooth structure and to the freshened *Half-Wall* and contoured into a durable endodontic coronal seal and build-up with a color-contrasting deep margin and the desired proximal contact. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

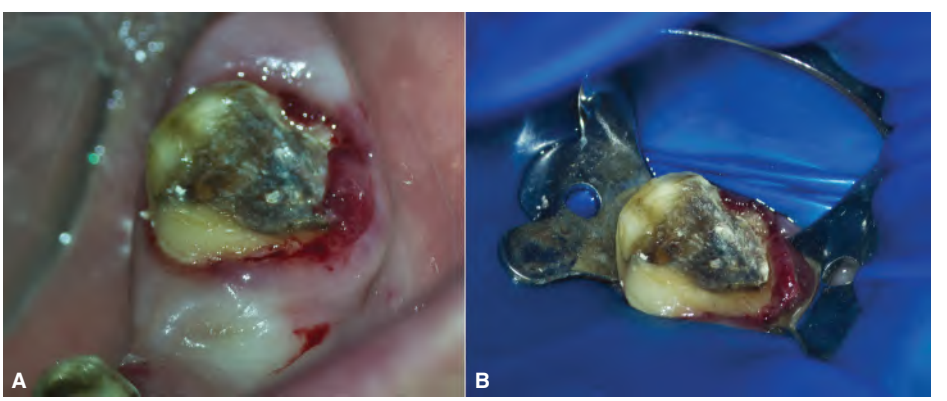

FIGURE 19-35 Isolation of a broken-down mandibular second molar using a clamp with apically inclined beaks to facilitate placement of a bonded composite button on the palatal. (Courtesy of Dr. Adham A. Azim, Buffalo, NY.)

normal limits, and there may even be adequate structure to support a restoration, clamp retention challenges alone can play a key role in the dentist's recommendation to extract a tooth. In addition to severe coronal breakdown, other factors affecting clamp stability include partially erupted teeth, misshapen teeth, or teeth with unfavorable contours such as in the case of teeth prepared with "draw" in anticipation of receiving a crown. Using common dental materials in uncommon ways, the Composite Donut offers the clinician an additional method for saving many teeth that might otherwise be extracted due to clamp retention challenges.

Technique

1. If the defect extends subgingivally, the gingiva is troughed beyond the margin of the defect with a

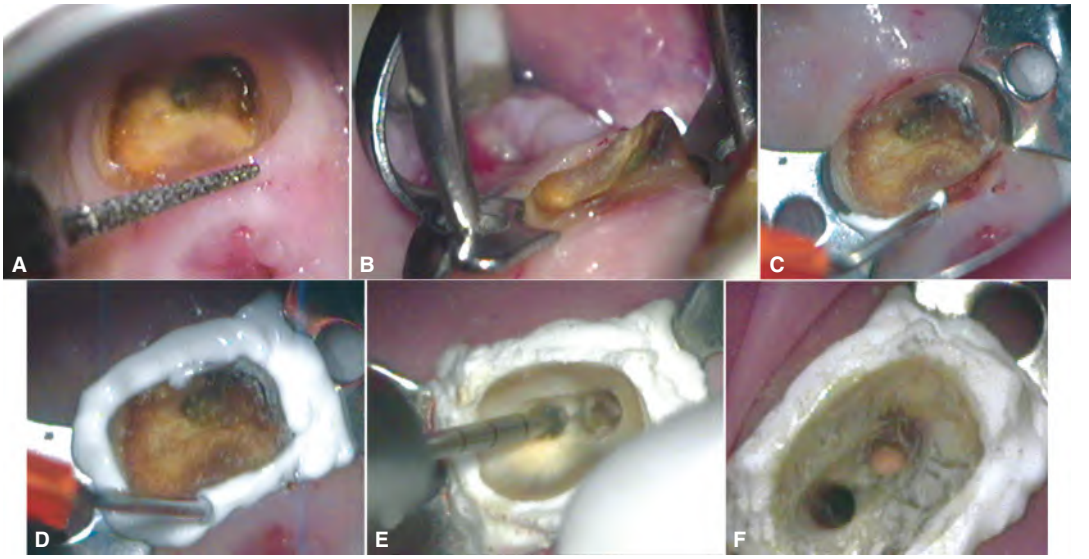

FIGURE 19-36 Composite donut technique. ***A***, Gingival troughing with high speed finger diamond to expose 1-2 mm of axial structure 360°. ***B***, Clamp is then placed. If required, hemostasis is achieved with ferric sulfate, and ***C***, a nonbonded composite bead is injected into the trough, ***D***, circumnavigating the tooth to close the loop, taking care to capture the beaks of the clamp within the bead. ***E,F***, Root canal treatment is then carried out under ideal endodontic isolation protocol. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

high-speed finger diamond (Figure 19-36A), radio-surgery device, or laser.

a. Prior infiltration of lidocaine with 1:50,000 parts epinephrine into papillae and crestal gingiva provides significant hemostasis.
b. 1–2 mm of exposed axial structure 360° is ideal for retention of the nonbonded composite donut.
c. Areas around the cervical perimeter where adequate axial wall is already exposed do not require gingival troughing.
d. As an alternative to 360°-gingival troughing, strategically positioned 1–2 mm deep gingival facets can be created with a high-speed finger diamond, radio-surgery device or laser in at least three well-spaced sites on the gingival cuff surrounding the tooth (Figure 19-37A–C) to provide tripodized stabilization of the composite donut. This can be done with the clamp in place, even if weakly- retained, so that the facets are not placed in areas where the beaks of the clamp will insert.

2. If required, hemorrhage is controlled with ferric sulfate.
3. Clamp is placed (Figure 19-36B) (if not already in place per previously mentioned Step 1d).
   a. If the clamp is secure, then the dental dam is placed at this point (proceed to Step 4).
   b. If the clamp is insecure, the dental dam should not be placed until after a clamp is stabilized with a composite donut.
   c. If single tooth isolation is required, and if there is a tooth on either side or both sides of the target tooth, then a spacer must be placed against the proximal aspect(s) of adjacent tooth/teeth so that the dam will be able to pass between the polymerized composite donut and the adjacent tooth. This spacer could be a material such as Cavit that can be thinly swaged against the proximal surface(s), or it could be a section of plastic matrix band, a short segment of a plastic drinking straw slit longitudinally or a piece of medical-grade Teflon PTFE tape (CS Hyde Company, Lake Villa, IL, U.S.A.) draped against the proximal surface(s) of adjacent tooth/teeth (Figure 19-38B,C).
   d. A flowable composite is syringed with a Centrix Needle Tube into the gingival trough and 360° around the cervical aspect of the tooth, without benefit of bonding, taking care to cover any exposed tissue, to block any gaps in the dam, and to capture the beaks of the clamp within the bead (Figure 19-36C,D).
   e. If facets were created as in previously mentioned Step 1d, the bead must flow into each of the facets as part of the 360°-donut (Figure 19-37D,E).

4. a. A 360° composite bead is critical as it provides the closed-loop reciprocation required for robust retention of the nonbonded donut.
   b. If multiple teeth are exposed, and there is proximal contact on the mesial and/or the distal of the target tooth, the composite bead must flow up and over the marginal ridge and down the other side to close the loop of the 360°-donut (Figure 19-39). In the case where a spacer has been placed against the proximal surface(s) of the adjacent tooth/teeth, removal of the spacer(s) after polymerization of the composite (Figure 19-38D) will

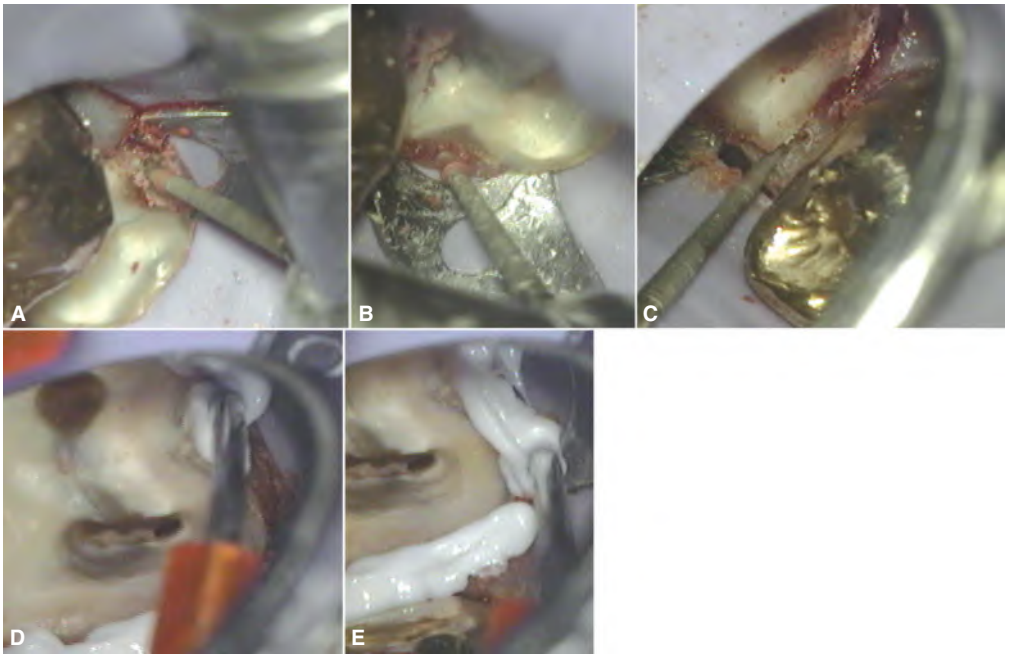

FIGURE 19-37 Gingival facets serve as an alternative to 360° gingival troughing to retain the Composite Donut. ***A–C***, Three or four well-spaced small facets created with a high-speed finger diamond offer the necessary reciprocation to stabilize the nonbonded donut. Facets should be cut with the clamp in place when possible, ***D,E***, to ensure they will not be obscured by the beaks of the clamp, but will remain accessible to the composite bead when the clamp is in place. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

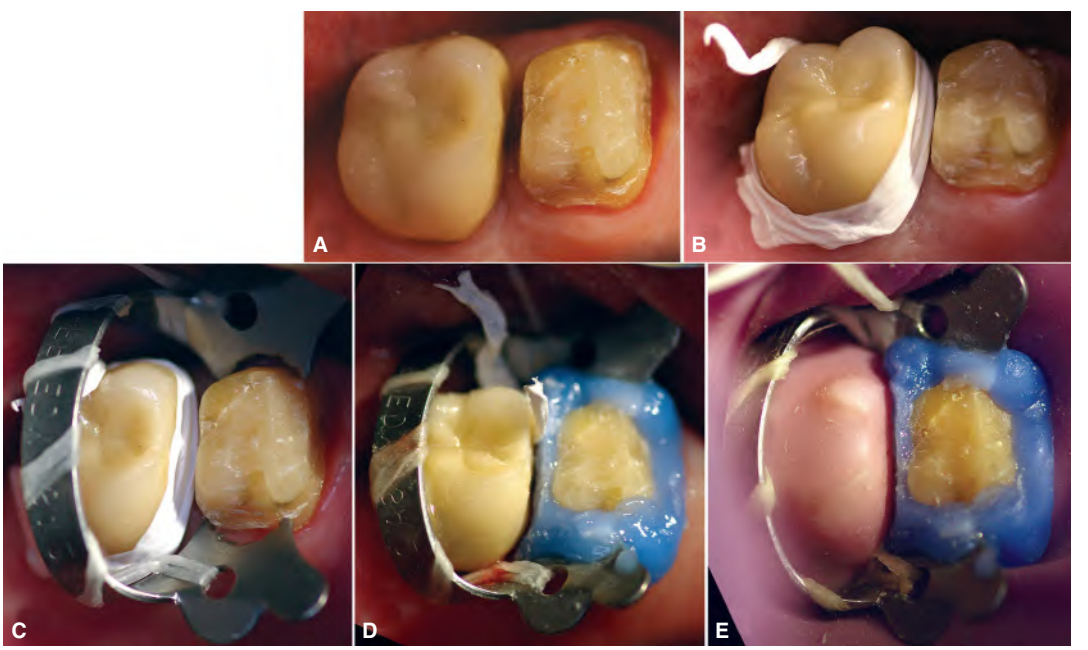

FIGURE 19-38 Various types of spacers. In this case, a section of medical grade PTFE tape can facilitate single tooth isolation with composite donut, allowing the margin of the dam to pass between the donut and the adjacent tooth/teeth after removal of the spacer upon polymerization of the composite. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

allow the margin of the dental dam to slide perfectly under the donut (Figure 19-38E).

5. At this point, the clamp should be completely stabilized, and the tooth under ideal endodontic isolation. If the composite bead overlies any portion of the deep margin(s) of the tooth (Figure 19-40A), it can be carefully cut back from within the cavity to the exact margin(s) with high-speed diamonds and/or surgical-length slow speed round burs (Figure 19-40B,C).

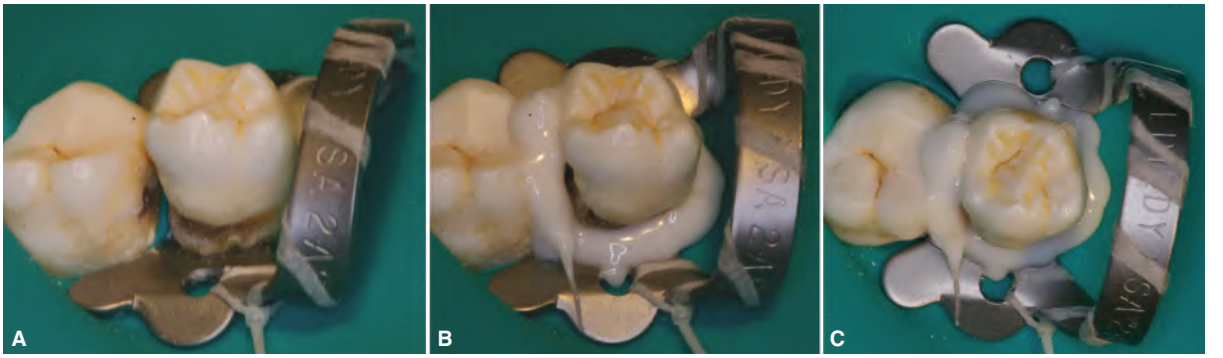

FIGURE 19-39 When multiple teeth with proximal contacts are exposed, the composite bead must flow up and over the marginal ridge, and then back down the other side to close the 360°-loop of the donut. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

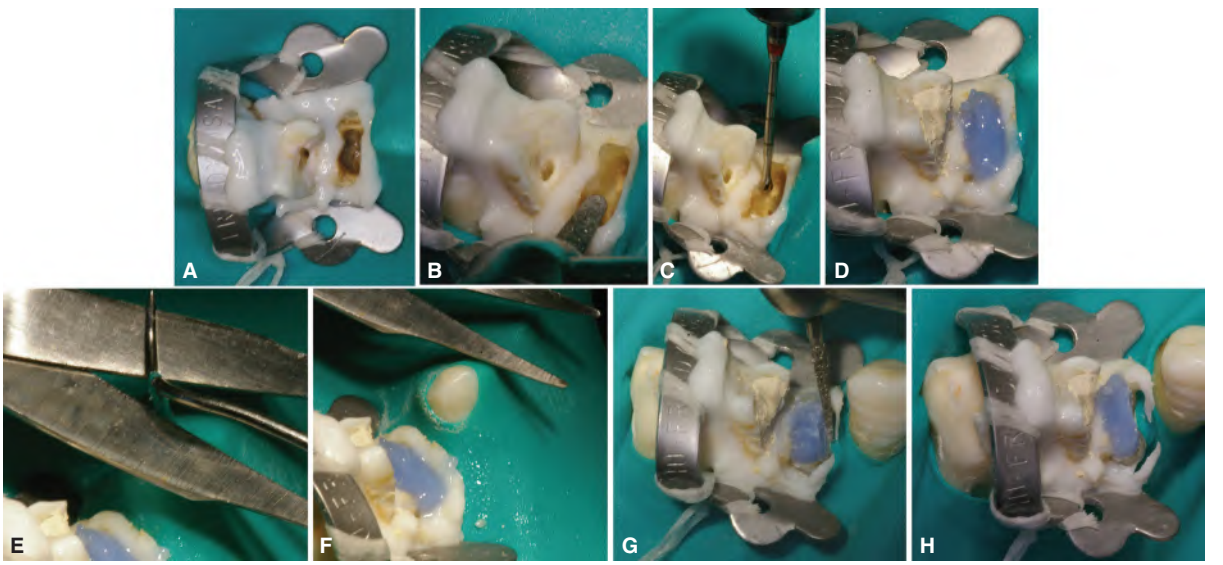

FIGURE 19-40 Any portion of the bead that overlies the margin of the cavity ***A,*** can be cut back to the precise margin with high-speed diamonds and/or surgical-length slow-speed round burs, ***B,C***. In anticipation of a subsequent visit, color-contrasting composite can be bonded to form a *Half-Wall* using the donut as a matrix barrier, ***D***. To facilitate dissection of the nonbonded donut from the bonded *Half-Wall* without interference from the interproximal dam, the dam is lifted from the adjacent marginal ridge with an explorer and snipped with an iris scissors, ***E,*** exposing the adjacent tooth, ***F***. A high-speed finger diamond dissects the composites of dissimilar colors from each other, ***G,H***. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

6. If a second visit is anticipated, a color-contrasting preendodontic bonded *Half-Wall* (see *Half-Wall* in subsequent subsection) can be placed using the composite donut as a matrix barrier (Figure 19-40D). The bonded *Half-Wall* will simplify isolation at the next visit. A *Half-Wall* color that contrasts with the nonbonded donut facilitates clean dissection of the nonbonded donut from the bonded *Half-Wall* with a high-speed finger diamond (Figure 19-40G,H) under high magnification and high-intensity illumination prior to dismissal. It is very important not to leave any residual color-contrasting nonbonded composite overlying the margins and captured under the bonded build-up material, as this would lead to marginal leakage and failure of not only the restoration, but possibly the endodontic therapy as well. Once dissected from the *Half-Wall*, the donut will readily crack away with a tweak of the clamp via the dental dam forceps.
7. If this will be the final visit, the final build-up should be placed at completion of this visit.
   a. If the build-up will be composed of a bonded composite or hybrid material, it should be of a color that contrasts with the nonbonded donut so that the donut can be completely dissected away from the build-up material with a high-speed finger diamond under high magnification and high-intensity illumination prior to dismissal. The donut will then readily crack away from the build-up with a tweak of the clamp via the dental dam forceps (Figure 19-41).
   b. An alloy build-up can be a convenient alternative as the composite donut is a durable matrix barrier able to withstand alloy condensation, and the donut does

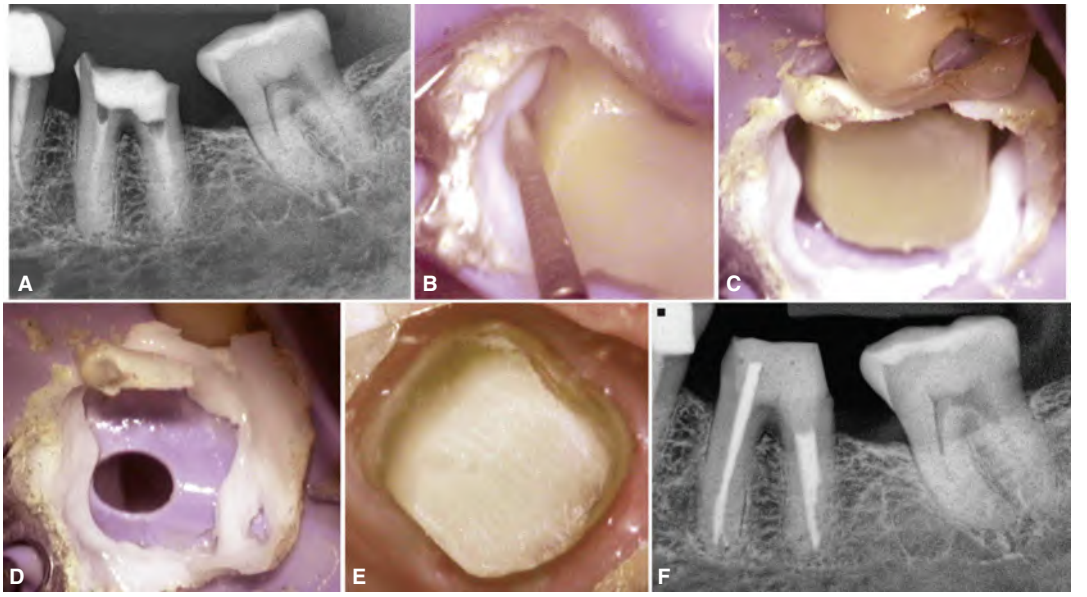

FIGURE 19-41 In a tooth with minimal supra-gingival structure, ***A***, the composite donut aids in clamp retention. At completion of root canal treatment, the color-contrasting donut is dissected from the final build-up with a high-speed finger diamond under high magnification and high-intensity illumination, ***B,C***. With a tweak of the clamp by the forceps, the donut cracks away, often nearly intact, ***D***, leaving the build-up ready for final preparation, ***E,F***. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

not require dissection from the dissimilar material and will readily crack away from the alloy with a tweak of the clamp via the dental dam forceps.

Pretreatment Build-up

Band Isolation

Teeth with more extensive loss of structure may require restorative procedures to facilitate isolation. The use of the copper band or the orthodontic band provides an effective method for building up structurally compromised teeth for the purpose of isolation. An orthodontic band may be quick and easy to place but a copper band can be customized to provide ideal contours and marginal adaptation.[86] The band serves as an external matrix barrier to contain composite, IRM, or other reinforced temporary restorative materials in order to provide adequate crown height. The band can also remain on the tooth until treatment is complete to act as part of the clamp-retention apparatus. Bands, however, may impinge on gingival tissues resulting in irritation and laceration, so caution should be exercised when using this method. Care should also be taken to avoid blockage of the canals with preendodontic build-up material.

The flow characteristics of Dycal make it ideal for securing a copper band so that the margin of the area, to be restored, has the matrix form properly contoured and fit (Figure 19-42). Wedges can be placed to ensure ideal adaptation. If necessary, a clamp with inverted beaks can be positioned on the gingiva to secure the dental dam prior to placement of the copper band. Although virtually all teeth can be aseptically isolated with the combination of a band and a sealing material like Dycal injected from a Centrix Needle Tube, it is very important to assess restorability before placement of a temporary build-up.

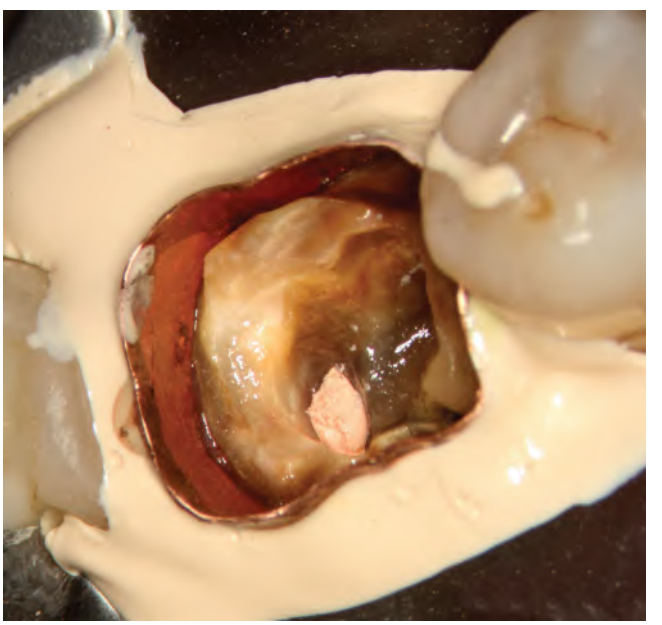

FIGURE 19-42 A copper band secured with Dycal facilitates aseptic isolation of teeth lacking undercuts for secure dam or clamp placement, and also creates an ideal custom matrix for placement of the postendodontic restoration. (Courtesy of Dr. Terrell F. Pannkuk, Santa Barbara, CA.)

Half-Wall

The preendodontic *Half-Wall* is a limited restoration of only that portion of the tooth required in order to facilitate ideal

endodontic isolation (see Figure 19-34, also previously referenced in Dycal/Cavit section). Because of its narrow purpose, the *Half-Wall* does not require development of the marginal ridge(s), proximal contact(s), or occlusal surface detail. The limited scope of the *Half-Wall* allows the clinician to hyperfocus on only the most compromised portion of the tooth and the surrounding tissues, achieving what is otherwise often very difficult, control of fluid contaminants, leaving all successive procedures to be performed under uncomplicated and ideal endodontic isolation protocol. At completion of endodontic procedures, the *Half-Wall* can be easily integrated into the full build-up.

Canal Projection

Canal projection is a technique that facilitates preendodontic build-up of broken-down coronal and radicular structure while preserving individualized access to the canals.[87,88] While any of a number of syringeable materials (glass ionomer, temporary cements, permanent cements, etc.) and even "packable" composites may be used to project the canals, bonded injectable autopolymerizing composites have proven to be the most versatile and reliable build-up material for this technique. The advantages of the technique are numerous.

1. **Isolation**: When deep coronal-radicular defects prevent clamp retention, a tooth is often considered to be endodontically untreatable. Canal projection essentially replaces missing structure to facilitate clamp retention, rendering many structurally debilitated teeth endodontically treatable.
2. **Seal of chamber floor**: The accessory canals that exit the chamber floor into the furcation are well documented[89–91] and can conduct inflammatory agents from the deteriorating pulpal system into the furcation.[92] Additionally, when caries or exploratory procedures lead to a paper-thin chamber floor, the risk of contaminants, extending into the furcal apparatus, increases significantly.[93] Often, such complicated cases require multiple visits, and the interappointment interval with customary temporary coronal seal provides opportunities for serious furcation degradation, particularly if a patient does not return in a timely fashion for completion of treatment.[94] Sealing the chamber floor early in treatment greatly reduces this risk.[95] As an initial step in the endodontic process, canal projection using bonded composite achieves this early objective.
3. **Elongation of canals**: The canal projection process elongates the "hydraulic chamber" of each canal from the access cavity floor to the occlusal surface, offering advantages during the hydraulic condensation of obturation materials. Essentially all currently accepted warm vertical condensation techniques (conventional warm vertical condensation, warm sectional condensation, continuous wave condensation) rely on the rheology that takes place between the chamber floor and the primary exit of the canal.[96] These methods focus their hydraulic effects on the apical region while only incidentally addressing those accessory and lateral canals that exit from the more coronal aspects of the system. In contrast, when the canal is projected, the hydraulic chamber is elongated as much as 5–10 mm such that the rheology occurs not just between the chamber floor and the primary canal exit, but also between the occlusal surface and the primary exit. This allows for additional warm vertical condensation "strokes," additional warm "sections" of gutta-percha, or additional milliseconds spent in the continuous wave of condensation. Regardless of the hydraulic condensation method used, canal projection provides greater opportunity to address coronally-positioned exits.
4. **Overlay of perforation repair**: Early repair of perforation defects can have a significant effect on success of perforation treatment.[97] As such, performing perforation repair, prior to completion of endodontic therapy, is often preferable. The delicate nature of these types of repairs, however, leaves them vulnerable to re-aggravation during subsequent cleaning, shaping and obturation procedures. It can be most disheartening to perform a complicated perforation repair procedure only to see it herniate into adjacent PDL and bone under obturation hydraulics. Delaying the repair until endodontic therapy is completed may put the tooth at risk if the treatment cannot be completed in a single visit. In addition, performing endodontic therapy in the presence of an un-repaired perforation may needlessly expose the perforation site to irritating solvents and irrigants and to accidental penetration by instruments. In many cases, perforation repair with MTA can be performed prior to completion of endodontic therapy. After confirmation that the MTA has set, canal projection offers a means of fortifying the repair by overlaying it with a bonded resin or glass ionomer prior to proceeding with endodontic therapy.
5. **Prevention of perforation**: In those circumstances that lead to a paper-thin chamber floor, the risk of inadvertent perforation with a file, explorer, or bur can be reduced or eliminated by placing a preendodontic build-up via canal projection.
6. **Recontour of irregularities on chamber floor and walls**: Irregularities on the access cavity floor and walls can lead to complications during treatment. Misdirected access cavities, defects and irregularities in canal walls, misdirected post preparations and ledges, and so on can be "corrected" by essentially reconstructing the walls and floor around "internal matrix barriers" via canal projection.
7. **Reduce potential for crack initiation and/or propagation**: On posterior teeth in particular, the time period between initiation of endodontic therapy and placement of a restoration that provides cuspal protection can introduce the risk of coronal-radicular fracture; the longer the time period, the greater the risk.[98] Bonded coronal build-up has been shown to reduce this

risk.[99,100] Bonded canal projection performed at the first endodontic treatment visit imparts a measure of protection against the development of a fracture from the time treatment is initiated until the tooth is permanently restored. In teeth that present with incipient fractures, early placement of a bonded build-up can reduce the likelihood of propagation of these fractures as well.

8. **Prevention of ingrowth of tissues**: When coronal or radicular defects extend to axial walls at or beyond the gingival level, tissue ingrowth leads to contamination during endodontic therapy and subsequent restorative procedures. Preendodontic build-up, as with canal projection, eliminates this complexity.
9. **Individualization of canals**: In multi-canal teeth, as the projectors pass through the build-up material to the occlusal surface, they can be oriented such that they are slightly separated from each other before the build-up material polymerizes. This can simplify management of canals that lie in close proximity to each other on the chamber floor. It also provides for individualization of the canals so that specific solvents, irrigants, medicaments, lubricants, and other agents can be placed into specific canals.
10. **Elimination of "blind exploration" on the chamber floor and improvement of mechanics related to handpiece-driven files**: Once all canals have been located, straight-line access created, and the canals projected to the occlusal surface of the build-up, the resultant projected orifices will lie clearly visible on the surface of the projection build-up, no longer obscured by prominent marginal ridges and other visual obstructions. This also creates a significant advantage for entry of handpiece-driven files, particularly nickel–titanium files, into mesial canals of molars. By virtue of their increased long-axis dimension, the tips of handpiece-driven files can be difficult to introduce into mesial canals, and nickel–titanium files even more so because they will not retain a bend. When the canals are projected onto the occlusal surface of the preendodontic build-up, however, it becomes a simple matter to place the file tip within the rim of the projected canal on the occlusal surface, and then by merely advancing the file, it is driven smoothly into the canal.

Technique Although root canal projectors, invented and patented by Dr. C. John Munce, are currently not commercially available, the technique as described in the fololwing remains a viable preendodontic build-up method, and plastic Capillary Tips (Ultradent, South Jordan, Utah, USA) can be cut to length to serve in every way as "root canal projectors" (Figure 19-43). As used in this section, the term "root canal projector" refers to a cut segment of a capillary tip. A variety of commonly available items have been used to project canals, including gutta-percha cones and silver points, and yet, a tapered plastic sleeve carried to the canal on a fluted file has proven to be most reliable, not only as a matter of remaining securely "pinned" to the canal and centered during the injection of flowable build-up materials, but the sleeve can also serve as a convenient stopper under the temporary seal in multiple-visit cases.

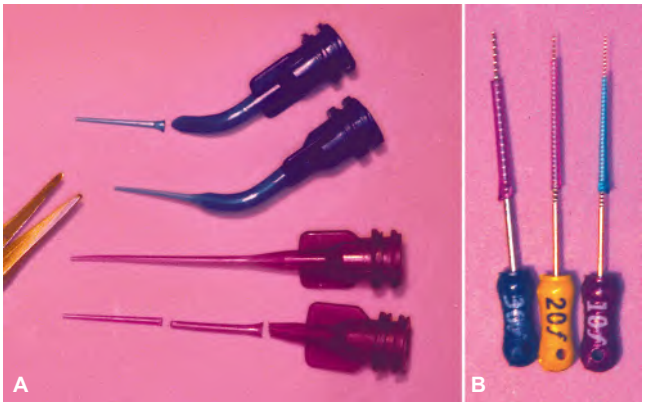

FIGURE 19-43 ***A,B***, Although root canal projectors are currently not commercially available, plastic capillary tips (Ultradent, South Jordan, Utah) can be cut to length to serve in every way as "root canal projectors." (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

1. In a tooth with severe coronal breakdown (Figure 19-44A–C), after gaining access and locating all canals, it is essential to perform a thorough high-magnification examination of the floor and walls and to examine as deeply as possible into all canals to ensure that all branching and other possible irregularities have been identified prior to proceeding.
2. In curved canals, straight-line access is created to the first significant curvature by removing dentin "triangles" (Figure 19-44D) with small slow-speed round burs, working into the bulk of each root (i.e., working away from the furcal aspect of the root). If there is no significant curvature in a particular canal, then the orifice should be dimpled to the depth of a #1 slow-speed round bur (Figure 19-44E).
3. An appropriate matrix barrier is applied, and all bondable surfaces are etched and primed. Files are selected that will fit well into the body of each canal, and root canal projectors, tapered plastic sleeves of various dimensions depending on orifice diameters and canal proximities, are placed onto the files. The files are then inserted into the canals, and the projectors are slid apically until they are drawn into the seat of the previously-prepared dimples or straight-line access. In this particular example (Figure 19-44F,G), medium-sized projectors were used in the distal and palatal canals and projectors of smaller dimension were used in the MB-1 and MB-2 canals to accommodate their proximity to each other. With the canals occupied by the files and projectors, auto-polymerizing bonding agent is applied and distributed over all primed surfaces with a blast of air. Auto-polymerizing composite build-up material is then injected from the chamber floor to the cavosurface of the access cavity with a Centrix Needle Tube.

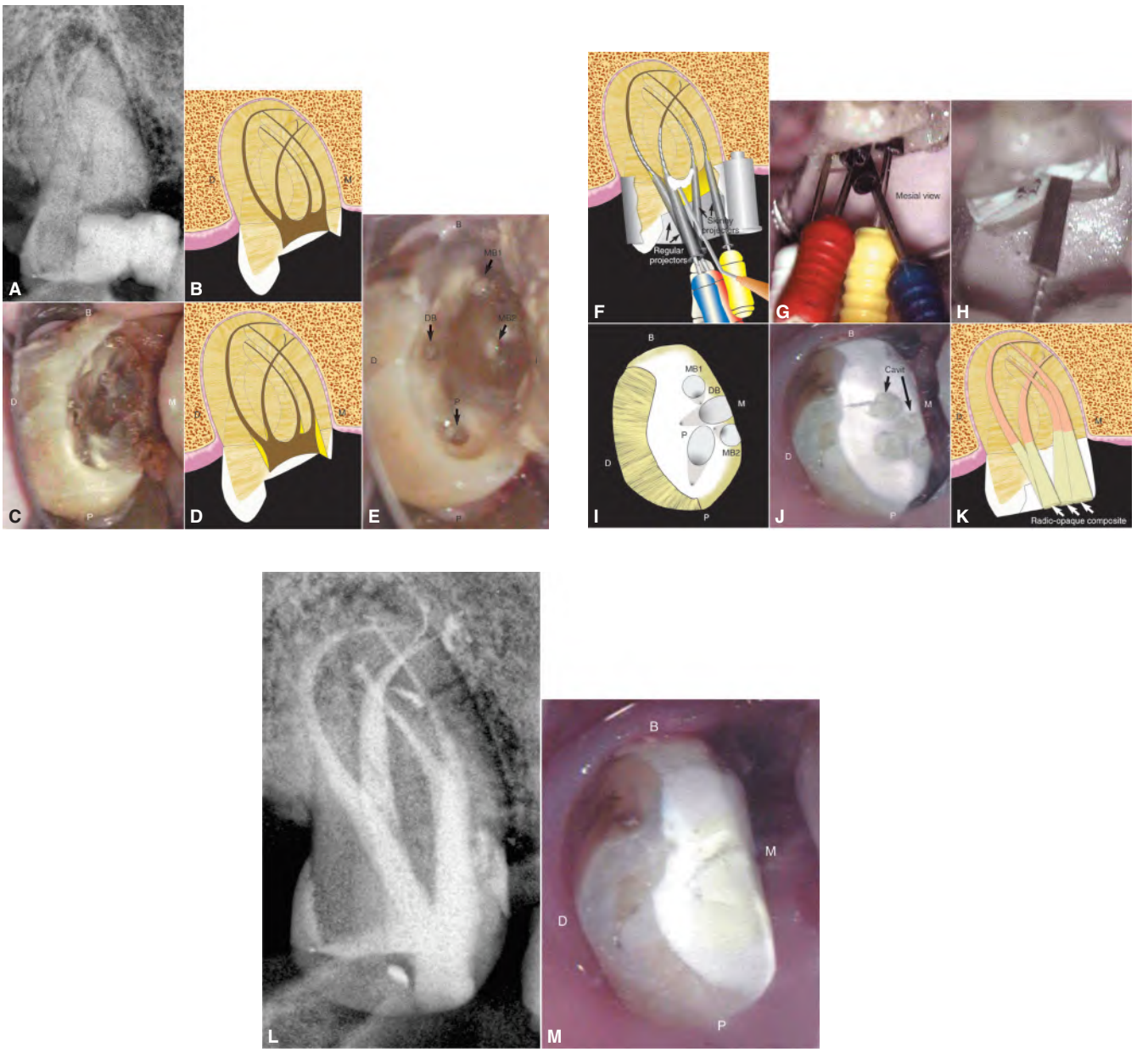

FIGURE 19-44 ***A–C***, Radiograph, illustration, and clinical view of maxillary third molar with severe coronal breakdown. ***D***, Straight-line access is created by removing dentin "triangles" (yellow) with small slow-speed round burs. ***E***, Straight-line access or dimpling of each orifice creates a seat to receive the projectors. ***F,G***, Bondable surfaces are etched and primed, and files with projectors are placed prior to application of bonding agent and injection of composite build-up material from the floor to the occlusal surface. In this example, medium-sized projectors were used in the distal and palatal canals and projectors of smaller dimension in the MB-1 and MB-2 canals to accommodate their proximity to each other. ***H***, On polymerization, the projectors are removed by engaging them with a Hedstrom file and withdrawing. ***I***, Projected orifices may lie in various configurations on the surface of the build-up. ***J***, Between visits, shortened projectors are replaced into their respective canals and sealed with Cavit. ***K***, Illustration of completed canal projection case. ***L***, Radiograph demonstrates "elongated" canals extending from root ends to occlusal surface. ***M***, Canal projection facilitates immediate crown preparation. (Courtesy of Dr. C. John Munce, Santa Barbara, CA. Adapted with permission from Cohen S and Hargreaves, KM (eds), *Pathways of the Pulp*, 9th ed. St. Louis, MO: Elsevier; 2006. pp. 127–132.)

Auto-polymerizing resin is preferred because of the typical depth of endodontic access cavities and because of the light-blocking effect of the files and projectors.

4. On early signs of polymerization, the files are removed, leaving the projectors imbedded in the build-up material. A bull-nosed diamond is used to contour the composite and to flatten the occlusal surface along with the imbedded projectors. Leaving the projectors imbedded at this stage prevents composite debris from collecting in the projected canals.
5. The projectors are removed by engaging a #60 Hedstrom file in the lumen of each projector and withdrawing

(Figure 19-44H). As with the original projectors, the external surfaces of capillary tips exhibit a releasing agent that provides for reliable removal from all types of build-up material. Depending on how the projectors are arranged relative to each other as they traverse from the chamber floor through the access cavity to the occlusal surface, the projected orifices will lie in various configurations on the occlusal surface (Figure 19-44I), and the particular orientation of the projectors is largely adjustable according the clinician's preference. This procedure creates durable endodontic reference points.

6. If a second treatment visit is required, the projected canals may be conveniently sealed by snipping 2 mm from the large-diameter end of the projectors, re-inserting them into their respective canals, and sealing with Cavit (Figure 19-44J). On the patient's return, the Cavit seals are removed with a #6 slow speed round bur, a #60 Hedstrom file is again engaged in the lumen of each projector, and the projectors are withdrawn, leaving the canals free of temporary filling debris.
7. Following cleaning, shaping and obturation, the projected portions of the canals are "dressed" with a high-speed football diamond and/or slow speed round burs to remove residual sealer and to freshen the composite, making it receptive to the final composite that is injected directly over gutta-percha from the sub-chamber level to the cavo-surface of each projected canal (Figure 19-44K). Selecting a composite for this final step that is more radio-opaque than the build-up composite creates a unique radiographic appearance showing each canal continuously flowing from the root end to the occlusal surface (Figure 19-44L). Crown preparation can proceed immediately (Figure 19-44M).
8. A post can be easily placed through the projection build-up at the completion of endodontic therapy. Using either a high-speed football diamond or a slow-speed #6 round bur, the projected portion of the canal to receive the post is enlarged to the level of the chamber floor. According to the particular type of post to be placed, the radicular aspect of the canal is prepared in the typical fashion, and the post is cemented or bonded into the canal. The coronal portion of the post should be surrounded by syringeable composite, effectively anchoring the projection build-up to the natural tooth structure.

Tissue Management Procedures

Large carious lesions or resorptive defects can lead to gingival tissue proliferation into the access cavity, impeding dental dam isolation. Methods for removal of this tissue include resection via scalpel, rotary instruments, laser, or electrosurgery if the remaining tooth structure lies above the bone. Electrosurgery allows clean removal of the gingival tissue combined with controlled hemostasis, simplifying subsequent isolation. Caution should be exercised when using electrosurgery, as it can lead to heat necrosis of the surrounding bone. Soft tissue laser is another method for removal of excessive gingival tissue with minimal resultant hemorrhage.

Crown Lengthening Procedures

Rapid Root Extrusion

The placement and retention of clamps during dental dam isolation can be challenging in teeth exhibiting inadequate coronal tooth structure (Figure 19-45). When indicated, rapid extrusion methods can facilitate clamp placement by repositioning structurally compromised teeth more favorably, coronal to the alveolar crestal bone. The treatment concept requires moving the tooth rapidly to minimize concomitant coronal migration of the supporting periodontium. Following the forced extrusion procedure, a stabilization period allows attachment fibers to accommodate the new coronal location.[101,102] Coronal repositioning can be achieved either orthodontically or surgically. However, unlike conventional orthodontic movement, rapid orthodontic extrusion involves forces greater than 50 grams.[103]

Heithersay[104] introduced the concept of orthodontic extrusion in 1973 as a treatment for fractures located in the coronal third of the root. Simon and colleagues,[105] proposed expanding the indications to include caries, root fractures, and iatrogenic perforations below the crestal bone. In separate reports, Emery[106] and Smit and colleagues[107] demonstrated the effectiveness of rapid extrusion for management

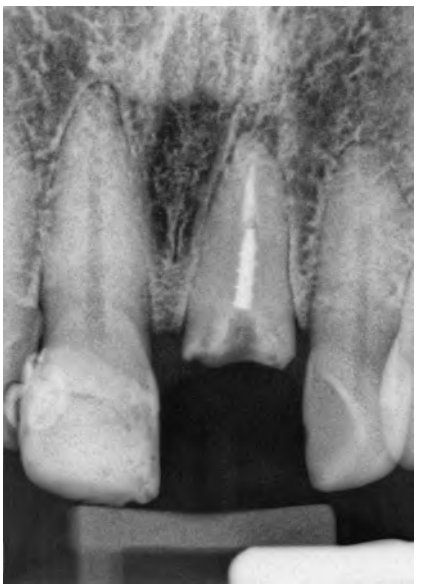

FIGURE 19-45 Maxillary central incisor with post fractured to subosseous level and coronal structure fractured to subgingival level. Difficult to place and retain a clamp and impossible to restore while respecting biologic and mechanical requirements. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

of resorptive defects at or near the crest. Among factors critical to the treatment outcome are crown to root ratio, periodontal health, oral hygiene, esthetic appearance, opposing occlusion, as well as tissue biotype, root length, and root morphology. A clear understanding of the risks, benefits and alternative treatment options will contribute to patient acceptance and compliance.

Rapid extrusion technique In compromised teeth where clamp placement is difficult because of insufficient tooth structure, the split dam technique may be used to create sufficient isolation to facilitate shaping the root canal system and placing calcium hydroxide prior to extrusion[108,109] to accommodate placement of a post hook (Figure 19-46). Orthodontic wire of sufficient rigidity (0.036 gage) is initially adapted to a study model either facially or lingually/palatally depending both on the relationship of the teeth adjacent to the target tooth and intercuspation with the opposing teeth. Though rigid, the wire should be flexible enough to be reshaped to pass directly over the tooth or teeth being treated to ensure a vertical extrusion path (Figure 19-47).

The technique uses one of various types of elastomeric agents to connect a bonded facial or lingual/palatal arch wire to the post hook that is cemented into the root canal of the target tooth. Adequate post retention requires a minimum post length equal to half the length of the remaining root structure.[105] The post hook can be cemented with crown and bridge cement or with IRM. This post hook will typically be removed following the post-extrusion retention period, so

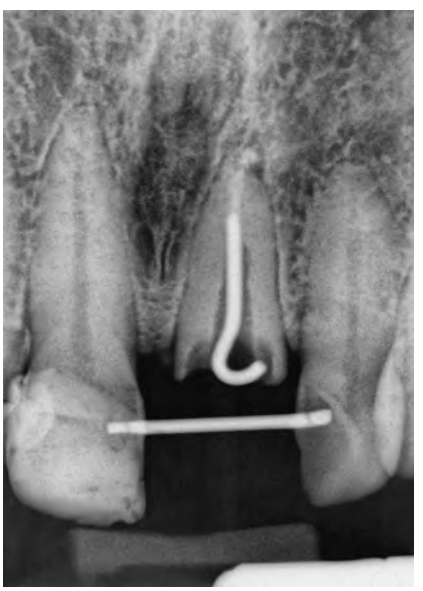

FIGURE 19-46 After removal of fractured threaded post under split dam isolation extending from the maxillary right central incisor to the maxillary left lateral incisor, the canal is shaped, and calcium hydroxide is placed in order to suitably stabilize the system for placement of a post-hook in preparation for extrusion. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

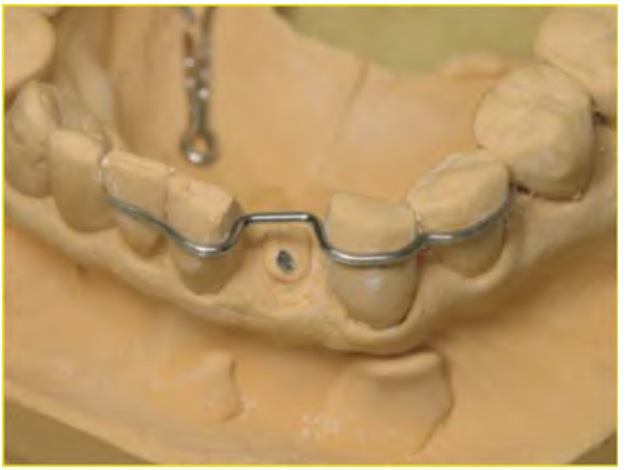

FIGURE 19-47 Stainless steel orthodontic wire of at least 0.036 gauge can be bent either in the mouth or on a stone model as in this case. (Courtesy of Dr. Fred A. Berry, Loma Linda, CA.)

the type of cement used should take this into account along with the dimensions of the post space. For example, if the space is long and parallel-sided, a cement of lesser retentive capacity can be used. Conversely, post spaces that are shorter and tapered, will require a luting agent with stronger retention properties. Post removal at treatment completion can be simplified by lubricating either one side of the post or the apical third or half of the post prior to cementation.

The labial arch wire is bonded with composite to the abutment teeth. Silane-enhanced bonding should be used if an abutment tooth has a porcelain crown. In the case of abutment teeth with full metal restorations, orthodontic bands with brackets can be attached to accommodate the wire. A segment of Elastomeric Chain (Rocky Mountain Orthodontics, Denver, CO, U.S.A.) (Figure 19-48A) can be attached to the hook and stretched over the arch wire and back to the hook to activate the appliance. This can be repeated several times with the same segment of chain to increase the extrusive force. Alternatively, many types of elastomeric thread are available (Figure 19-48B) that can be used to activate the appliance by feeding a 5-inch segment of thread through the hook, stretching it tightly, triple-knotting over the arch wire and snipping the ends. Often several such segments will be required to provide adequate extrusive force. Depending on the occlusion, it may be necessary to rotate the knots away from the functional fossa.

The shape of accessible surfaces may allow bonding of orthodontic brackets directly onto both abutment teeth near the incisal edge. A similar bracket can also be gingivally positioned on the target tooth. The apparatus is then activated with either a wire (Figure 19-49) or elastic thread (Figure 19-50). Regardless of the type of elastic agent selected, an additional convenient means of attaching to the target tooth involves placing a TMS pin (Coltene/

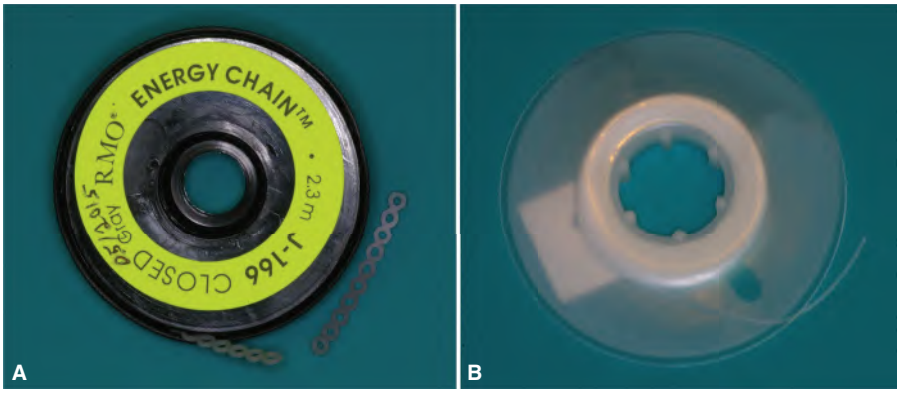

FIGURE 19-48 ***A***, Elastomeric Chain (Rocky Mountain Orthodontics, Denver, CO). For rapid extrusion purposes, chain segments will be short, consisting of only 2-4 "links" in order to exert adequate force when activated. ***B***, Elastomeric Thread (Smart Practice, Phoenix, AZ). For rapid extrusion purposes, thread segments should be 4-5 inches in length for convenience in activating and tying.

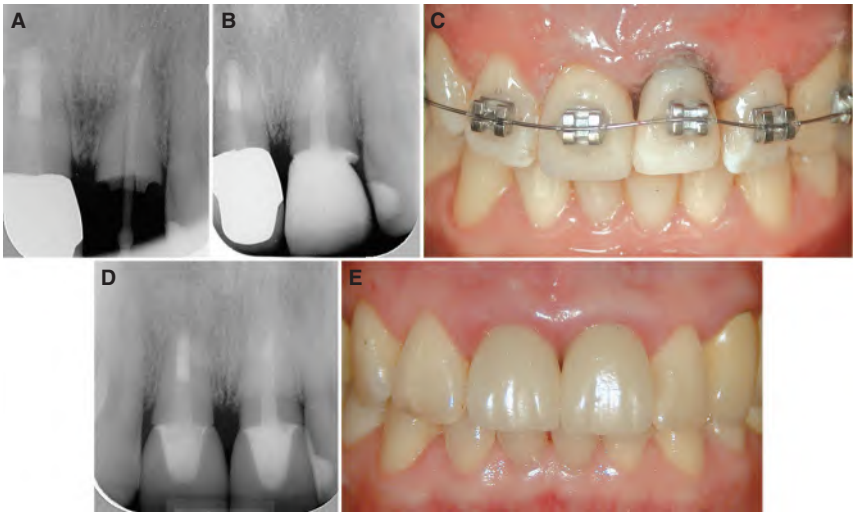

FIGURE 19-49 ***A***, Severely debilitated maxillary central incisor completely lacking supragingival axial structure. ***B***, Apical seal with MTA, fiber post bonded and a temporary crown cemented. ***C***, With bonded brackets on adjacent teeth and a gingivally offset bracket on the target tooth, an arch wire was inserted to apply extrusive force. ***D***, Eight-year follow-up radiograph showing completely normal periapex. ***E***, Clinical photograph at 8 years showing pristine gingival architecture. (Courtesy of Dr. Marga H. Ree, Middenbeemster, the Netherlands.)

FIGURE 19-50 Bonded plastic brackets on the maxillary right canine and the maxillary right central incisor and a gingivally offset bracket on the maxillary right lateral incisor. Elastomeric thread is used to apply extrusive force (arrow).

Whaledent, Cuyahoga Falls, OH, U.S.A.) (Figure 19-51) at the gingival crest on either the facial or the lingual/palatal, and attaching elastomeric chain (Figure 19-52) or multiple segments of elastomeric thread to the TMS pin (Figure 19-53). Weekly monitoring is recommended to assess extrusive movement.

When using brackets instead of bonding a wire to adjacent teeth, there is a risk of abutment teeth tipping toward each other. Under certain circumstances, tipping can occur even with a bonded wire. Double abutting the wire will usually prevent this complication (Figure 19-54). As a precaution, frequent checks are required in order to arrest unfavorable movement if detected. In 2 weeks of activation, 3–4 mm of extrusion can be expected.[110] Once fully extruded, an 8-week-stabilization period is required to prevent a potential intrusive relapse.[105]

Histologically, extrusion methods have shown evidence of periodontal fiber stretch, osteoblast activation, and

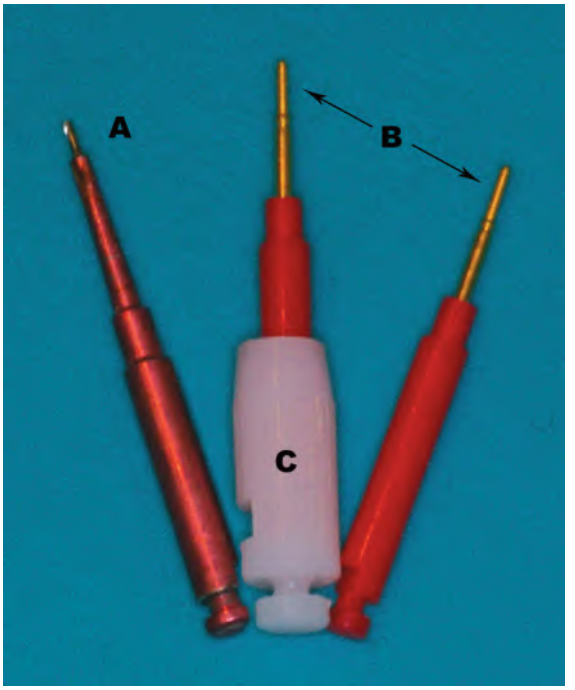

FIGURE 19-51 TMS Link Series. ***A***, Kodex Drill. ***B***, Minikin Single-shear Pins. ***C***, Universal Hand Driver.

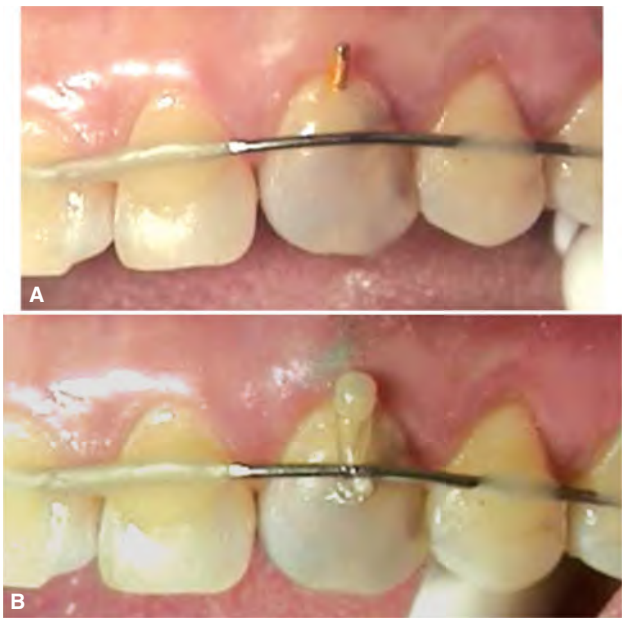

FIGURE 19-52 ***A***, TMS Pin inserted at the gingival crest on the facial. ***B***, Extrusion activation via elastomeric chain stretched over a bonded arch wire. (Courtesy of Dr. Fred A. Berry, Loma Linda, CA.)

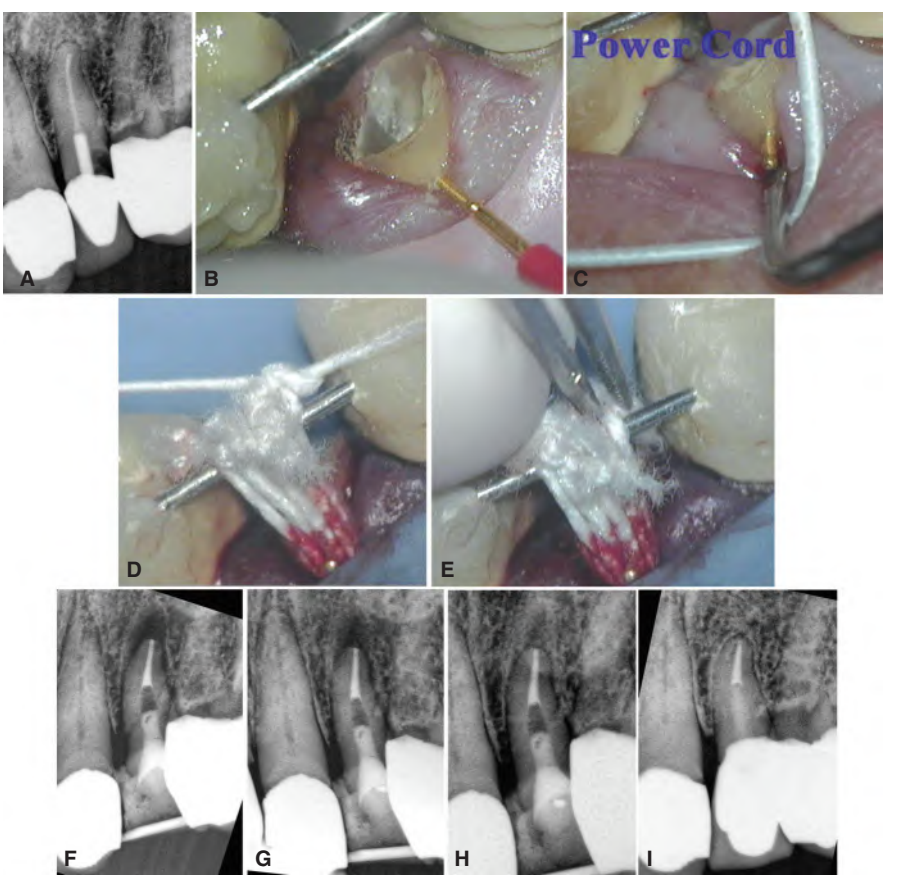

FIGURE 19-53 ***A***, Pretreatment radiograph showing failing restoration with severe coronal-radicular breakdown extending to the post. ***B***, TMS pin inserted at gingival crest on the facial. ***C***, Multiple 5-inch segments of elastic thread looped under the pin. ***D***, Ends triple-knotted over the bonded arch wire. ***E***, Knots rotated out of functional fossa. ***F***, Radiograph shows "extrusion void" and widened PDL space full length on mesial and distal at start of stabilization period. ***G***, Thirty days postextrusion. ***H***, Sixty days postextrusion. ***I***, Four years postextrusion. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

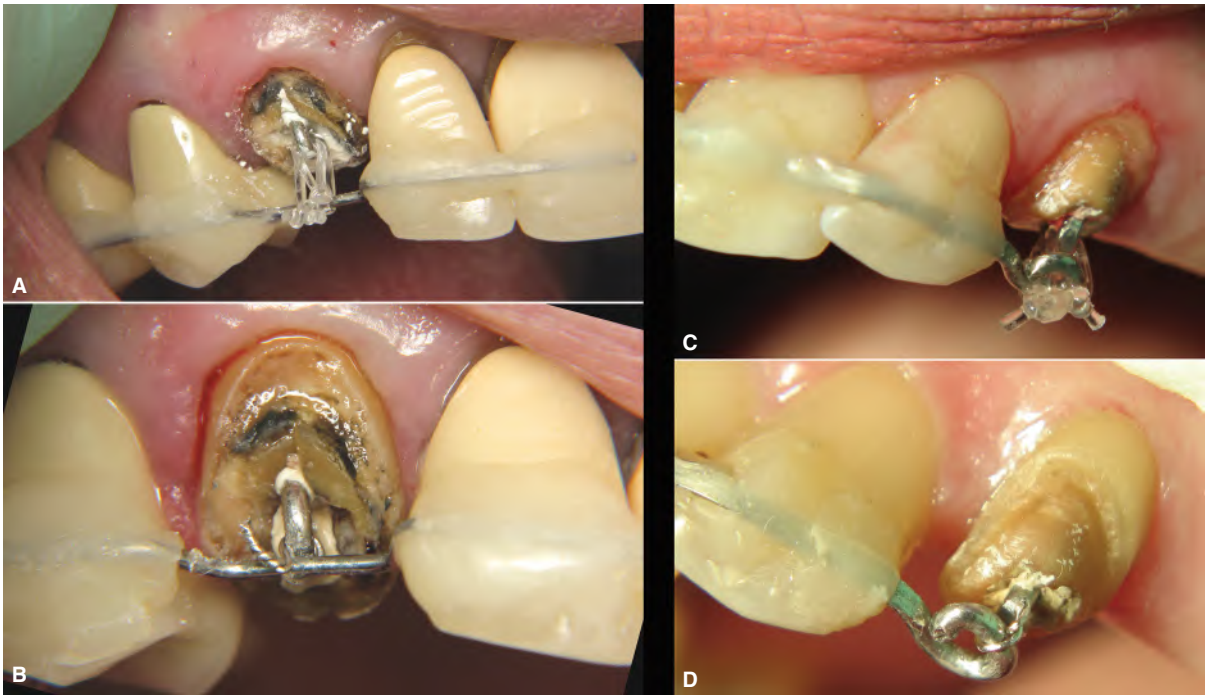

FIGURE 19-54 Rapid extrusion with double abutment to prevent tipping of abutment teeth. Case 1 (left side)—typical double abutment on both sides of target tooth. Initiation, ***A*** and stabilization, ***B***. Case 2 (right side)—cantilever double abutment. Initiation, ***C*** and stabilization, ***D***. (Courtesy of Dr. Fred A. Berry, Loma Linda, CA.)

elevation of crestal bone that accompanies tooth eruption. Osteoblastic activity can be arrested by repeated fibrotomy during or immediately after eruption.[103,111] However, if overwhelming extrusive forces are applied, Sharpey's fibers become disorganized along their intermediate portions. Although the fibers retain their end-point attachments to cementum and bone, they become flaccid, losing their capacity to exert tension on the bone and subsequently fail to promote osteoblastic activity.[101,112] According to Simon,[101] the "mid-portion of the PDL appears disjointed, but the fiber attachments remained intact. Attachments to cementum and bone are not lost. The periodontal ligament apparently takes this movement in stride and eventually reorients the fibers in a normal direction. Apparently, our force was within tolerable limits. Surprisingly, new immature bone is laid down within 2 weeks and mature bone within 4 weeks."[101]

This biomechanical phenomenon differentiates rapid extrusion from typical orthodontic movement that uses light forces over extended time periods. During the 8-week-stabilization period, the fibers reorganize into the ideal perpendicular root-to-bone attachment, and the site is characterized by new immature bone formation at 2 weeks and mature bone deposition within four weeks.[101]

Limitations While teeth with tapered, short single roots are simplest to extrude, certain other root morphologies and conditions can adversely impact the effectiveness of rapid extrusion. In its simplest form, rapid root extrusion does not involve osteoclastic activity as it moves a tapered root out of bone to a new, more coronal position, stimulating only osteoblastic activity, and primarily in the apical and mid portions of the root. Osteoclastic activity, however, is involved under certain conditions, such as (a) when there are dilacerations, (b) when the root surface has bumps or indentations, (c) when there are divergent roots, or (d) when the root is bulbous. In such cases, extrusive activity exerts pressure on the bone at the coronal aspect of the irregularity and tension on the bone at the apical aspect. Lighter extrusive forces should be applied in such cases. In the presence of excessive extrusive force, osteonecrosis can occur on the pressure side, and this can halt the extrusion process until the necrosis resolves and lighter extrusive pressures are applied. Because teeth that are candidates for extrusion often have a history of trauma, spot ankyloses, not easily detected radiographically, can be involved and may impede or even prevent extrusion.

Emergence profile presents another limitation, particularly with regard to maxillary central incisors because they are viewed side-by-side. In select cases, when the mesiodistal aspects of both maxillary central incisors are relatively parallel, extrusion of one incisor may not create an emergence profile discrepancy. If tapered, however, there could be an unacceptable esthetic result, and the greater the amount of both the taper and the extrusion, the greater the discrepancy. Lip line is an important determinant in such cases.

Outcome and prognosis There are important factors to consider during treatment that can influence the outcome of rapid root extrusion procedures. Malmgren et al.[113] concluded that there is a risk of relapse after orthodontic extrusion, and therefore suggested that fibrotomy should be performed prior to the retention period to avoid this complication. It was also demonstrated by Weissman[112] in two case

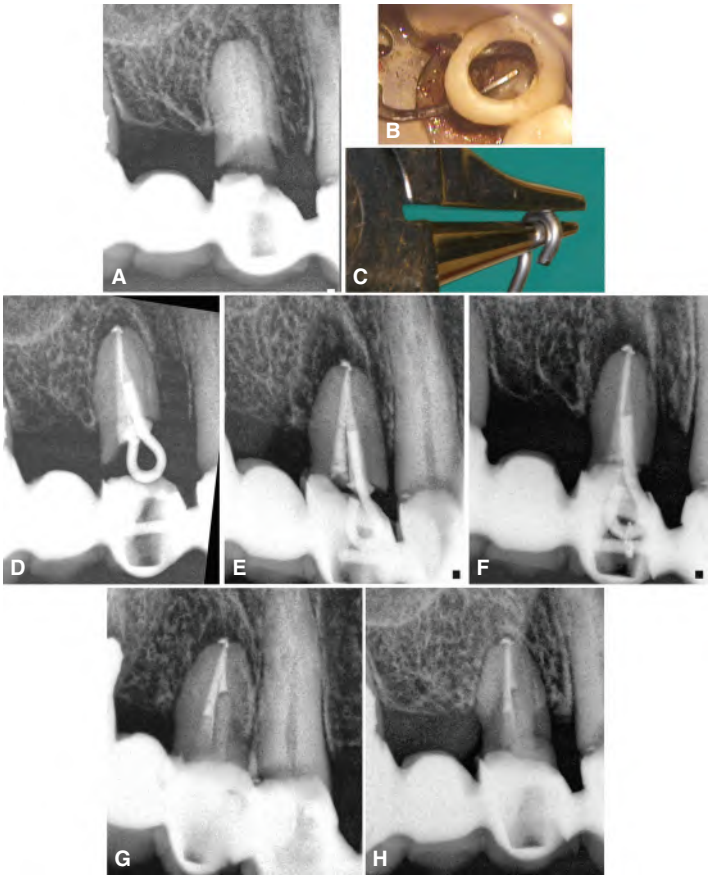

FIGURE 19-55 ***A,B***, Pretreatment radiograph and video frame capture showing the extent of coronal breakdown. ***C***, Hook-post fabricated from stainless steel orthodontic wire. ***D***, Endodontic therapy completed, hook-post cemented and extrusion activated. ***E***, Extrusion completed. Note the enlarged apical void resulting from the extrusion procedure. ***F***, Four weeks of stabilization. ***G***, Eight weeks of stabilization. ***H***, Ten months postextrusion. (Courtesy of Dr. C. John Munce, Santa Barbara, CA.)

reports that immediate gingival fibrotomy before extrusion limits gingival migration. Simon's observations,[101] however, suggest that a primary means of preventing bone from elevating along with the root is the speed with which the root is erupted. Therefore, while selective fibrotomy may be considered as a secondary means of precluding osteoblastic activity in select cases, the extrusion process itself serves to prevent comigration of bone along with the root when the extrusive movement is performed with adequate rapidity.

In a study on dogs by Simon and colleagues,[101] at 2 weeks, extruded teeth showed radiolucent areas surrounding the roots. On histologic examination, these areas were found to contain new immature bone or osteoid and a periodontal ligament of normal width. At 7 weeks, the roots were radiographically within normal limits, and this was confirmed histologically. Figure 19-55 is a compilation of radiographs and clinical images showing the case of rapid extrusion on an 85-year-old patient with severe coronal breakdown on a maxillary right first premolar with radiographic findings demonstrating the sequence of radiographic findings described by Simon and colleagues.

Rapid root extrusion is a treatment option that can be considered for saving compromised teeth exhibiting limited coronal structure or radicular defects at or near the alveolar crest. The goal is to move teeth into a more favorable position to facilitate endodontic isolation and subsequent restoration. This strategy is useful in all tooth groups provided root morphology and other factors are favorable. The technique is critically important in the anterior region because it preserves the natural esthetic contours of the gingiva and papillae. It is a safe and beneficial procedure that predictably retains the natural dentition with minimal risk of complications.

Immediate Surgical Extrusion

Single-rooted teeth requiring endodontic treatment and having simultaneous involvement of the coronal third of their root by caries, fracture, perforation, or resorption can challenge the clinician during the restorative and endodontic phases of treatment. Endodontic treatment demands adequate isolation during treatment and prosthetic rehabilitation demands adequate healthy tooth structure in harmony with the contiguous periodontal soft and hard tissues. In order to reestablish biologic width[114] and an adequate ferrule,[115] various corrective procedures utilized are clinical crown lengthening, orthodontic extrusion, or the forgotten modality of immediate surgical extrusion.[116] Clinical crown lengthening has the limitation of potentially causing collateral damage by removing healthy bone support on adjacent teeth and its effect on esthetics in

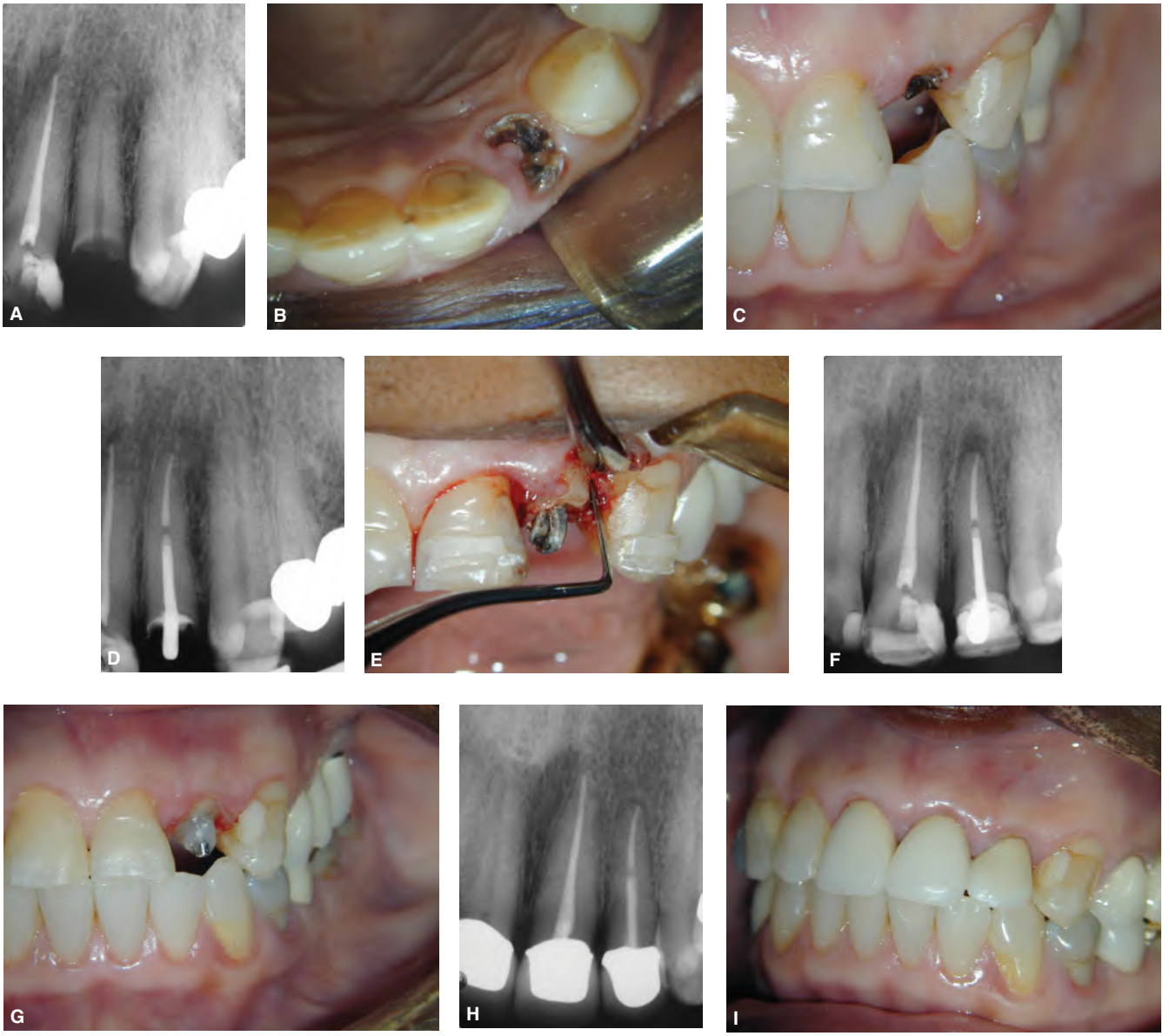

FIGURE 19-56 ***A***, Preoperative radiograph showing carious destruction of maxillary left lateral incisor and its relationship to alveolar crest and adjacent teeth. ***B***, Preoperative clinical view showing compromised tooth structure, occlusal view. ***C***, Preoperative clinical view showing compromised tooth structure, facial view. ***D***, Postoperative radiograph after completion of root canal treatment and internal reinforcement with a temporary post. ***E***, Reflection of an envelope flap and predictable placement of a periotome. ***F***: Immediate postextrusion radiograph demonstrating extent of extrusion. ***G***, Clinical view prior to cementation of crown at 4 months. ***H***, Radiographic follow up after 1 year shows slight periodontal ligament space widening apically. ***I***, Postoperative clinical view showing optimum gingival health and architecture around the permanent crown. (Courtesy of Dr. Rajiv G. Patel, Flower Mound, TX. Adapted with permission from Schwartz RS, Canakapalli V (eds), *Best Practices in Endodontics: A Desk Reference*. Hanover Park, Quintessence; 2015. p. 279.)

the anterior region can be detrimental whereas orthodontic extrusion is time dependent, relying on patient compliance and sometimes the need for additional remedial crown lengthening therapy consequent to mobilization of the tooth.

This section will focus on the modality of immediate surgical extrusion. Immediate surgical extrusion is the intentional and controlled luxation of the root coronally with the objective of stabilizing the root in a favorable restorative position (Figure 19-56).

The information extrapolated from the dental trauma literature with regards to extrusive luxation[117] supports a favorable prognosis with a low incidence of adverse events for the immediate extrusion procedure.

Indications The overall strategic value of the tooth in the comprehensive treatment plan is a prime consideration. Immediate surgical extrusion can be considered in cases where there is involvement of the coronal third of the root by a compromise such as crown root fracture, cervical caries,

root resorption, or iatrogenic perforation. The procedure has an application in selected cases of trauma where an oblique crown root fracture is observed and the rotation of the tooth could position the tooth in a more favorable restorative relationship. Age is an important qualifier for surgical extrusion, especially for young growing individuals with healthy adjacent teeth who are not physically ready for replacement with an implant but esthetics, bone and space, needs to be retained in the interim. On an average, young women complete growth at age 17 years and 8 months and young men at age 20 years.[118]

Clinical considerations With regards to the timing of endodontic intervention, surgical extrusion might be necessary to provide optimum isolation prior to initiation of endodontic treatment. Certain root configurations can benefit from fortification of the root with a temporary composite build up that will prevent any iatrogenic event such as fracture during the application of any luxation forces on the root.

The amount, location and level of healthy tooth structure will determine the crown root ratio anticipated after the extrusion procedure. The level and location of actual tooth structure remaining after caries clean out needs to be noted while making estimations of the desired crown root ratio following the extrusion. A minimum of 1:1 crown root ratio is desirable and will facilitate stability of the tooth in function.[119]

Armamentarium and technique The materials required for the procedure are similar to those necessary for an exploratory surgery. The instruments include Periotomes, forceps, elevator, methylene blue dye, sutures, nylon fishing wire, and composite resin.

The technical procedure involves a full-thickness mucoperiosteal envelope flap extending at least one tooth adjacent to the tooth to be extruded to allow for adequate visualization and atraumatic placement of a periotome. Adequate visualization with the aid of magnification and illumination can minimize the risks associated with the procedure. An atraumatic procedure will minimize trauma to the osseous housing especially in areas with a thin cortical plate. A significant association was found between the type of gingival biotype and a thickness of cortical plate, a thin gingival biotype representing a thin underlying cortical plate.[120]

The periotome is manipulated circumferentially in a rocking crown-down manner with the objective of shearing the attachment apparatus of the root. Light forces with the aid of an elevator might be necessary to facilitate luxation of the root. After sufficient mobility is achieved, the residual healthy root structure is repositioned approximately 4–5 mm above the alveolar crest to allow for establishment of an adequate biologic "height" and ferrule. Methylene blue dye can be used to stain the root surface, highlighting any defects or suspected cracks. The occlusal clearance is verified and a periapical radiograph taken to assess the degree of extrusion. The flap is repositioned and sutured. The tooth is stabilized by using a flexible splint bonded onto adjacent teeth for 2 weeks. The sutures are removed at 48 h. The stability of the tooth is verified at 6 weeks and the root canal treatment completed if it was not completed prior to extrusion. The tooth can be reinforced with a prefabricated post and core or a cast post and core prior to restoration with a crown.

Technical considerations The root geometry, configuration, and density of bone surrounding the root can pose technical challenges during the luxation procedure. The degree of root taper, abrupt curvatures or dilacerations, root divergence or inclination, the type of cross-sectional morphology of the root, root proximity, and the length can all have a consequence on the complexity and risks associated with the surgical extrusion procedure.

The profile of the root being conical in shape, the narrower portion of the root is re-positioned coronally after extrusion. This can create a wider embrasure space. This change in emergence profile, diameter and contour of the root needs to be compensated during the restorative phase, or it can result in compromised esthetics. A high smile line has a potential for exposing the embrasure spaces and can pose an esthetic detriment, whereas a low lip can be an advantage in masking open embrasures.

The type of occlusion can affect the long-term prognosis, for example, parafunction or trauma from occlusion could be the very etiology that resulted in the fracture of a prior restoration. Patients with a collapsed vertical dimension of occlusion may need a more comprehensive treatment plan to restore form and function. There must be sufficient clearance available to extrude the tooth. In case of insufficient clearance alternative therapy such as crown lengthening might be necessary. Periodontally compromised teeth with deep bony pockets and or associated mobility related to the same are poor candidates for surgical extrusion.

Risks The typical risks of resorption, ankylosis, or persistent mobility anticipated with a traumatic luxation are minimal with the controlled procedure of surgical extrusion due to the lack of drying of the periodontal ligament associated with an extended extraoral time and minimal trauma to the attachment apparatus.[121]

A histological study by Kim et al.[122] comparing orthodontic and surgical extrusion in a dog model, confirmed that ankylosis was a minor event in surgical extrusion group and only transient in nature. They also concluded surface and inflammatory resorption were repaired eventually in both groups.

On the hierarchy of the evidence pyramid, this procedure has a reasonably lower level of evidence due to documentation mainly in terms of case series and case reports. In 2014 Elkhadem et al.[123] published a systematic review that involved 8 case series and 11 case reports that included 243 teeth from 226 patients pooled in the study. Most of the case series and case reports were performed between 1978 and 1999. It was found that 30% of cases showed nonprogressive root resorption. Three percent progressive root resorption was observed mainly in teeth with apical surgery and bone

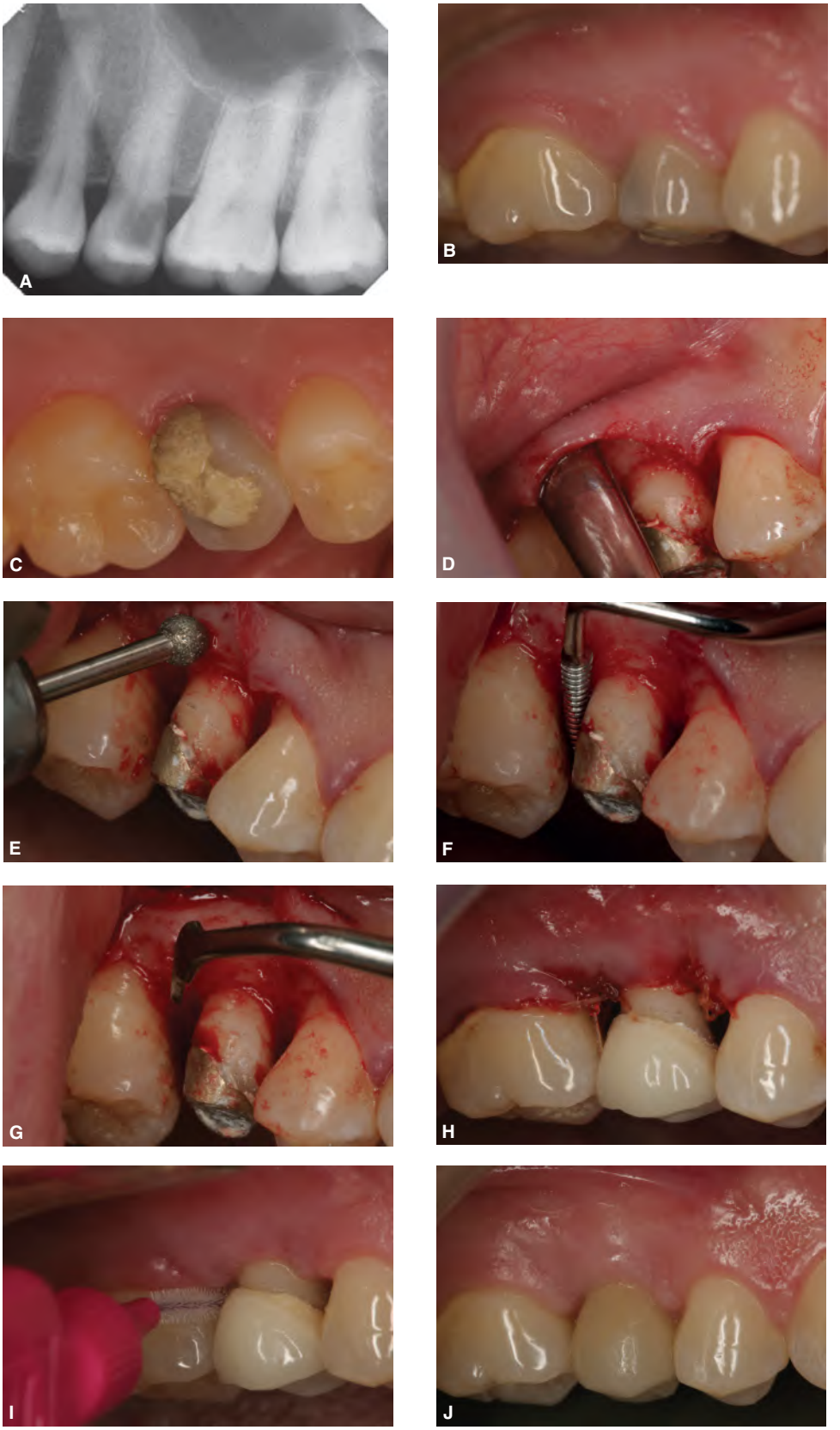

FIGURE 19-57 ***A***, Periapical radiograph showing a large carious lesion on the distal aspect of the maxillary right premolar. ***B***, Preoperative photograph of the affected tooth. ***C***, Occlusal photo showing a large temporary restoration that extends subgingivally. ***D***, A full thickness buccal flap reflection using a periosteal elevator. ***E***, Surgical round bur used for osteoplasty. ***F***, Sugarman file, hand instrument for interproximal bone reduction. ***G***, Hatchet, hand instrument used for osteoplasty. ***H***, Interrupted sutures following the ostectomy and osteoplasty. ***I***, Use of an interproximal brush 1 week postoperatively. ***J***, Final restoration completed 6 weeks postoperatively. (Courtesy of Dr. Ziv Simon, Beverly Hills, CA.)

grafts in studies with the older techniques. No ankylosis was reported in any of the teeth. Five percent of teeth were lost mainly due to repeated trauma.

It appears that surgical extrusion is a valid treatment option in restoratively compromised single-rooted teeth.

Surgical Crown Lengthening

The crown length is defined as the distance between the gingival margin and the most incisal/cuspal aspect of a tooth. Crown lengthening surgery is a procedure intended to increase this distance. It is commonly indicated for the management of deep subgingival caries or fractures, perforations, or resorptive defects extending to or beyond the osseous crest. The procedure improves the ferrule effect and contributes to retention for a predictable, long-lasting restoration with minimal periodontal damage. Crown lengthening can also assist in endodontic tooth isolation when tooth structure is compromised. In the presence of both restorative and endodontic indications, it is an ideal solution for exposing additional tooth structure. The procedure involves resection of soft as well as hard tissues (Figure 19-57). Because impingement of restorative margins in the biologic tissue can lead to irreversible damage in the form of tissue recession and bone loss,[124,125] it is essential to consider this procedure to prevent such undesirable ramifications.

In order to perform an effective crown lengthening procedure, it is necessary to know the dimensions of the attachment mechanism around the involved teeth as well as the commonly acceptable restorative principles (such as retention and ferrule effect). A ferrule effect is defined as "a 360°-metal collar of the crown surrounding the parallel walls of the dentin, extending coronally to the shoulder of the preparation."[115] Restoratively, 1—2 mm of healthy tooth structure apical to a build-up is required for the ferrule effect. Additionally, a dimension of about 2 mm of biologic width is necessary to accommodate the connective tissue fibers and junctional epithelium.[114,126,127] Adding both the ferrule effect dimension and the biologic width dimension, it can be concluded that exposure of 3–4 mm of sound tooth structure coronally to bone is necessary for a successful crown lengthening procedure. Restoratively, it is also important to consider the sulcus depth that is variable in different locations along the circumference of teeth. Should subgingival restorative margins be planned, additional crown lengthening may be necessary, depending on the desired margin location and the particular sulcus depth.

Prior to recommending crown lengthening, tooth restorability should be assessed. Additionally, risks and benefits as well as treatment alternatives should be discussed with the patient. The procedure entails tissue resection by design and therefore is contraindicated if the esthetics will be compromised. An alternative to surgical crown lengthening in the esthetic zone is orthodontic extrusion that does not necessitate tissue resection and also preserves esthetics. Preoperative radiographic and periodontal evaluation is crucial to ensure an excellent prognosis following crown lengthening. The surgery reduces the bony support and should not be performed on mobile teeth. If a crown lengthening procedure is not performed, the possible consequences should be discussed with the patient. These include recurrent caries, the inability to obtain proper impressions, and difficulty in removal of cement or bonding material from restorative margins. Periodontal complications due to limited hygiene access and tissue impingement could result in recurrent gingival inflammation and subsequent bone loss, not only on the restored tooth, but adjacent teeth as well.

Crown lengthening performed to accommodate a clamp and dental dam for endodontic isolation will generally provide adequate tooth exposure for restorative purposes. It is important to convey the necessity of the procedure also from an endodontic perspective and reiterate the need for restorative treatment due to anticipated changes in the gingival margin (i.e., an expected side effect of recession).

Crown lengthening surgery involves three main steps that are of equal importance: soft tissue excision, resection of supporting bone (ostectomy), and resection of nonsupporting bone (osteoplasty). The extent of each component depends upon the circumstances of the particular case.

Incision and flap design Tissue quality needs to be assessed prior to the procedure, and the amount of keratinized and attached gingiva should be measured. For teeth that present with an abundance of attached and keratinized tissue, it is acceptable to resect 1–2 mm of tissue as part of the procedure. The incision type will be an internal bevel incision (also termed a reverse bevel incision) that creates a discard of tissue. In order to perform this incision, the blade is positioned at an acute angle to the tooth and bone. The incision is full thickness, through the periosteum to the hard bone and tooth structure in order to ensure a complete severing of tissue to be excised. For teeth that present with minimal to no attached and keratinized tissue, an intrasulcular incision design is preferred. The latter preserves the maximal amount of tissue and will prevent the formation of a mucogingival defect and further periodontal problems.

A blunt dissection with a periosteal elevator is the method of reflection, and the lateral extent depends upon the number of teeth requiring treatment. For example, if crown lengthening one tooth, a full thickness flap reflection is recommended for both of the adjacent teeth. The flap is reflected in an apical direction, beyond the mucogingival junction, to ensure proper mobilization and protection of the soft tissue during osseous recontouring.

Two types of osseous recontouring Upon reflection of a full-thickness flap, the supporting alveolar bone may be assessed. The particular morphology (i.e., bone excess or deficiency) as well as its vertical position will dictate the amount and pattern of osseous recontouring necessary. This osseous recontouring component of the procedure is critical, as it will dictate the healing of the overlying soft tissue. As noted previously, 3–4 mm of sound tooth structure coronal to the alveolar crest should be exposed. This accounts for 1–2 mm of the ferrule effect dimension and 2 mm for the biologic width dimension. Vertical bone reduction should satisfy these requirements. This vertical bone reduction, or ostectomy, is the removal of supporting bone, and constitutes

the first type of osseous recontouring in a crown lengthening procedure. It is accomplished using surgical burs and hand instruments (hoes, chisels, and files). The main bur for this purpose is an end-cutting bur that is atraumatic to the mesial and distal aspects of adjacent teeth. It reduces bone in the vertical dimension and thus creates a step in the bone. This step must then be eliminated with a round diamond bur and hand instruments. The soft tissues will heal based on the new bone height that was created and the biologic width will reform itself in a more apical position. An alternative method to reduce bone height is by thinning the coronal bone using a round diamond bur and carefully removing the resultant thin shell of bone using hand instruments. Regardless of the technique, it is prudent to avoid contact with the root surfaces in order to prevent irreversible tooth damage.

Osteoplasty, the second type of osseous recontouring in a crown lengthening procedure, is performed after the ostectomy is completed, and involves the removal of nonsupporting bone. It is meant to reshape the osseous architecture around teeth and to facilitate both soft tissue healing and proper flap approximation. Excessive bone structures such as tori and exostoses should be recontoured at this time for the same reasons.

Suturing Primary intention closure is not always possible in functional crown lengthening. This is due to tissue resection in the interproximal areas as part of the flap design and tissue discard. It is therefore anticipated that, in certain circumstances, the interproximal areas will heal by secondary intention with very few complications or side effects. For crown lengthening procedures, it is often sufficient to apply simple interrupted or "figure 8" sutures for flap stability. Despite many different and elaborate suturing techniques, the osseous architecture on which the flap will lie is a critical determinant in the success of the procedure. Postsurgical care typically requires minimal use of analgesics or antibiotics as postoperative pain is generally minor and infections are rare (approximately 2%).[128] Patients should be instructed in careful oral hygiene methods and wound care for 2 weeks. Complete healing can be expected in approximately 6 weeks after which definitive restorative treatment may be initiated.

Summary Crown lengthening is a resective periodontal procedure generally intended to expose tooth structure for restorative purposes but it can also assist in endodontic isolation. With proper planning and case selection, followed by soft tissue resection (ostectomy and osteoplasty), clinicians can achieve successful results with predictable periodontal, endodontic, and restorative outcomes.

ELECTRONIC APEX LOCATORS

The Apical Anatomy

Although it remains a matter of controversy, some researchers have advocated that root canal instrumentation should extend from the canal orifice coronally to the dentin-cementum junction apically,[129–131] while others hold that, in order to remove all diseased tissues, treatment should extend to a point more well-defined and consistently measurable than the dentin-cementum junction.[132] The dentin-cementum junction is often the point of the narrowest diameter in the root, forming the apical constriction. The location of the apical constriction can be up to 1 mm or more from the apical foramen.[133] The anatomy of the apical constriction varies among different teeth and even within the same tooth through the course of aging.[130,134] Kuttler recorded an increase in the distance between the apical constriction and the vertex of the root in aged teeth between 0.5 and 0.6 mm.[130] This distance was later used as a measurement to subtract from the radiographic apex to roughly determine the location of the apical constriction.[135]

Despite ongoing debate regarding the exact apical extent of endodontic treatment,[136] there is broad consensus regarding the fact that as much of the canal system as possible should be treated. Used in conjunction with multiangled radiographs, and three-dimensional imaging when necessary, apex locators assist the clinician in being more accurate in determining the apical extent of treatment.[137]

Limitations of the 2-Dimensional Radiographic Methods

For decades, periapical radiographs have been used to determine the root canal length. Despite the advancement in digital radiography and the ability to estimate the tooth length by direct measuring, digital radiography is not universally accepted as superior to conventional radiographs for endodontic length determination.[138–142] Periapical radiographs are susceptible to elongation, shortening, and added magnification.[143] Interpretation of the position of the file in relation to the radiographic apex can often be challenging even when utilizing the bisecting angle technique.[144,145] Root variations and superimpositions of anatomical landmarks can result in error when determining the working length (Figure 19-58). Periapical radiographs

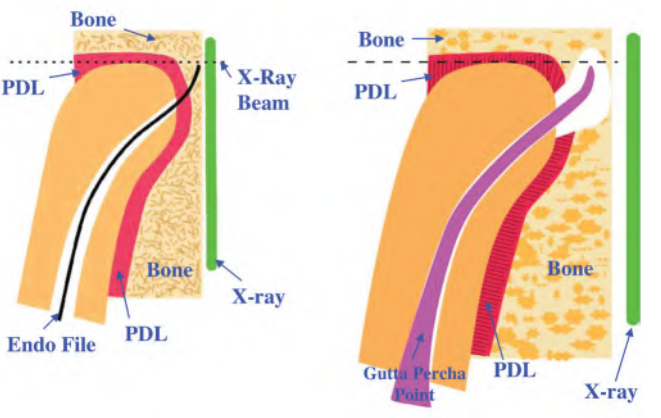

FIGURE 19-58 An illustration showing how endodontic files and gutta-percha can be extruded beyond the apical foramen and appear superimposed on the radiograph apex. (Courtesy of Dr. Allan S. Deutsch, New York, NY.)

present a two-dimensional image of a three-dimensional object and the working length from periapical radiographs can vary from the actual working length. Interpretation of the file position using radiographs has also been shown to vary among clinicians with a decrease in agreement when the file is further away from the radiographic apex.[146] Considering the inaccuracy of the periapical radiographs together with the increased radiographic exposure concerns, the electronic apex locator (EAL) became an acceptable adjunctive method for determining canal length.

Electronic Apex Locator Uses and Designs

EALs are devices used in root canal therapy to determine the position of the apical foramen and thus determine the length of the root canal space. The concept of measuring the canal length electronically was proposed in 1918 by Cluster.[147] The principle design and development of EALs was later reported by Suzuki in 1942.[148] He found that electrical resistance between the periodontium and oral mucous membrane in dogs was of a constant value. Based on these findings, Sunada introduced the first EAL in 1962.[149] He showed that with the use of a direct current, the location of the apical foramen could be estimated since the resistance between the periodontium and the oral mucous membrane was constant at 6.5 KΩ. Since then, EALs have passed through major improvements resulting in more accurate, consistent, and reproducible measurements. EALs have gained popularity among clinicians and are widely used for working length determination. In a recent survey, 77% of the general dentists in the United States used the EAL to determine the root length during root canal procedures.[150] They are useful in situations where anatomic structures are superimposed over apices or when acquiring radiographs is challenging as in the case of handicapped patients, pregnant women, sedated patients, young patients or those with extreme gag reflex. EALs are also capable of differentiating between the canal system and root perforations regardless of the perforation size.[151–153]

Fouad and Reid[154] showed that the use of EAL among dental students resulted in better length control of obturation material and reduction in radiographic exposure. Multiple in vitro studies have shown that EALs are very accurate in determining the apical foramen to within +/–0.5 mm up to 95% of the time.[155–157] Several clinical studies have shown that the effectiveness of EALs varies between 81.5% and 97% for the intervals previously determined in relation to the radiographic apex.[158–160] In a randomized clinical trial that included 188 roots, the accuracy of EALs superseded those of radiograph, 90.4% versus 82.1%.[160] Hassanien et al.[161] compared the accuracy of EALs to radiographs in determining the apical foramen. In 20 patients with teeth scheduled for extraction, canal length was determined by EAL in 10 patients and radiographically in 10 patients. In the EAL group, a file was inserted until the apex locator indicated "apex," and it was then cemented into place. In 10 patients, the termination point was determined radiographically with a file in place. After the radiographic apex was located, 0.5 mm was subtracted, and the file was positioned to this point in all 20 teeth. The teeth were extracted, and the buccal wall of the root at its apical 4–5 mm was trimmed to gradually expose the inserted file and the apical anatomy. The file tip was located, on average, 0.21 mm from the apical foramen when EALs were used, whereas by radiographic determination, it was 0.56 mm. The difference was statistically significant.

Despite their relative effectiveness, studies have shown that the accuracy of EALs may be affected by immature apices, retreatment, calcified or blocked canals as well as severe hemorrhage and inflammatory exudate into the canals.[162–165] The accuracy of EALs may also decrease as the size of the apical diameter increases.[166] For best results, a master cone radiographic control should be taken to minimize possible errors.[167] EAL reading can also be affected by contact between the file and metallic restorations and materials.[168] This can be overcome by drying the pulp chamber, leaning the file away from the restoration or isolating the file with a section of a plastic capillary tip (Figure 19-59).

In the past, it was recommended by EAL manufacturers that these devices should not be used in patients with pacemakers. Garafalo et al.[169] suggested that EALs may be used with caution and only after consultation with the patient's cardiologist. This concern, however, proved to be unnecessary. All modern pacemakers are shielded in hermetically sealed metal cases with capacitors that filter out electromagnetic interference signals.[170,171] In a clinical study, Wilson et al.[172] showed that there was no interference between EALs and the functionality of pacemakers or cardioverter/defibrillators. In a recent bench-top study, different EALs were directly connected to a pacemaker and demonstrated no interference with the functionality.[173] Nevertheless, some manufacturers still warn clinicians not to use EALs in patients with pacemakers.

Since their development, EALs have been evolving in an attempt to improve accuracy and reproducibility of measuring the root canal length in various clinical conditions. Currently, EALs can be divided into six generations:

First Generation

The first generation of EALs, also known as resistance EALs, measure opposition to the flow of direct current or resistance. The first device, The Root Canal Meter (Onuki Medical Co., Tokyo, Japan), was developed in 1969. Pain was commonly felt while using this device due to the high current. The device was later improved and released as the Endodontic Meter and the Endodontic Meter SII that used lower currents of less than 5 μA.[174] Other devices in this generation included Sono Explorer (Salatec) Neosono-D, MC, and Ultima EZ (Amadent) Apex Finder (EIE—old version). These first generation devices caused sensitivity during use, required a dry environment and files that had a snug fit in the canals. They were also contraindicated in patients with pacemakers. Ultimately, they proved unreliable

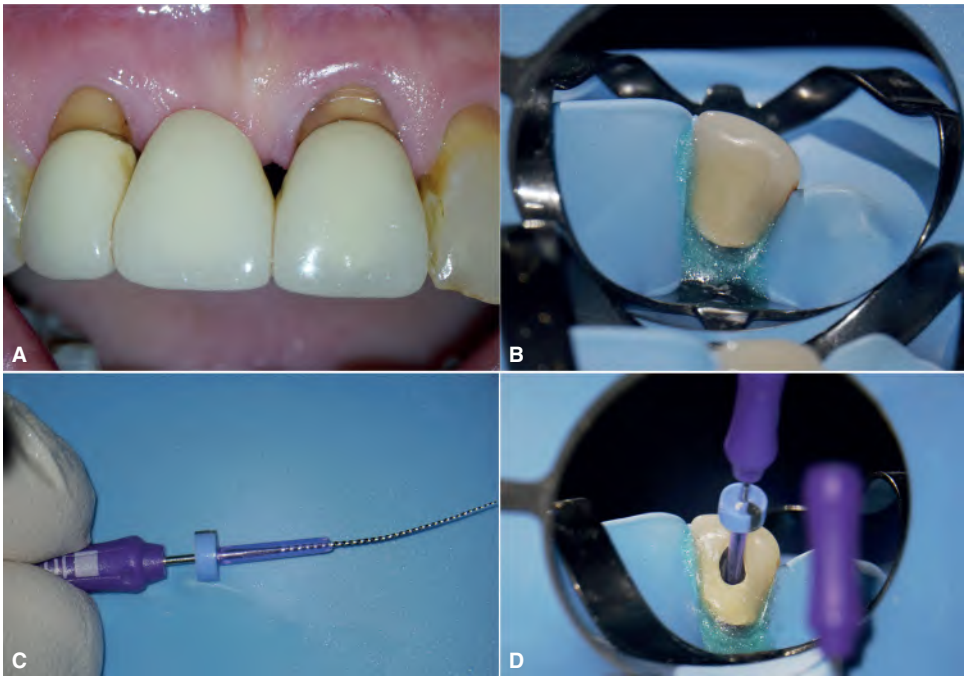

FIGURE 19-59 ***A***, EAL accuracy will be impacted by the porcelain fused to metal (PFM) crown; ***B***, target tooth is isolated; ***C***, use of a segment of a plastic capillary tip (Ultradent, South Jordan, UT) to insulate endodontic file; ***D***, EAL working length determination. (Courtesy of Dr. Carlos Aznar Portoles, Amsterdam, the Netherlands.)

when compared with periapical radiographs, as many of the readings were inaccurate.[175]

Second Generation

Second generation EALs, also known as impedance EALs, measure opposition to the flow of alternating current or impedance. These devices operate on the principle that there is electrical impedance across the canal walls. The tooth exhibits variable electrical impedance across the walls of the root that is greater apically than coronally. This method was introduced in 1971 as the Sono-Explorer (Hayashi Dental Supply, Tokyo, Japan). The second-generation EALs used single high frequency impedance measurements instead of resistance to measure locations within the canal. These second generation devices produced measurements that were inaccurate and required calibration at the periodontal pocket of each tooth. Some models, such as the Apex Finder and the Endo Analyzer (Analytic, Sybron Dental, Orange, CA, U.S.A.) were self-calibrating but also delivered varying degrees of accuracy.

Third Generation

Third generation EALs were similar to second generation but used multiple frequency rather than single frequency impedance, and the first one commercially available was the Apit, also known as Endex (Osada Electric Co, Tokyo, Japan). This device worked by comparing the difference in impedances using the relative value of two alternating currents at frequencies of 1 kHz and 5 kHz. As the file advanced apically, the difference increased until it reached its maximum value at the apical constriction. With their powerful microprocessors they were able to handle mathematical quotient and algorithm calculations to provide accurate readings. The Endex was still difficult to operate due to the need for calibration. In 1991, Kobayashi[176] introduced the ratio method that allows two electric currents with different sine wave frequencies to have measurable impedance that can be compared as a ratio. In 1994, the first Root ZX EAL (J Morita, Tokyo, Japan) was launched. The ratio method allowed the Root ZX to self-calibrate by measuring the impedances of 0.4 kHz and 8 kHz at the same time, calculate the quotient of the impedances, and express this quotient in terms of the position of the file inside the canal. This allowed more accurate reading regardless of the type of electrolyte in the canal. Other third generation EALs included the Endo Analyzer Model 8005 (Analytic, Sybron Dental, Orange, CA, U.S.A.) that used five different frequencies (0.5 kHz, 1 kHz, 2 kHz, 4 kHz and 8 kHz).

Fourth Generation

This generation EALs also used multiple frequencies, but only one frequency at a time yielding increased accuracy.[177] In 2003, the Elements Diagnostic Unit and EAL (SybronEndo, Anaheim, CA, U.S.A.) was introduced to the market and claimed by the manufacturer to provide more accurate measurements by comparing the resistance and capacitance measurements to a stored database to determine the distance to the apex of the root canal.[178,179] It used a composite waveform of two signals, 0.5 kHz and 4 kHz, instead of 8 kHz and 0.4 kHz used with the Root ZX. A significant disadvantage of the fourth generation was the requirement of dry or semi-dry canals that rendered it impractical in a variety of clinical conditions.[180,181]

Fifth Generation

This generation EALs were developed in 2003. They also measure the capacitance and resistance of the circuit separately as did the fourth generation EALs, but they were able to overcome disadvantages inherent in the fourth generation devices. Nevertheless, these devices had considerable difficulties operating in dry canals,[180] and they also had measurement reliability factors that varied with pulpal and periapical conditions.[182]

Sixth Generation

The Adaptive Apex Locator is claimed to be a sixth generation EAL with the ability to overcome the problems associated with the fourth and fifth generation devices. This device functions in dry or wet canals by continuously defining the canal humidity via mathematical analysis and algorithm to adapt according to the moisture characteristics of the canal to accurately determine canal length.[180]

EAL Benefits

EALs can accurately measure canal length and may perform better than radiography alone. When combined with canal length radiographs and master cone radiographs, the patient benefits from reduced errors and reduced radiation exposure when determining the working length.[183]

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Chapter 20

Preparation of the Coronal and Radicular Spaces

Michael Hülsmann, Edgar Schäfer

INTRODUCTION

Root canal preparation is frequently considered the most important and challenging step during root canal treatment.[1–3] It involves using mechanical and chemical means to remove pulpal tissue, reduce microbial load, and enable appropriate obturation of the radicular spaces. Mechanical preparation can be divided into six main parts:

- Preparation of the coronal access cavity
- Preparation of the pulp chamber
- Identification of root canal orifices
- Preparation of a secondary access to the root canals and coronal flaring
- Preparation of a glide path to the apical foramen
- Preparation of the middle and apical parts of the root canal.

HISTORICAL DEVELOPMENT

In his 1733 book, "Le chirurgien dentiste," Pierre Fauchard described instruments for root canal preparation. The basic treatment technique at that time was cauterization of the pulp with heated instruments. At the end of the 18th century, only primitive hand instruments and excavators, some iron cauter instruments and very few thin and flexible instruments for endodontic treatment were available. In the 19th century, enlargement of root canals was performed with broaches. In 1885, the Gates Glidden drill was introduced, and in 1904, the K-file was developed by the Kerr Company. Although Trebitsch in 1929 and Ingle in 1958 suggested standardizing the shape of endodontic instruments, it was not until 1974 that the ISO-specifications for endodontic instruments were published. In 1961, Dr. Ingle presented a detailed technique for preparation of root canals, the "standardized technique."[4]

Oltramare, in 1892, described the first rotary system for root canal preparation. Fine needles with a rectangular cross-section were mounted onto a dental handpiece, passively introduced into the root canal upto the apical foramen and then rotated. Interestingly, in 1889, William H. Rollins had designed an endodontic handpiece rotating at low speed of approximately 100 rpm and with a 360° rotation, similar to the motors used today for canal preparation with NiTi instruments. In the first half of the 20th century, techniques such as iontophoresis, using electrical current, gained some popularity.

During the 20th century, several motors and handpieces for automated root canal preparation appeared in the market. In 1928, the "Cursor filing contra-angle" (W&H, Bürmoos, Austria) was introduced combining rotational and vertical motions of the file. This handpiece was followed by the Racer-handpiece (W&H, Bürmoos Austria) in 1958 and the Giromatic (MicroMega, Besançon, France) in 1964, operating with reciprocal motions (90° clockwise, 90° counterclockwise)[5] (Figure 20-1).

The Canal Finder System (CFS) (SET, Gröbenzell, Germany), designed in 1984, represented the first endodontic handpiece with a partially flexible motion. The amplitude of the vertical file motion depended on the rotary speed and the resistance of the file inside the root canal. It changed into a 90° rotational motion with increasing resistance. For the first time, the individual root canal anatomy had some impact on the motion of the instruments. The CFS was later used with integrated permanent irrigation (i.e., sodium hypochlorite) and electrical control of working length, features also found in more modern endodontic systems.

An important advance was made by the introduction of ultrasonics and sonics systems by Richman in 1957. It was Martin and Cunningham, however, who made ultrasonic devices popular in the 1970s. The first ultrasonic device was marketed in 1980 and the first sonic device in 1984.

Electrosurgical devices (Endox, Lysis, Munich, Germany) and non-instrumental technique (NIT) by Lussi, using a vacuum pump for cleaning and filling of root canals seemed to be promising, for some time. The NIT was designed to clean the root canal system with intermitted positive and negative pressure and without any instrumentation or removal of root dentin. These techniques did not gain popularity or broader acceptance despite the innovative ideas.[4]

Instruments made from nickel-titanium (NiTi) were first described in 1988. Motors with a 360° rotation or with a reciprocal motion (already developed decades before), became popular again, making the use of NiTi more efficient. In 2010, the SAF system was introduced.

AIMS OF MECHANICAL ROOT CANAL PREPARATION

The aims of root canal preparation, also termed as enlargement, shaping, or instrumentation of the root canal, have been outlined by Herbert Schilder in 1974.[1] In his paper on root canal preparation "Cleaning and shaping the root canal

FIGURE 20-1 ***A***. Endocursor and ***B***. the Racer handpiece were among the first automated devices for root canal preparation.

system" Schilder suggested to define five mechanical and five biological objectives for root canal preparation.

The **mechanical objectives** are:

I. Continuously tapering funnel from the apex to the access cavity.
II. Cross-sectional diameter should be narrower at every point apically.
III. The root canal preparation should flow with the shape of the original canal.
IV. The apical foramen should remain in its original position.
V. The apical foramen should be kept as small as practical (Figure 20-2).

The **biologic objectives** are:

I. Confinement of instrumentation to the roots themselves.
II. Not forcing necrotic debris beyond the apical foramen.
III. Removal of all tissue and debris from the root canal space.
IV. Creation of sufficient space for intracanal medicaments.
V. Completion of preparation in one appointment.

Some of these objectives are still valid more than 40 years later.

The main goals of root canal preparation are as follows:

- Removal of all vital and necrotic pulp tissue.
- Elimination, or substantial reduction, of microorganisms and microbial biofilm (see Chapter 21, Irrigants and Intracanal Medicaments).
- Neutralization of microbial toxins and byproducts in the affected dentin.
- Creation of space for obturation.
- Preservation of the integrity and location of the apical canal anatomy.
- Avoidance of iatrogenic damage to the canal system and root structures.
- Avoidance of further irritation and/or infection of the periradicular tissues.
- Preservation of sound root dentin to allow long-term function of the tooth.

These goals can be achieved by using several techniques of root canal preparation including (a) manual preparation with different instruments; (b) automated preparation with conventional stainless steel instruments; (c) rotary systems using NiTi-instruments; (d) sonic and ultrasonic preparation; (e) lasers; and (f) non-instrumental techniques.[2,3,6]

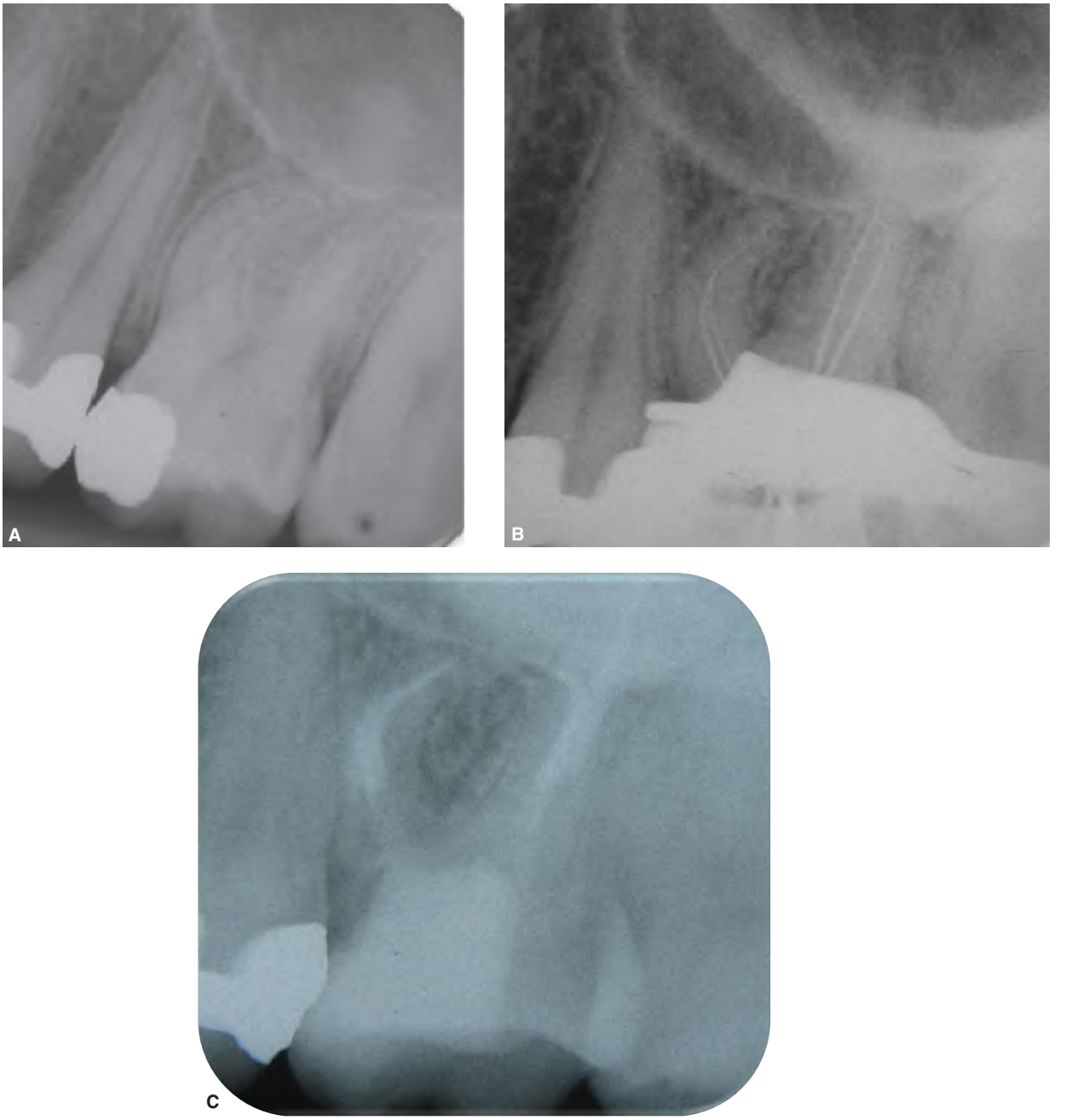


FIGURE 20-2 ***A-C.*** Preparation of these severely curved root canals demonstrates a good flow of the preparation following the original axis of the four root canals without radiographically visible deviation, straightening, or apical transportation. Note complete severity of the curves becomes evident only when the length determination radiograph is taken.

PLANNING FOR ROOT CANAL PREPARATION

No root canal resembles another. Consequently, each root canal has to be addressed individually. Several factors must be considered when planning and performing root canal preparation. These include anatomy, microbiology, and the instruments available for preparation.

Basic principles for canal preparation:

- Each root canal has to be addressed individually.
- Root canal preparation should be planned thoroughly before starting treatment.
- Careful evaluation of radiographs is mandatory.

Root Canal Anatomy

The complex anatomical structures of the root and root canal system make root canal preparation a challenging task. For more details, see Chapter 1, Anatomy and Morphology of Teeth and Their Root Canal Systems. The number of canals, curvature and diameter, cross-sectional shape, lateral and accessory canals and ramifications, isthmi and communications between the root canal and the periodontal tissues, and the furcation area have to be addressed by preparing an adequate access cavity, instrumentation and disinfection (Figure 20-3).

The nature of canal curvatures in terms of radius and angle is of importance for proper selection of instruments (size, taper, flexibility, risk of fracture). The nature of a curve is three-dimensional, with only two planes of the curve visible in the radiograph. It has been demonstrated that in certain cases, three-dimensional imaging using cone-beam computed tomography (CBCT) may be helpful to identify anatomical intricacies, apical pathosis and intraoperative complications.[7] For more details, see Chapters 9C (Cone Beam Computed Tomography) and 10 (Imaging Interpretation).

Microbiology

In case of pulp necrosis, the pulp tissue and root dentin will harbour microorganisms, many in a biofilm form, as well as microbial toxins.[8-10] Adequate preparation will significantly reduce the number of microorganisms. For more details, see Chapter 3, Microbial and Nonmicrobial Etiologies of Endodontic Disease.

Instruments

Mechanical preparation alone, even when using highly flexible instruments, is not sufficient to render a disinfected root

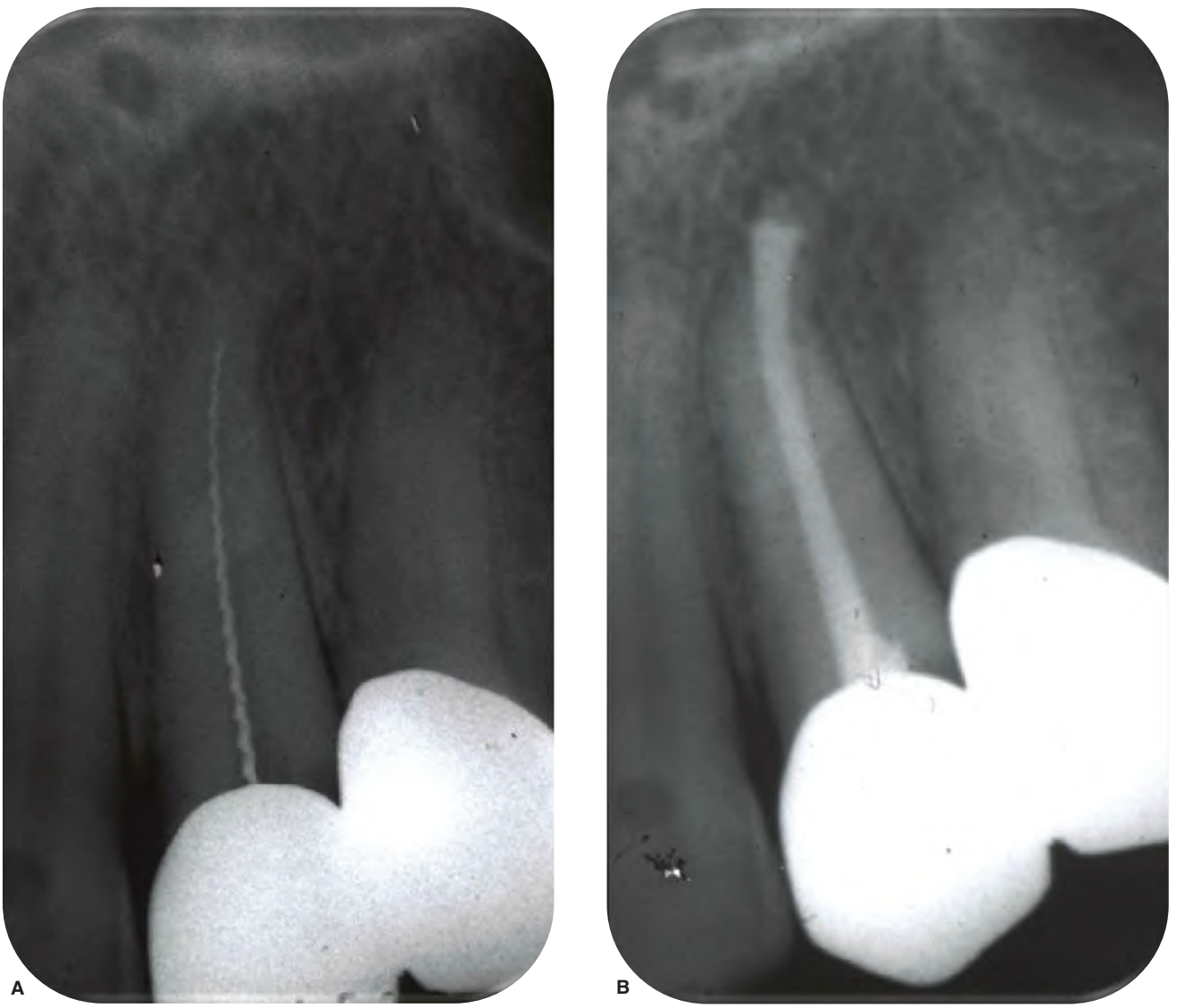


FIGURE 20-3 ***A.*** Underestimation of the curvature has resulted in severe straightening and apical transportation. ***B.*** This is mainly due to improper evaluation of the radiograph and inadequate treatment planning.

canal system. Most endodontic instruments are designed to prepare a round root canal. This may inevitably be associated with under-preparation at one part of the root canal system, leaving behind tissue debris and microorganisms, and over-preparation at the other part, resulting in excessive and unnecessary removal of dentin and possibly weakening the root. Therefore, chemical irrigation should always be done in tandem with mechanical preparation. For more details, see Chapter 21, Irrigants and Intracanal Medicaments.

The final factor influencing the performance of any instrument is the clinician's ability, routine, and skill in steering and guiding the instrument inside a narrow and tricky confines of a root canal. It has been demonstrated that the influence of the operator's skills on the result of instrumentation in terms of shape, working time and number of procedural errors is higher when using stainless steel instruments and lower when using rotary NiTi instruments.[2,3,6]

CRITERIA FOR EVALUATION OF INSTRUMENTS AND TECHNIQUES FOR ROOT CANAL PREPARATION

When evaluating the advantages and disadvantages of any instrument or preparation technique the following points should be considered[2]:

Cleaning Ability

Endodontic instruments are basically designed to cut and remove dentin and create space for effective irrigation. Cleaning of a root canal can be assessed by scoring the treated area for the amount of remaining smear layer and debris, using scanning electron microscopy. It can also be assessed by counting the number of colony forming units (CFUs) of microorganisms remaining after preparation and/or disinfection. Both methods show some specific shortcomings.[2]

Shaping Ability

The shape of the prepared root canal should show a constant taper that flows with the shape of the original canal without transportation of the axis or the foramen.[2] A round diameter of the prepared root canal is essential as long as single-cone obturation techniques (gutta-percha or silver cones) are used. When using cold or warm compaction techniques, the diameter of the root canal seems to be less important, hence, the primary objectives should be to instrument and prepare the canal circumferentially.

In curved canals the continuous taper has to extend to the apical part of the canal to avoid apical straightening and mishaps such as elbow-, teardrop-, and zip-configurations.[2,3]

Shaping ability of root canal instruments and preparation techniques have been well studied. Traditionally, radiographic and microscopic techniques have aided investigations. Lately, micro-CT was introduced allowing an excellent non-destructive visualization of the pre- and post-operative root canal shape and exact qualitative and quantitative measurement of the alterations resulting from preparation.[7]

Work-Related Mishaps and Safety

The most common work-related mishaps are:

- Fracture of instruments.
- Ledging.
- Apical blockage.
- Loss of control of working length during preparation.
- Over-instrumentation.
- Apical extrusion of debris.
- Development of dentinal cracks.
- Heat damage.
- Perforation.

ANATOMY AND PRIMARY ACCESS CAVITY

Preparation of the root canal system starts with preparation of a properly designed access cavity.[11]

The access cavity should:

- Allow full control of the pulp chamber (Figure 20-4).
- Allow removal of all tissue from the pulp chamber and proper disinfection. This will result in elimination of a significant load of microorganisms.

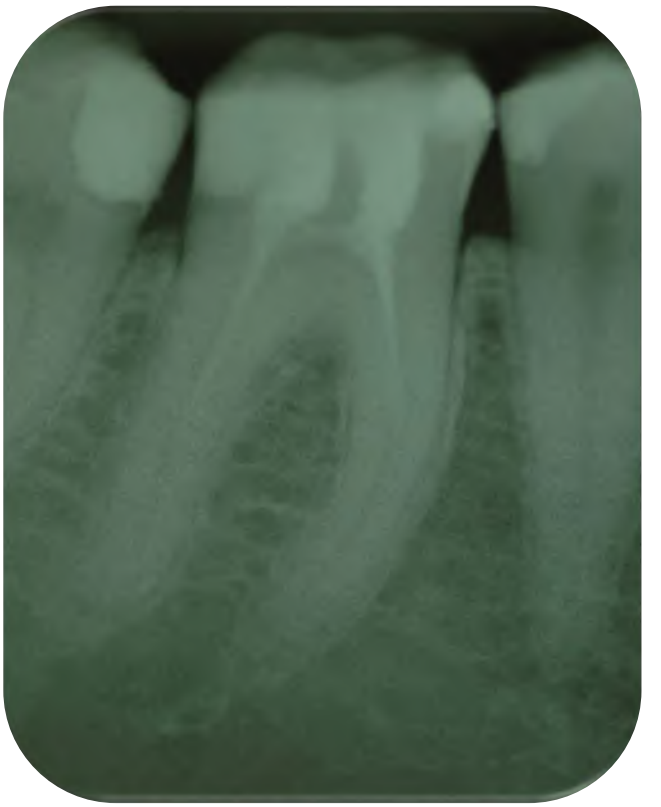


FIGURE 20-4 Incomplete removal of the roof of the pulp chamber resulting in insufficient root canal preparation. At least one root canal has not been detected.

- Allow a proper intracoronal diagnosis (cracks, dentin colors, pulp stones, calcifications).
- Allow location of all root canal systems and relevant anatomical structures (isthmi, lateral extensions) (Figure 20-5).
- Allow fast and straight-line introduction of endodontic instruments without coronal interferences. Ideally, the straight-line access should be extended to the beginning of the curvature (Figure 20-6).

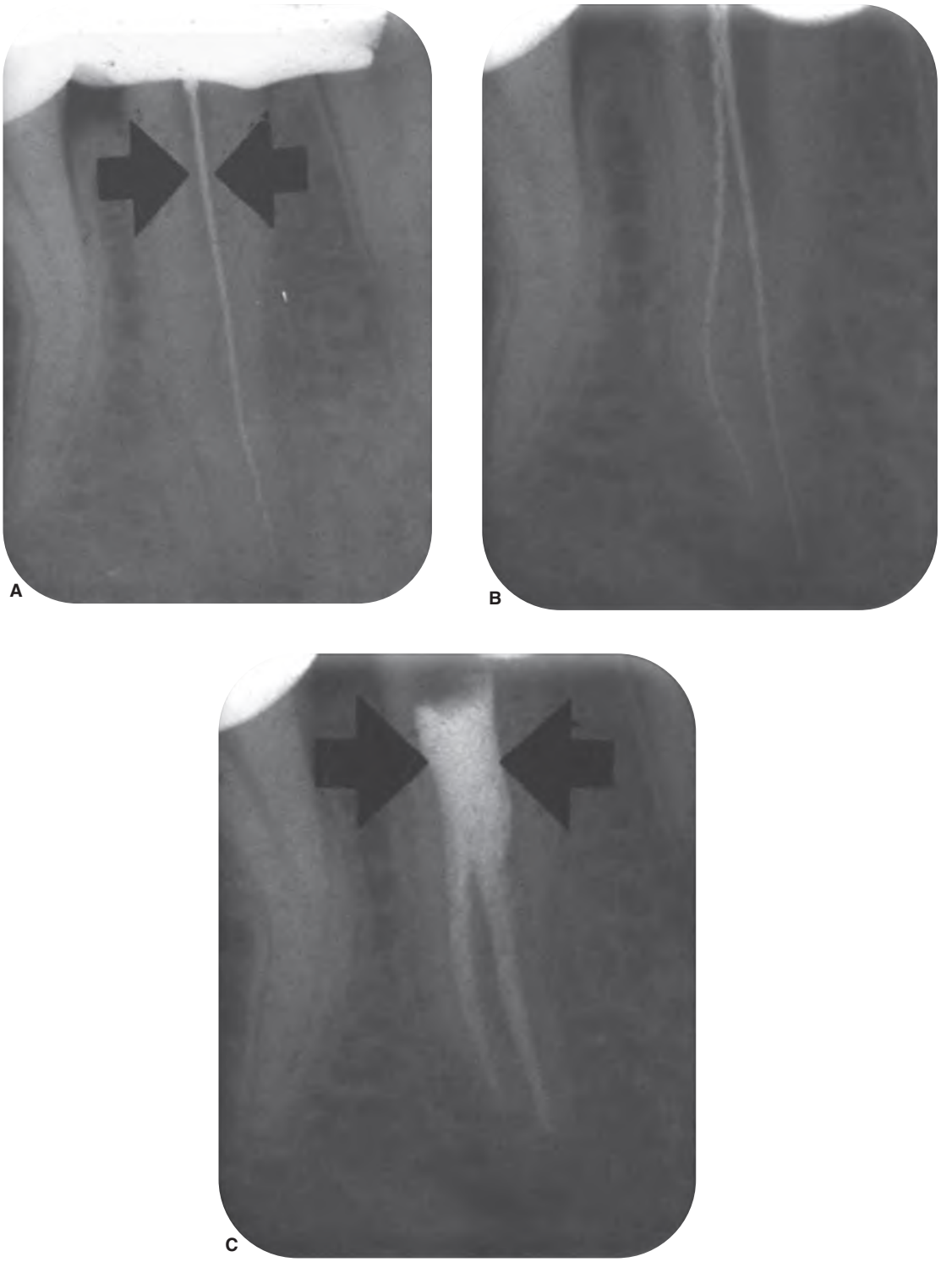


FIGURE 20-5 ***A.*** Improper primary and secondary access did not allow detection and preparation of all root canal systems. ***B.*** Three root canals could be located but improper secondary access still prevents complete instrumentation. ***C.*** Following extension of the access all three root canals could be accessed, prepared and obturated.

Key principle: Most apical problems originate coronally! The severity of coronal problems increases on the way to the apex!

The main objectives of the coronal access preparation are to

- Provide a straight line access to the root canals[11]
- Confirm the etiology of pulp pathosis
- Assess restorability of the tooth

A straight access is necessary to allow unhindered introduction of instruments, irrigants, and medicaments to the entire length and circumference of the canal, with minimal loss of dental structural integrity. Existing restorations, caries, and unsupported enamel and dentin should be removed and the remaining tooth structure examined under an operating microscope for presence of cracks, height and thickness of the remaining dentinal walls for ferrule effect, relationship between the remaining coronal tooth margins and the osseous crest, root length, location of the furcation, and amount and quality of attached gingiva.

A more difficult situation arises when an existing crown is present on the tooth that requires endodontic treatment. Abbott[12] found that there was less than 60% chance to detect caries, fractures, and marginal breakdown, without complete removal of restorations. However, it is impractical to suggest removing all crowns prior to root canal treatment, and therefore a careful assessment of the integrity of the crown must be attempted through history-taking and clinical examination before and during treatment. Preparation of the access cavity requires thorough knowledge of tooth and root anatomy and their variations.[11,13]

Guidelines for Preparation of the Primary Access Cavity

Determination of the point of penetration: Usually, entry is in the center of the occlusal table but in certain teeth (e.g., maxillary molars) it is deceiving, as the center of the occlusal table does not reflect the center of the pulp chamber. Anatomy and strategies for access are detailed in sections on individual teeth below.

Assessment of occlusal and external root form: Once the point of entry has been determined, the bur's angulation in three dimensions has to be mentally envisioned. This is determined by taking into account the angulation of the teeth in the jaws and assessing the external root surface at the level of the CEJ.

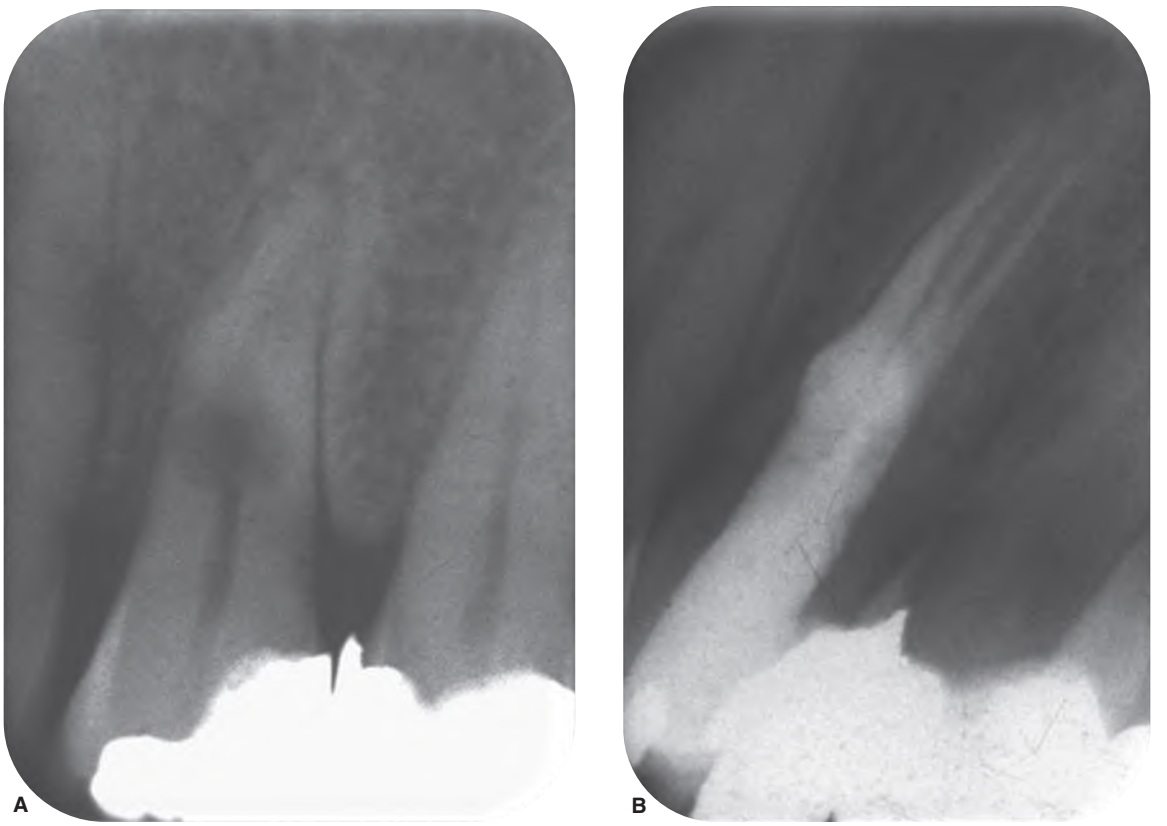


FIGURE 20-6 ***A & B.*** Only after enlargement of the coronal access cavity, proper detection and preparation of all three main root canals became possible.

Radiographic measurement of the depth of the pulp chamber roof from the occlusal table: The initial bur in the high-speed handpiece is placed against a radiograph or a measurement determined from a calibrated digital image.

Assessment of complicating factors: Rotations/tipping of teeth, calcifications (pulp stones, mid-root calcification), deep restorations, buccal/lingual restorations, root length, width, and curvature affect the angle of entry and the degree of extension of the access cavity in the horizontal and vertical dimensions.

Radiographic assessment: Angled views should be taken in an attempt to visualize the breadth of the roots and the position of the canal within it. One also has to assess the angle at which the canal leaves the pulp chamber.

Maxillary Central Incisors

The roots are usually straight and have the least incidence of dilacerations.[14]

Initial penetration should be approximately in the middle of the lingual surface of the tooth, just above the cingulum, almost perpendicular to the lingual surface. After locating the canal, long tapered diamonds can be used to extend the access into a roughly triangular outline (Figures 20-7 and 20-8).

Maxillary Lateral Incisors

The root typically curves to the distal although some can be straight or curving to the mesial. There is often a deep developmental groove running along the cingulum on the lingual surface. Usually the tooth shows one single root canal.

Maxillary Canines

The position of the cusp tip relative to the long axis of the root is in line with the center of the root tip in the labial

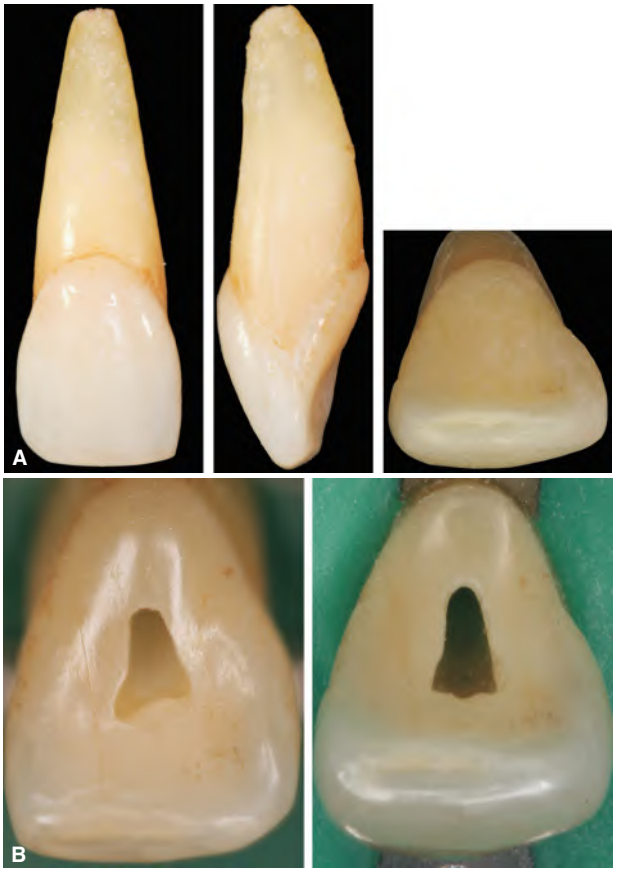


FIGURE 20-7 Example of a maxillary central incisor shown in labial, mesial, and incisal views. ***A.*** The access preparation can be assessed from a more incisal view and a lingual view ***B.*** The width of the preparation in the cingulum is 1.1 mm. Note also the extension of the mesial and distal incisal areas to open up the pulp horns.

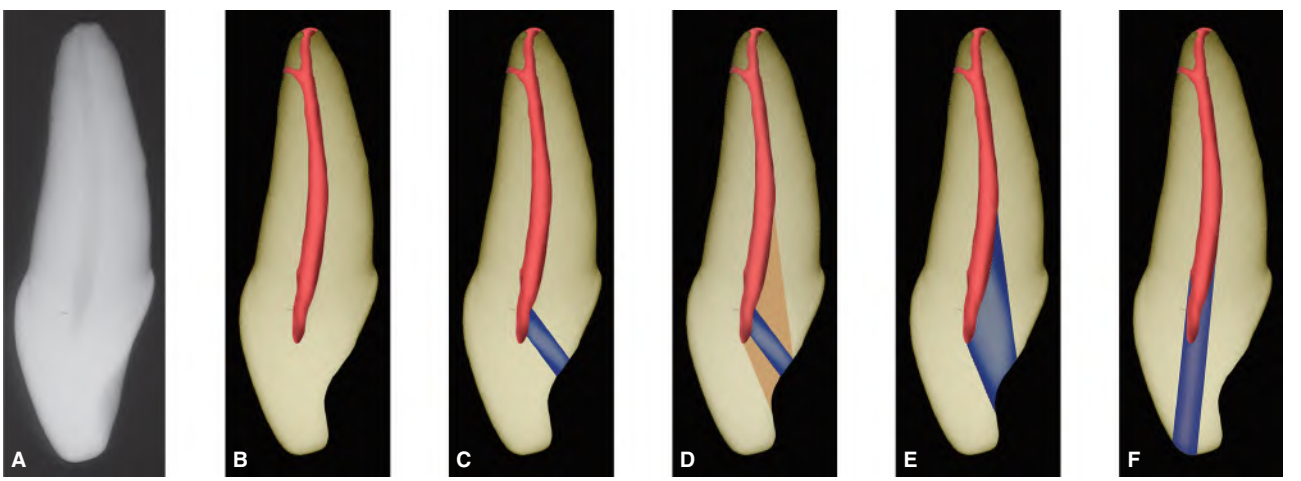


FIGURE 20-8 Potential design of access into a maxillary incisor with schematic drawings based on an original radiograph ***A.*** The preoperative relation of the canal within the root can be estimated ***B.*** Note how the true straight-line access (SLA) is labial to the incisal edge ***C.*** The initial access entry site and angulation ***D.*** tan-colored highlights show the restrictive dentin impeding SLA. ***E.*** The completed lingual access preparation with SLA can be compared to a completed access preparation done from a labial approach ***F.*** Note how far more tooth structure may be conserved with a labial access but at the cost of disrupting the labial esthetic surface.

view but lies labial in the proximal view. It is usually the longest tooth and the largest root in the mouth and often curves apically. As with the central and lateral incisors, the lingual triangle must be removed. The main difficulty with these teeth is that they can be long, often over 30 mm (Figure 20-9).

Maxillary First Premolars

It must be assumed that at least two canals are present, and the third canal is shown to exist in high enough numbers that the access preparation must be designed to search for it.[15] When three canals are present, the pulp chamber morphology resembles that of a maxillary molar (Figure 20-10). The point of entry is centrally in the fossa, aiming at the center point at the CEJ. The outline is an elongated slot and can extend almost to the cusp tips depending on the angle. However, the operator must search for a third canal, usually the mesiobuccal canal. If three canals are found, the orientation is very similar to that of the maxillary molar (Figure 20-11).

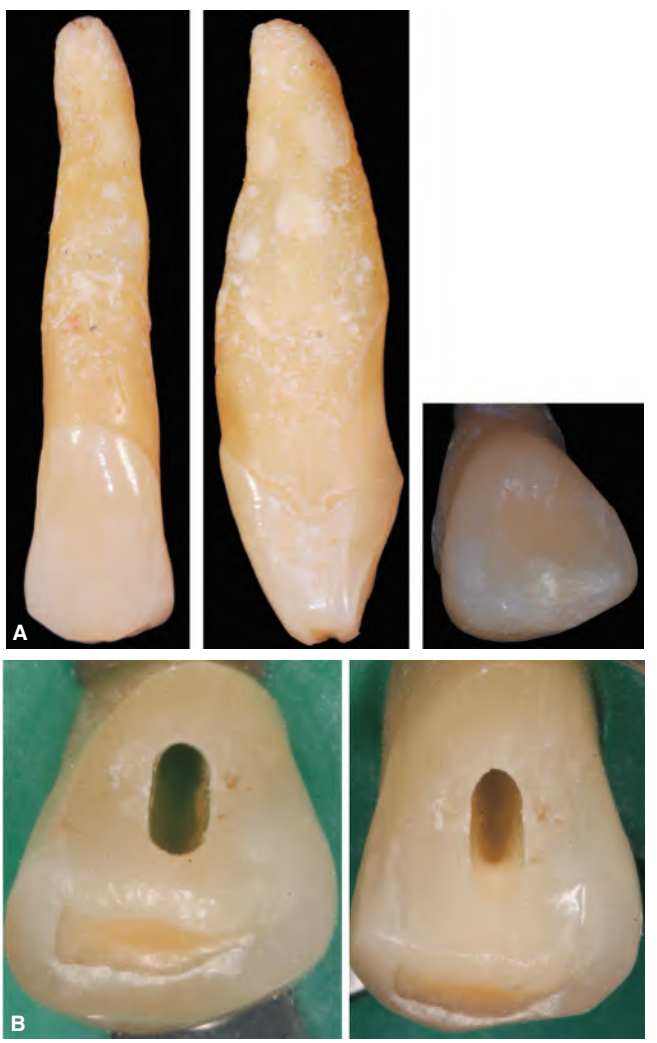


FIGURE 20-9 Example of a maxillary canine shown in labial, mesial, and incisal views ***A***. The access preparation can be assessed from a more incisal view and ***B***. From a lingual view.

Maxillary Second Premolar

This tooth usually has a single root, the incidence of two canals is significantly less but when present they are not spaced so far apart from each other[16] (Figure 20-12).

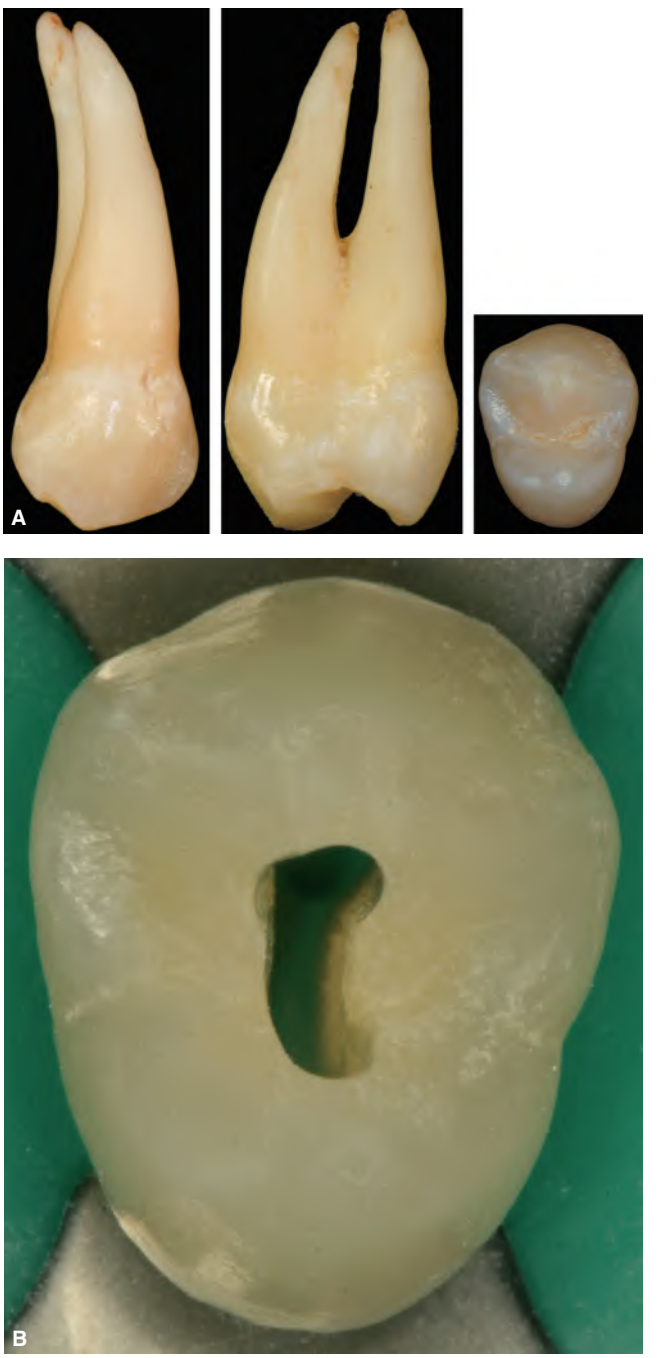


FIGURE 20-10 ***A***. Example of a maxillary first premolar shown in buccal, mesial, and occlusal views. ***B***. A typical access preparation. Note how the buccal part of the preparation has been extended mesiodistally to explore a potential second buccal canal.

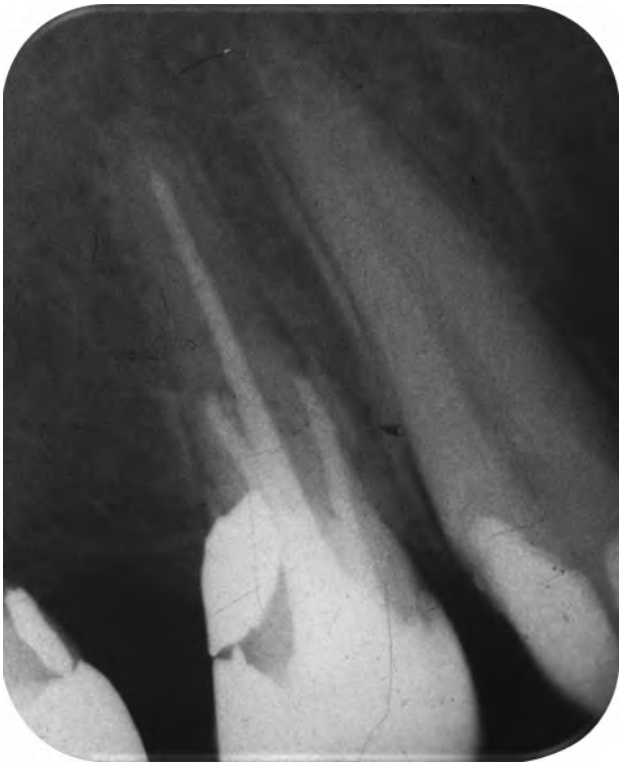


FIGURE 20-11 Maxillary premolar with three root canals. The buccal canals separate deeper apically than the palatal and the main buccal root canal orifices.

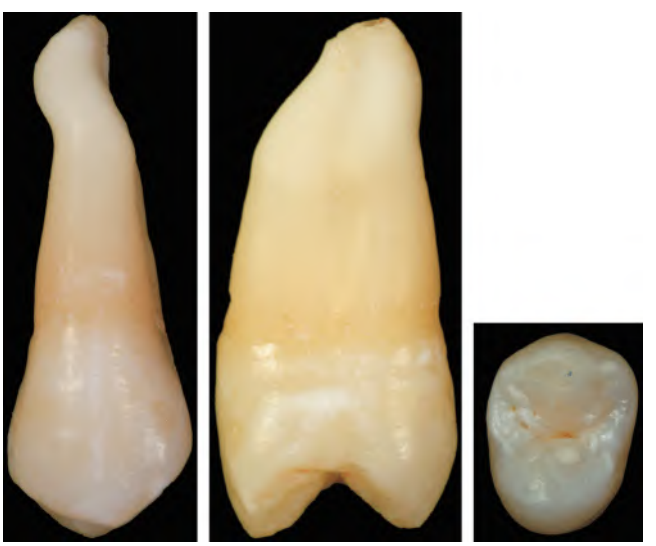


FIGURE 20-12 Example of a maxillary second premolar shown in buccal, mesial, and occlusal views. The access preparation is virtually identical to the one of maxillary first premolar.

Maxillary First Molar

It is the largest tooth in the maxillary arch with four well-defined cusps and a supplemental cusp of Carabelli on the mesiolingual cusp. From the occlusal view, it has a roughly rhomboidal outline. For the purpose of access cavities, the molars can be viewed as having a triangular arrangement of the cusps (without the distolingual cusp). In fact the access cavity is usually kept mesial to the oblique ridge (Figure 20-13).

Maxillary molars can present with mild to severe curvatures and usually have two or three canals in any root (but most commonly in the mesiobuccal root). Locating the second mesiobuccal canal (MB-2) orifice routinely can be difficult as it is often buried under a bridge of dentin.[17-19] The canal can have a severe curvature to the mesial and the buccal in its coronal section and is usually much smaller than the principal first mesiobuccal canal (MB-1). The access shape should be quadrilateral to allow for troughing 2–3 mm lingually to the MB-1 canal to search for the MB-2 canal.

Entry should be in the mesial fossa and should be kept small initially. Initial penetration may be aimed at the large palatal canal orifice. There is ample evidence to demonstrate that the mesiobuccal root has a second canal with such frequency that it must be accommodated for in the initial access and always searched for.[17,18,20]

The mesiobuccal canals have two separate apical foramina or, more commonly, join to exit at one foramen. The access preparation for the DB canals does not have to be extended as far toward its line angle as the mesial canals. The distal canal is distally inclined and so the file projects out of the orifice to the mesial naturally. Therefore, the transverse ridge can usually be preserved.

Maxillary Second Molars

Maxillary second molars are very similar in form and function to the maxillary first molars and the access cavity is similar.

Maxillary Third Molars

From an occlusal viewpoint, this tooth usually presents with a heart-shaped triangular outline similar to the second molar. The roots are often fused forming one large root. The access cavity is similar to the other maxillary molars but needs to be modified according to the specific anatomical variations that this tooth presents.

Mandibular Central and Lateral Incisors

The mandibular central incisor is usually the smallest tooth in the mouth. Bilateral symmetry is common. There are slight differences between central and lateral incisors, but the most important with regard to access is that the crowns of lateral incisors are not as symmetrical as central incisors from the incisal view as they curve distally to accommodate the curvature of the arch and correspondingly the cingulum is displaced slightly to the distal. Lateral incisors are also slightly larger than the centrals.[21,22]

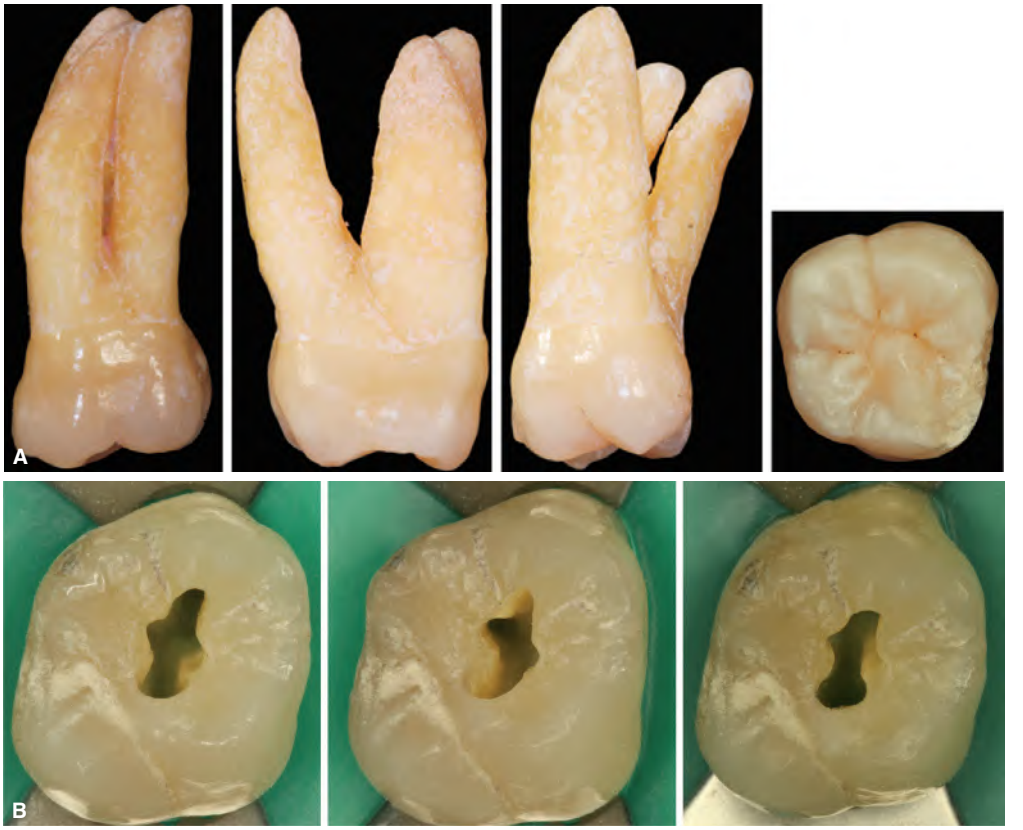


FIGURE 20-13 Example of a maxillary molar shown in buccal, mesial palatal and occlusal views. ***A.*** Access into both mesiobuccal canals and distobuccal and palatal canals is gained ***B.*** Note that the preparation has not been extended much on the occlusal table for the distobuccal canal as the canal projects distally naturally.

The entry point for access should be just above the cingulum with the bur angled perpendicularly to the surface of the entry point (Figure 20-14). As these teeth are narrow mesiodistally, the main concern is the width of the preparation. Once the chamber or the canal is found, the access can be precisely widened for each individual tooth. Approximately 40% of mandibular incisors have two canals, one buccal and one lingual with only 2% to 3% having separate apical foramina (Figure 20-15). It is natural for the hand to angle the bur toward the buccal (thereby running the risk of gouging the labial wall). The lingual canal lies 1 mm to 3 mm away from the buccal, directly under the cingulum. Even when two canals are present, there is often a fin or a groove with pulp tissue between them.

Mandibular Canines

These teeth are very similar to their maxillary counterparts but are usually narrower (approximately 1 mm) and shorter in root length by 1 to 2 mm (Figure 20-16). The mesial edge is almost straight and therefore the access cavity can be prepared more mesially to the center point of the lingual surface. The shape of the preparation ranges from an oval to a rounded slot, depending on the size of the pulp chamber inside.

Mandibular First Premolars

The mandibular first premolars have characteristics of a small canine because of a sharp buccal cusp (only occluding cusp) and a small lingual cusp that sometimes resemble a cingulum. There is a characteristic mesiolingual developmental groove that makes the tooth asymmetrical.[23,24]

The entry point is in the middle of the central groove, and the bur is directed to the buccal. Again, the access shape is an oval slot. As the crown is lingually inclined, the access cavity will result in removal of more of the buccal cusp than the lingual (Figure 20-17).

Mandibular Second Premolar

Access is similar to the first mandibular premolars, taking overall dimensions into account[23,24] (Figure 20-18).

Mandibular First Molars

Access preparations often encroach on the buccal cusp tip (mesiobuccal) but rarely onto the lingual.

Mandibular molars are known to have complex anatomy with usually four canals but three and five canals are well documented. It is important to note that there is almost always a concavity on the furcal side of both the mesial and distal roots; providing a high risk of strip perforation. The roots start to bifurcate 3 mm below the CEJ.[25,26] Both mesial and distal roots are broad and usually have two canals. Often they have an interconnecting fin that can be extremely variable in its persistence at the apex. Two distal roots can occur but are rare.

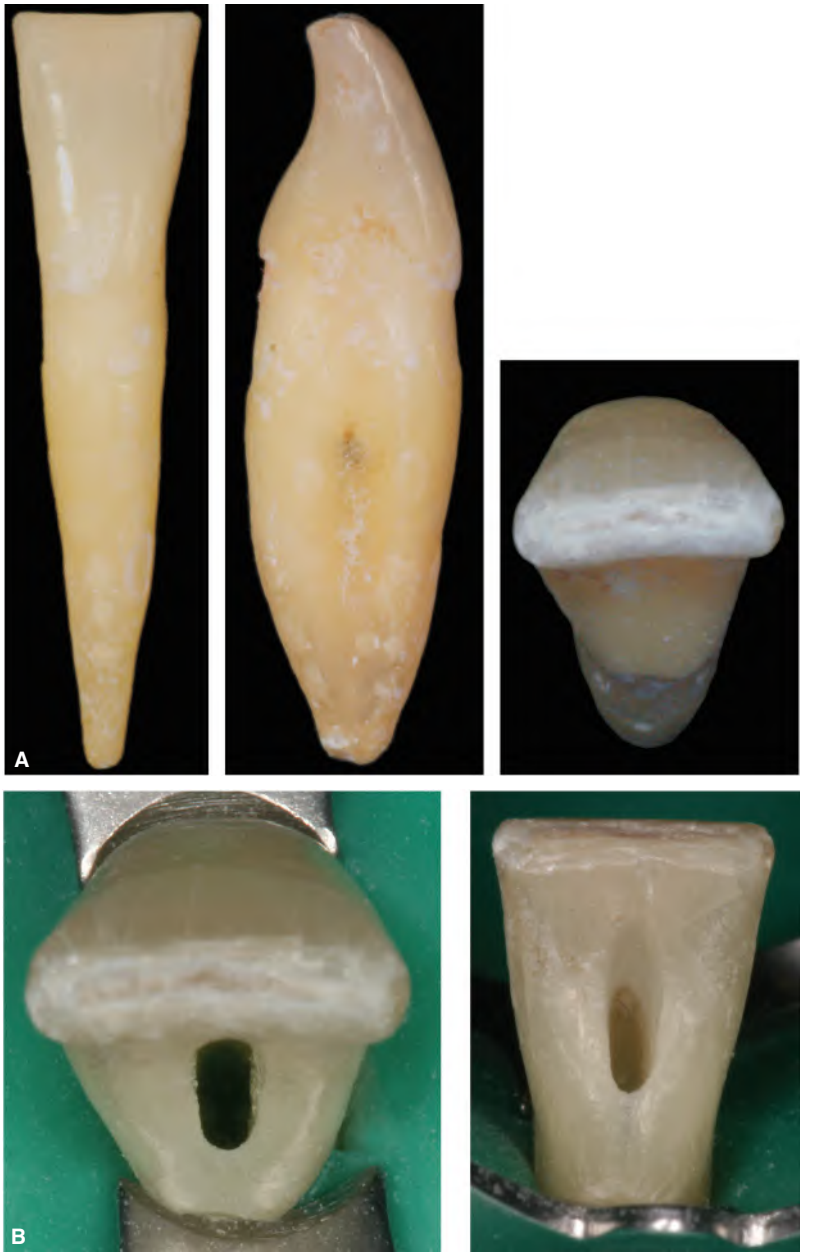


FIGURE 20-14 ***A.*** Example of a mandibular incisor shown in labial, mesial, and incisal views. ***B.*** The access cavity.

The entry point is just mesial to the central pit. Initially, the access preparation should not be extended further distally as the distal canals project mesially toward the occlusal table. Once the angle of the canals has been determined with files, the decision can then be made to extend the access distally, if needed. However, the access will be located largely in the mesial half of the tooth (Figure 20-19). Sometimes, it is necessary to encroach on the mesiobuccal cusp tip to achieve straight line access.

The groove between the mesiobuccal/mesiolingual and distobuccal/distolingual canals must always be troughed with a bur and/or ultrasonics and checked with files to search for mesial canals. The distal canal is ovoid or figure eight-shaped buccolingually if only one canal is present. There can be a second distal canal that bifurcates in the apical third.

Mandibular Second Molars

Generally, mandibular second molars differ from the first molars in their capacity for variation.[25,26] The access cavity may have to be extended more toward the mesial marginal ridge than for first molars. The roots, and therefore canals, are usually closer together and can be fused to a single conical root with varying internal anatomy or often C-shaped canal systems.

The entry point of the access cavity should be mesial to the central pit similar to the first molars, but may not require the same degree of mesial extension if the roots are closer together. Otherwise, a similar preparation as for the first molar can be made.

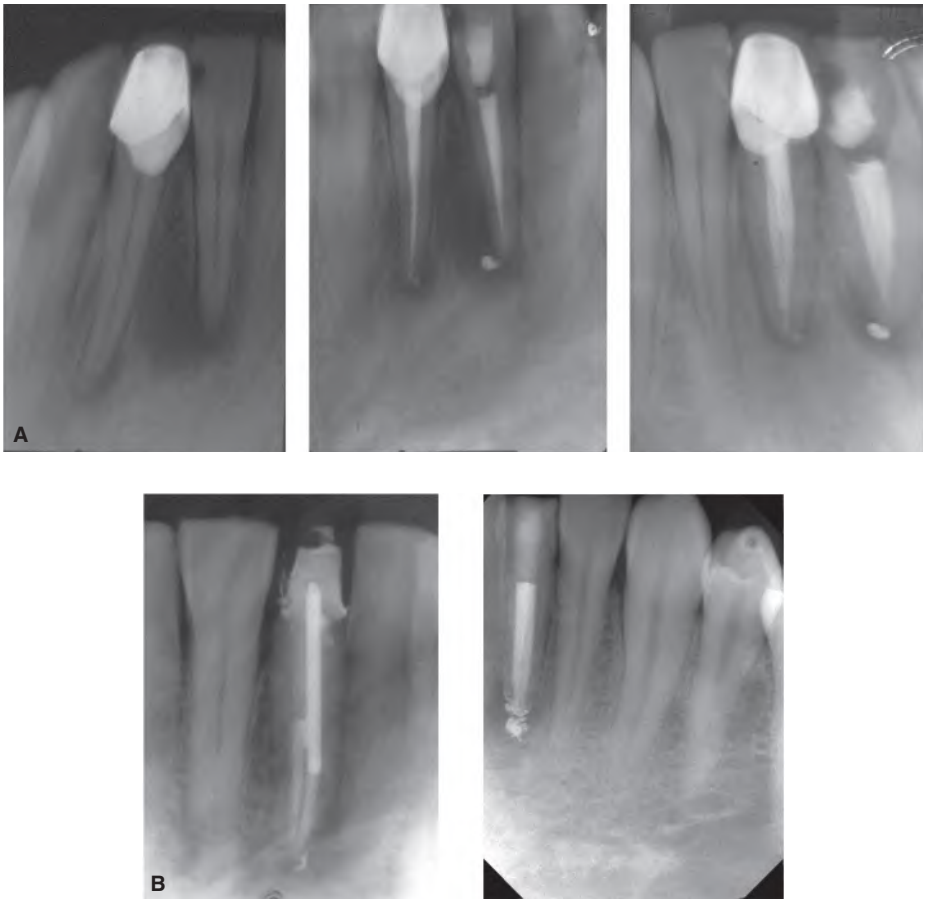


FIGURE 20-15 Radiographs illustrating variations of lower incisors with two canals. ***A.*** Although usually both canals exit from a common apex, they can have two separate or a figure eight-shaped foramen. ***B.*** Preparation can be assessed from a more incisal view and a lingual view. Note how the lingual extension of the access preparation extends well into the cingulum.

Mandibular Third Molars

Third molars have significant variations and anomalies. They are usually less developed, with oversized crowns and undersized roots. Due to the lack of space in the jaws, they are often impacted and may have very curved roots. Usually third molars have two roots—a mesial and a distal root that can be bifurcated or fused. When fully erupted and in functional occlusion, third molars provide excellent protection for the first and second molars by spreading the occlusal load. In such situations, keeping these teeth is beneficial. The access cavity needs to be adjusted according to the specific anatomical variations that this tooth presents.

Procedural Mishaps

Successful endodontic treatment originates from a well-designed and executed access preparation. The opposite is also true; errors during root canal treatment can often be traced back to a problem originating from an inadequate access preparation. Errors generally stem from two access cavity characteristics: underextension and overextension. Additionally, perforations of the pulp chamber walls are possible mishaps.

Underextension

Not opening up the access cavity across the width of the root sufficiently can result in the operator missing canals. They can often be buried under calcified dentin. However, if the canal is found but the cavity is not extended away from the furcation sufficiently, the file will preferentially cut the furcal dentin causing a strip perforation. Another possibility, of not extending the access far enough, is that the coronal canal curvature is not removed. This will increase chances of instrument separation as the file attempts to go around more than one curve, or transportation at the apex as the coronal canal curvature renders the file tip uncontrollable. Underextension of the access preparation also limits the final diameter of the apical preparation size. In anterior teeth, if the pulp horns are not adequately exposed and cleaned, the remaining pulpal tissue will cause coronal discoloration.

Overextension

Generally, overextension will cause unnecessary removal of tooth structure that weakens the remaining crown and ultimately decreases the long-term prognosis of restoration, and therefore the tooth.

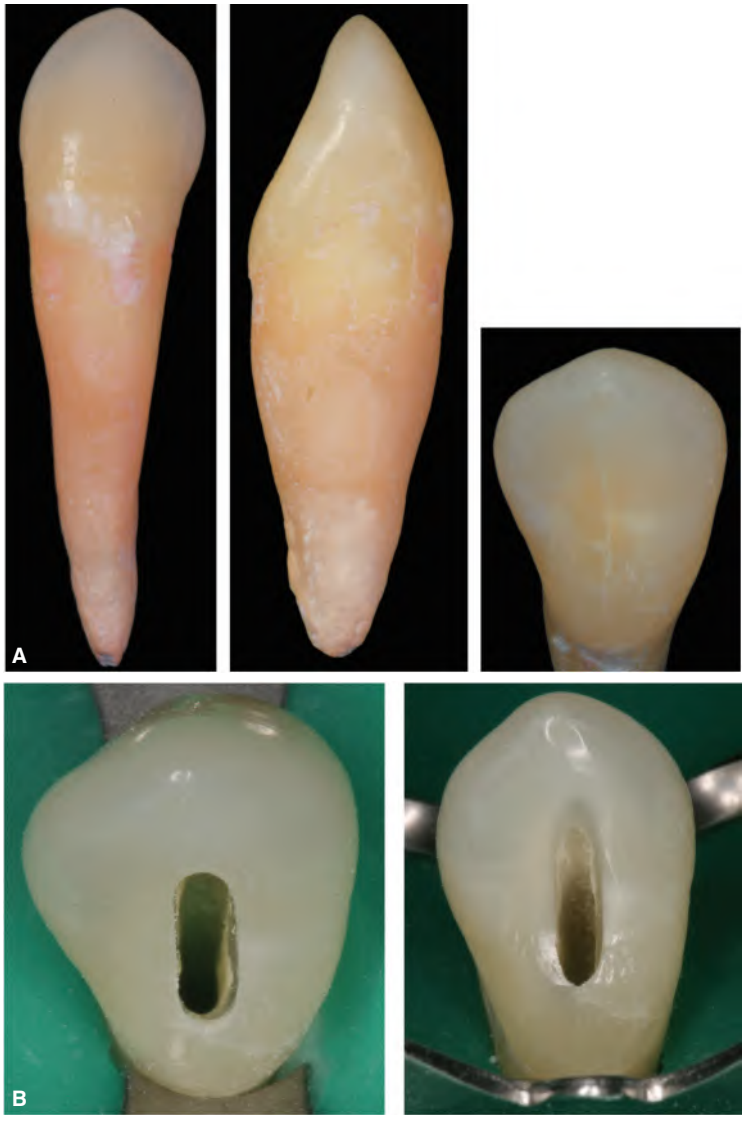


FIGURE 20-16 ***A.*** Example of a mandibular canine shown in labial, mesial, and incisal views. ***B.*** The access preparation can be assessed from a more incisal view and a lingual view. Note that the access is extended lingually to detect a second, usually lingually- located canal.

Perforation

Perforations of the pulp chamber may appear on the pulp chamber floor as a result of improper search for root canal orifices. In severely calcified teeth, the pulp chamber sometimes is extremely narrow and forced preparation may result in missing the pulp chamber space and perforation of the pulp chamber floor. Second reason for perforation can be incorrect angled introduction of a bur resulting in lateral perforations, mostly at the mesial or distal aspect of the crown. When in doubt, the access cavity can be prepared before placement of the dental dam, thus allowing orientation at the neighbouring teeth.

Preparation of the Pulp Chamber

Following removal of the pulp chamber's roof, the chamber itself should be cleaned meticulously from all necrotic or vital tissue. Preparation should be extended to a degree allowing complete removal of all tissue remnants even from minor undercuts. It should be noted that in a tooth with a necrotic, infected pulp the majority of microorganisms are located in the pulp chamber. Instrumenting a root canal through a contaminated pulp chamber will force microorganisms further into the root canal and transport them apically. Mechanical cleaning can be done using rose burs, and ultrasonic or sonic instruments (i.e., SonicFlex, KaVo). The pulp chamber is repeatedly flooded with copious amounts of sodium hypochlorite thus initiating disinfection of the endodontic cavity immediately upon starting treatment.

Preparation of the pulp chamber is complete when a completely clean coronal access cavity is achieved. The pulp chamber floor should be completely free from any remnants of hard or soft tissue.

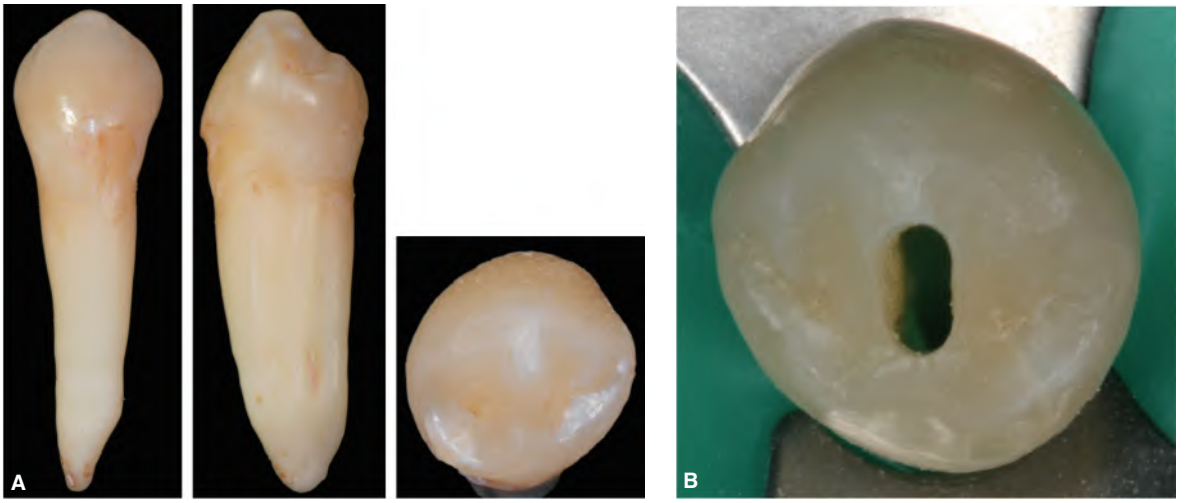


FIGURE 20-17 ***A.*** Example of a mandibular first premolar shown in buccal, mesial, and occlusal views. ***B.*** The access preparation can be assessed from an occlusal view.

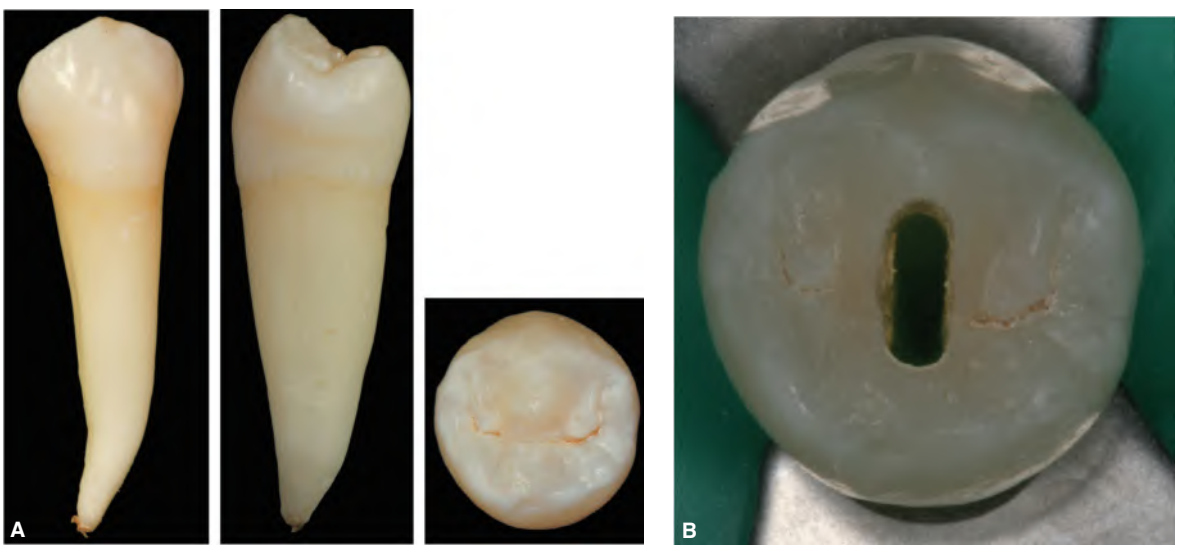


FIGURE 20-18 ***A.*** Example of a mandibular second premolar shown in buccal, mesial, and occlusal views. ***B.*** The access preparation can be assessed from an occlusal view.

Following proper irrigation and drying, the pulp chamber should be thoroughly inspected, preferably by a dental operating microscope or loupes, with proper illumination and magnification (Figures 20-20).[27,28]

Pulp chamber inspection should look for the following:

- Remnants of vital or necrotic soft tissue.
- Remnants of filling materials (amalgam, gold or ceramic particles, cement).
- Caries.
- Secondary and tertiary dentin.
- Perforations.
- Fractures, cracks, and craze lines.
- Calcifications.
- Pulp stones.
- Root canal orifices.

Pulp Stones and Coronal Calcifications

Calcifications and pulp stones may present a challenge even for an experienced clinician since they can cause difficulties to locate and prepare all root canal orifices.[2,29]

Sometimes, larger pulp stones can be identified on radiographs, especially on bitewing or single tooth radiographs. Detection and identification of pulp stones can be best achieved with enhanced illumination (and magnification) of a meticulously dried pulp chamber, as the color and structure of pulp stones are different from that of dentin. Pulp stones show a round or oval form, no sharp contours, and a yellowish, slightly transparent, amber-like color.

The ease and technique of removal depend on the size and type of the pulp stone. If lightly attached to the dentin, pulp stones can often be chipped away using a dental probe or an

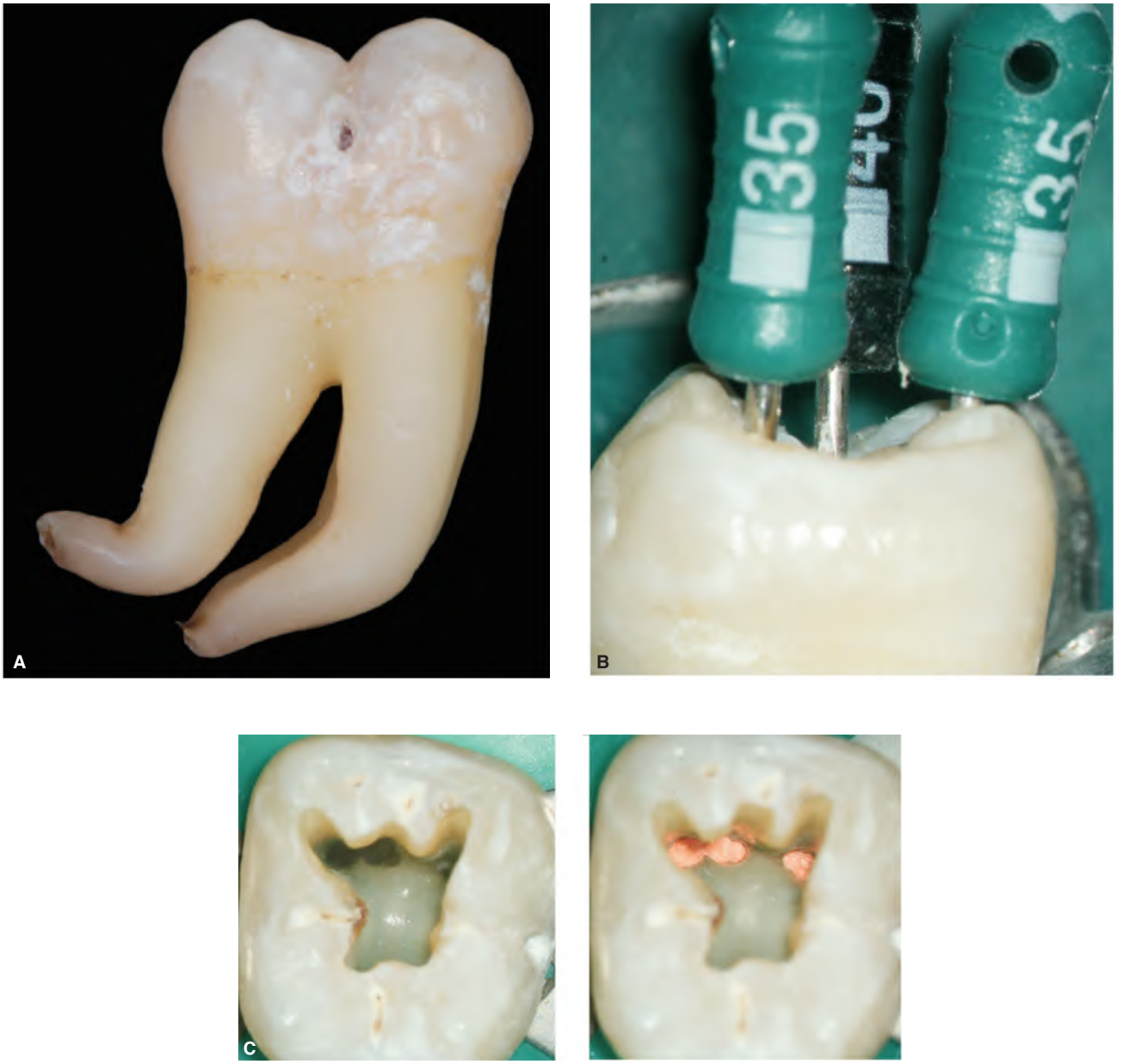


FIGURE 20-19 A. Access preparation in a mandibular molar with severe root curvature. B. Hand files can be placed to length in all three mesial canals after adequate access and canal preparation. C. However, the access preparation requires an extreme extension for the mesiobuccal and mesiolingual canals due to the root curvature.

ultrasonic tip. If firmly attached to the dentin, the use of a bur or a diamond-coated ultrasonic or sonic tips is advantageous (Figure 20-21).

It should be noted that pulp stones may be associated with certain systemic diseases such as renal problems or coronary heart diseases, or with consumption of certain medications.[30-32]

Coronal calcifications and constant narrowing of the pulp chamber are a result of deposition of secondary and sometimes also tertiary dentin. Calcification can be accelerated in the presence of active caries, stimulating dentin secretion by odontoblasts, and has been detected in more than 90% of patients older than 40 years.[33] Calcifications can be identified by color and texture following thorough drying of the pulp chamber; enhanced magnification and illumination again are helpful. Small instruments such as pathfinding files and similar files (see later: pathfinding instruments) will facilitate bypassing of pulp stones and locating orifices in calcified pulp chambers.

Complete canal calcification is the exception and can be detected following dental trauma. Otherwise, clacification is usually incomplete and limited to the coronal part(s) of the root canal. Calcification is incomplete if:

- A canal space can be detected on the radiograph.
- The tip of a file is engaging inside the canal.[34]

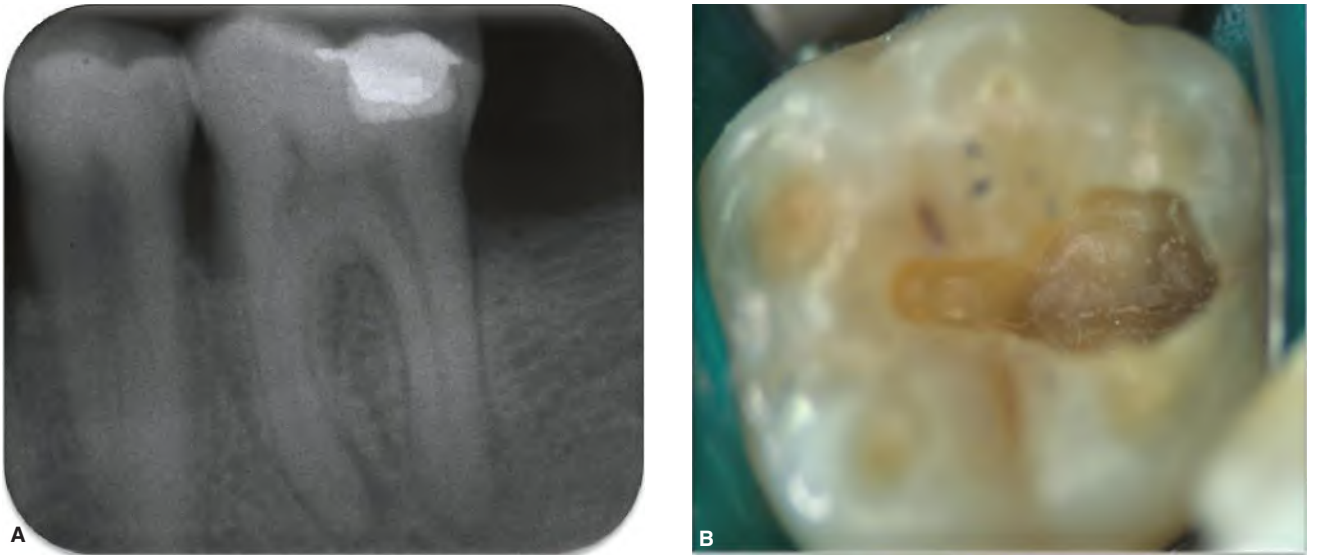


FIGURE 20-20 ***A & B.*** Intracoronal diagnostics. Following removal of the restoration the etiology for pulp necrosis could be identified (i.e., a crack in the pulp chamber's roof).

- Electronic determination of root canal length reveals a signal.
- An apical lesion is present.
- Bubbles can be observed evading from an irrigated root canal (see later).
- Pain upon instrumentation occurs.

Although present, an extremely calcified root canal sometimes cannot be instrumented to sufficient length, due to sharp curvatures or too narrow a diameter of the canal lumen. It has been demonstrated, on extracted teeth, that a patent root canal was present in all teeth with apical lesions but only in 1 out of 14 root canals without apical pathosis[35] (Figure 20-22). In 11 of 12 teeth presenting with apical periodontitis that could not be instrumented to the apical foramen and radiographically did not show a visible root canal, the presence of a root canal could be demonstrated with a contrast medium.[35] A reduced success rate for teeth with non-penetrable root canals was reported only when a preoperative apical lesions was present (97.9% success without lesion; 62.5% with lesion).[36] In a clinical study on 57 cases (114 paired teeth: one tooth with penetrable root canal and one tooth with a non-penetrable root canal), teeth with penetrable canals showed a success rate of 94.7% one year postoperatively.[37] Teeth that could not be instrumented to the foramen showed a success rate of 75.4%. Non-penetrable teeth with an apical lesion showed a 4.8 higher odds of treatment failure.[37]

Identification of Root Canal Orifices

It has been demonstrated that missed root canals contribute to endodontic failures.[38] Several techniques, instruments, and strategies for detection of root canal orifices as well as some basic anatomic rules have been described in the endodontic literature.[39,40]

Anatomic Rules

Krasner and Rankow[40] described some patterns in orifice location, pulp chamber size and shape, and hard tissue color and shape:

Law of Centrality: The floor of the pulp chamber is always located in the center of the tooth at the level of the Cemento Enamel Junction (CEJ).

Law of Concentricity: The walls of the pulp chamber are always concentric to the external surface of the tooth at the level of the CEJ, that is, the external root surface anatomy reflects the internal pulp chamber anatomy.

Law of the CEJ: The distance from the external surface of the clinical crown to the wall of the pulp chamber is the same throughout the circumference of the tooth at the level of the CEJ. The CEJ is the most consistent repeatable landmark for locating the position of the pulp chamber.

Laws of Symmetry:

1. Except for the maxillary molars, the orifices of the canals are equidistant from a line drawn in a mesial–distal direction, through the pulp chamber floor.
2. Except for the maxillary molars, the orifices of the canals lie on a line perpendicular to a line drawn in a mesial–distal direction across the center of the floor of the pulp chamber.

Law of Color Change: The color of the pulp chamber floor is always darker than the walls.

Laws of Orifice Location:

1. The orifices of the root canals are always located at the junction of the walls and the floor.
2. The orifices of the root canals are located at the angles in the floor–wall junction.
3. The orifices of the root canals are located at the terminus of the root developmental fusion lines.

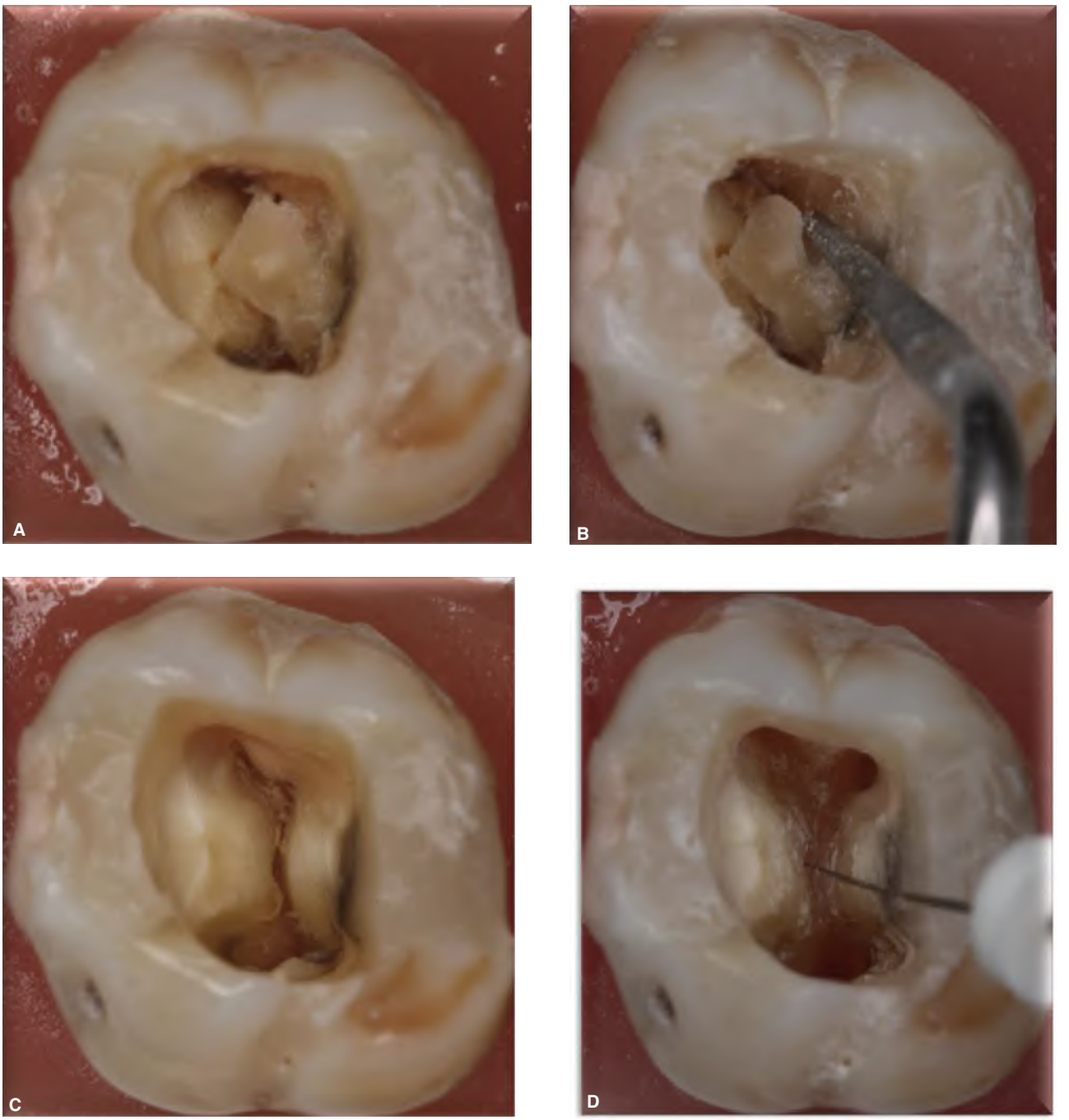


FIGURE 20-21 ***A.*** A large pulp stone blocks the pulp chamber. ***B.*** Removal of the pulp stone. ***C.*** Preparation of the pulp chamber floor and localization of the root canal orifices. ***D.*** Detection of an MB-2 canal.

As the external root surface is a reliable guide, it can be quite frustrating to place a dental dam that completely obscures it. Therefore, in difficult cases, it is prudent to prepare the initial access shape and locate the pulp chamber or at least one orifice prior to dental dam placement.

Key principle: A tooth has only one root canal if no second one can be detected!

Techniques and Devices

Dental Explorer

Using a small and sharp endodontic explorer, all irregularities and grooves on the pulp chamber floor should be probed. Sharp and stiff endodontic files can also be used. Additionally, debris can be accumulated on the pulp chamber floor clogging canal orifices. Chipping away the dentinal shavings with explorers or endodontic instruments can disclose the orifice.

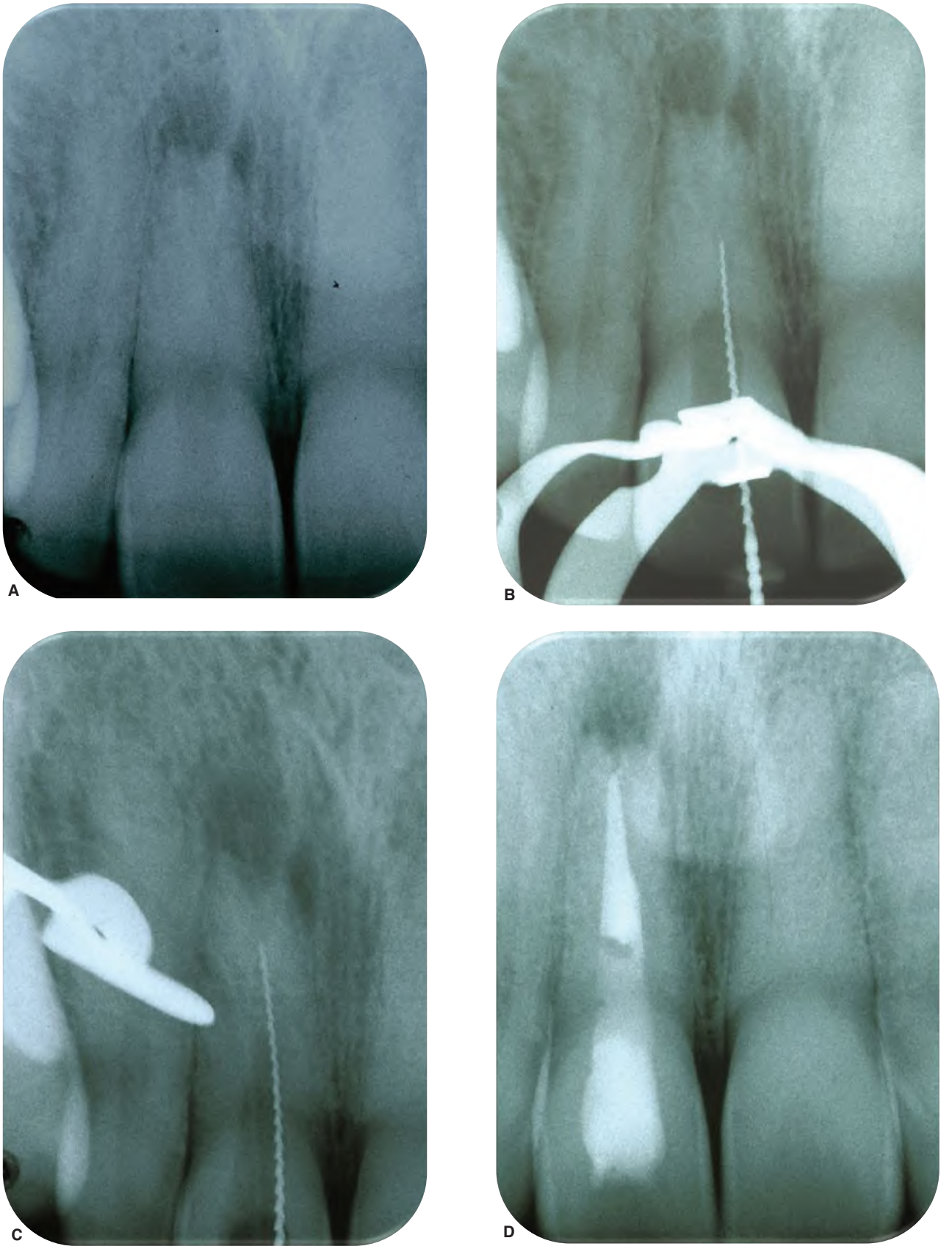


FIGURE 20-22 ***A-D.*** Radiographically calcified root canal system in a tooth with periapical pathosis that was difficult to negotiate.

Dyes

Dyes such as erythrosine (caries detector) or methylene blue can be used. After application of the dye, the cavity is washed out and searched for traces of remaining dye that may indicate the location of a previously undetected orifice.

Sodium Hypochlorite

A drop of sodium hypochlorite is applied to the access cavity floor and observed under magnification and illumination for bubbles evading from a hidden root canal (champagne test). The bubbles are a result of disintegration of remaining pulpal tissue induced by the sodium hypochlorire.

Preparation of the Secondary Access Cavity (Root Canal Orifices)

Coronal Flaring

Coronal flaring should always be done as the first step of canal preparation. Nevertheless, the following should be considered when planning the extent of the coronal flaring:

Key principle: Preparation of the secondary access cavity should never be initiated before preparation of the primary access and cleaning of the pulp chamber are completed.

- A wide orifice facilitates easy insertion of instruments and irrigants and allows large amounts of irrigant to be introduced into the endodontic space
- A wide and deeply flared root canal reduces friction of the instruments and thereby reduces the risk of instrument fracture. Reduced friction allows for better control of the instrument, improves tactility, and allows more precise manipulations of the files in the apical part of the root canal. If an instrument has too much coronal friction, it is difficult or even impossible to direct it apically, as the friction will dictate the extent and direction of the apical preparation.
- Flaring outside of the curvature can reduce the degree of the curvature and facilitate further preparation of the middle and apical thirds of the canal (Figure 20-23).

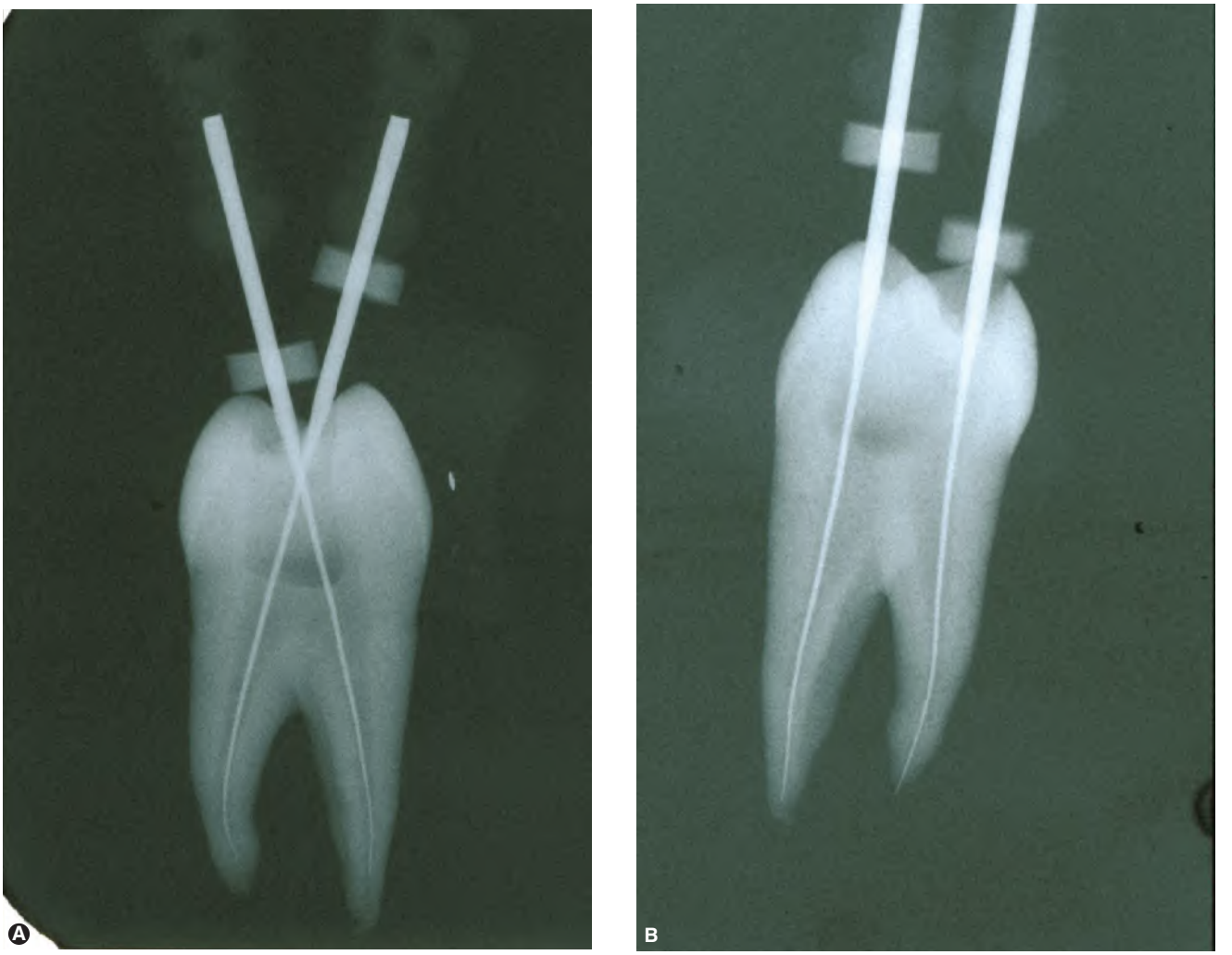


FIGURE 20-23 ***A & B.*** Coronal flaring reduces the degree and length of the curvature and increases the radius thereby reducing the difficulty of preparation.

- On the other hand, a narrow and conservative enlargement of the orifice will save dentinal hard tissue and increase stability and fracture resistance of the root. In molar teeth, the risk of perforation or strip perforation is reduced.

Presently, there are no uniform criteria for the optimal shape preparation of the orifice.

Objectives

Coronal flaring has the following objectives:

- Facilitates insertion of instrument without coronal interferences.
- Improves straight-line access to the middle and apical parts of the root canal.
- Removes reparative dentin at the root canal orifice
- Reduces angle and increases the radius of the root canal curvature.
- Allows early application of sufficient amounts of irrigant into the canal.

Instruments for Coronal Flaring

Gates-Glidden

Gates Glidden (GG) drills are made of stainless steel and are designed with a non-cutting tip. They are also available as NiTi-instruments.

GG are available in six sizes and are rotated in a handpiece at approximately 1000 r.p.m. (manufacturer's recommendation), but frequently are used at higher speed (up to 8000 r.p.m.). GG is safe, relatively inexpensive and effective for root canal preparation. However, it is not intended to prepare the entire root canal. Due to its inflexible shaft, GG cannot bypass a curvature, limiting its action to the coronal straight part of the root canal. GG is an excellent instrument to remove calcifications frequently found just below the orifices of the canal (Figure 20-24). The tip of the smallest GG size (No. 1) equals to an ISO size 50.

The number of rotations till fracture has been reported to be up to 2000 and more,[41] making the instrument relatively safe to use. Fracture usually occurs high at the shaft, making removal easy.

Using larger sizes of GG carry the risk of perforation at the furcal area of the root canal. Crown-down preparation techniques and anticurvature preparation are recommended.[42,43]

The use of GG has been reported to shorten working time and results in better-shaped root canals.[44] When used, the external temperature of the root may rise by 3°C-5°C, depending on dentin thickness, working time, pressure, size of the drill, and rotational speed.[45] This temperature increase is within tolerable levels of the periodontal ligament.

Drux Burs

Drux burs are designed similarly to the GG burs, but have a flexible shaft (Figure 20-25).

Peeso Drills

Peeso drills are designed similarily to the GG but have a longer cutting part. This makes their use in curved root canals hazardous due to the high risk of strip perforations. Today, Peeso drills are not recommended for routine endodontic use.

Orifice Openers

Some rotary NiTi systems are equipped with Orifice Shapers, Intro-Files, or similar instruments. These instruments usually have large tapers and short shafts. Examples are:

- IntroFile (FlexMaster, VDW, Munich, Germany): 12 mm cutting part, 11% taper, non-cutting tip.
- Orifice Opener (ProTaper U, Maillefer, Ballaigues, Switzerland) 14 mm cutting part with 19% taper at the tip decreasing to 3.5% at 9 mm, non-cutting tip.

Preparation of a Glide Path

Contemporary preparation techniques using flexible NiTi instruments start with manual preparation of a glide path for the rotary instruments that follow. The introduction of reciprocal preparation systems, questions the need of a glide path, however, further research is necessary.[46,47]

Objectives

The objectives of preparing a glide path are:

- Early exploration of the root canal system to be prepared.
- Assessment of canal width, content, curvatures, and ledges.
- Reduction of friction of small NiTi instruments thereby lowering the risk of instrument separation.

Pathfinding Instruments

Conventional ISO size instruments, independent of the alloy used, possess a low resistance to buckling. When these instruments are used to penetrate narrow canals they are submitted to a load parallel to their long axis. As a result, instruments with a low resistance to buckling deform easily and their progression towards the apex is hindered. Therefore, it is of clinical importance that special instruments exhibit sufficient resistance to buckling when used for the initial negation of narrow root canals. Thus, these instruments must be resistant to a vertically applied load.

On the basis of these considerations, special stainless steel pathfinding instruments (pilot instruments) for manual use were introduced to improve negotiation of narrow and calcified canals. These instruments are characterized by design features that improve their buckling resistance.[48] Some instruments are heat tempered and their length is reduced to 18 mm or 19 mm, so that more vertical pressure can be used to negotiate calcified

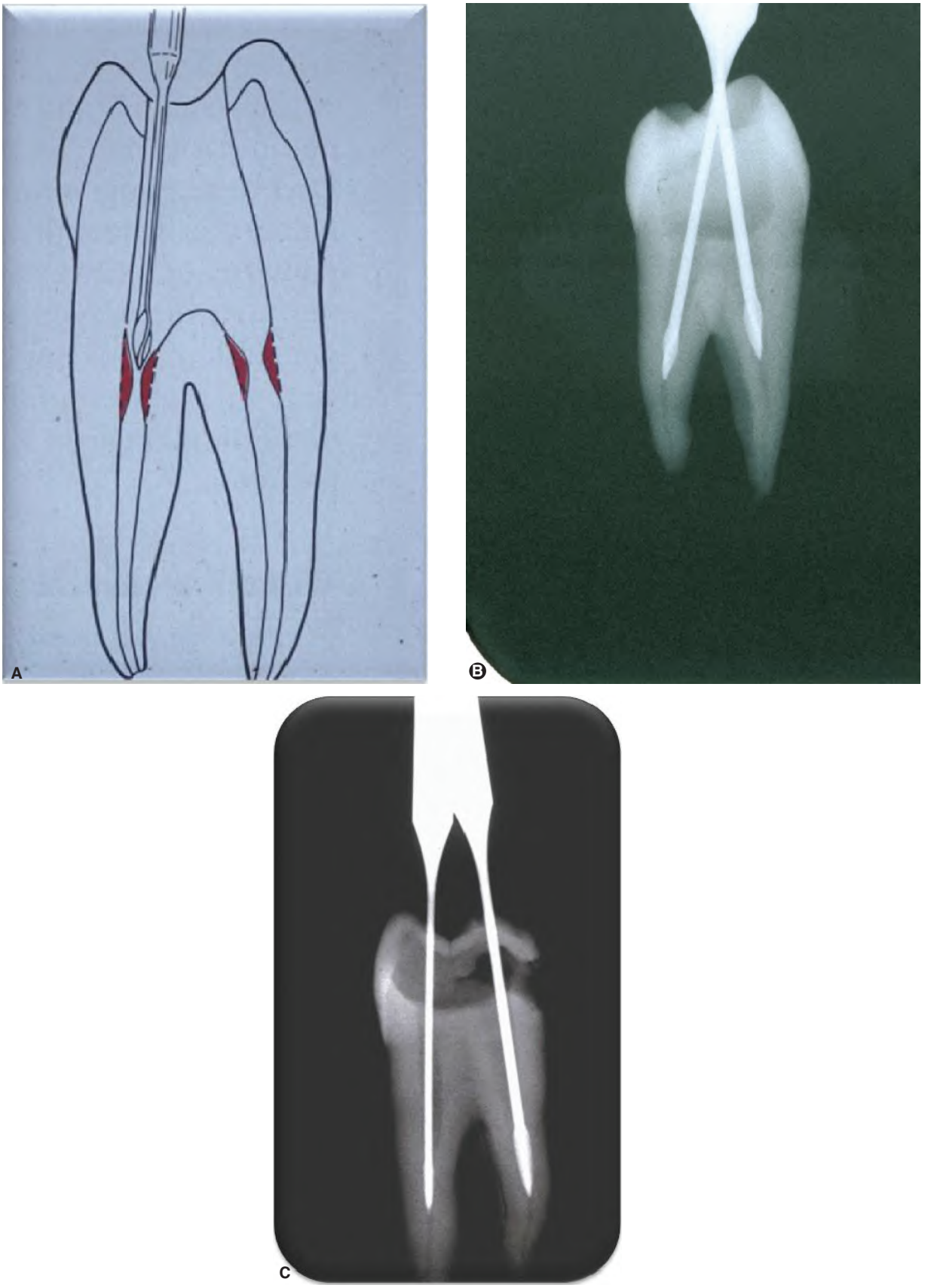


FIGURE 20-24 ***A-C.*** Removal of secondary dentin below the root canal orifice can accelerate and facilitate root canal preparation. Gates Glidden burs can be used safely upto the beginning of the curve.

FIGURE 20-25 Drux bur with a flexible shaft.

TABLE 20-1 Hand and Rotary Instruments Recommended for Preparation of the Glide Path

Manual use
K-files (ISO-sizes 06, 08, 10)
C+-Files (Dentsply/Maillefer, Ballaigues, Switzerland)
C-Pilot-Files (VDW, Munich, Germany)
MMC-Files (MicroMega, Besancon, France)
S-Finder (Sendoline, Täby, Sweden)
Hi-5 (Miltex, New York, U.S.A.)
D-Finder (Mani, Tochigi-Ken, Japan)
Pathfinder CS and SS (Kerr/SybronEndo, Orange, U.S.A.)
Pathfinder CS (Kerr, Orange, CA, U.S.A.)
Farside and Deepstar (Maillefer, Ballaigues, Switzerland)
Antaeos Stiff C-File (Swed, Kew Gardens, U.S.A.)
Stiff C-file (Brasseler, Savannah, U.S.A.)
C File (Roydent, Hoboken, U.S.A.)
Rotary use
Scout RaCe (FKG, La-Chaux-de-Fonds, Switzerland)
G-files (MicroMega, Besancon, France)
PathFiles (Dentsply/Maillefer, Ballaigues, Switzerland)
ProGlider (Dentsply/Maillefer, Ballaigues, Switzerland)
PathGlider (Komet, Lemgo, Germany)

canals.[49] The tip design is modified and less angled than the ISO tip (ProFinder with a 65° tip) to improve negotiation of narrow canal orifices. Some instruments are available with intermediate sizes (e.g., sizes 12.5, 13, 17). The conicity of the ProFinder is less than the ISO standard (1.75% at the tip) and decreases over the working part.

A comparative study revealed that the stainless steel pathfinding instruments (C+ and C-Pilot file) were more resistant to buckling than NiTi pathfinding instruments, whereas the C+ files showed significantly better results than the C-Pilot instrument.[50] These pilot instruments possess an excellent tactile feedback and can be pre-bent easily.[51] Therefore, they are best suited for bypassing ledges at the root canal wall. Normally, ledges are located at the outer side of a curved root canal and when probing a ledge with a hand instrument the feeling is of hitting against a hard structure. In such cases, the stainless steel hand instrument should be pre-bent in such way that that the tip of the instrument can slide along the unchanged inner side of the root canal. Sometimes, the ledge can be bypassed, smoothed or rounded off by moving the instrument gently up and down.[52]

Preparation of a glide path includes initial scouting of the root canal with small and flexible, sometimes non-cutting instruments. A number of different instruments are available for scouting and preparation of a glide path (Table 20-1).

K-files (ISO-sizes 06, 08, 10): K-files ISO-sizes 06, 08, and 10 are regarded as the classical instruments for initial instrumentation of narrow and calcified root canals. They are used in a watchwinding motion with slight apical pressure. Negotiation of a narrow canal can sometimes require discarding several bent instruments before completion.

C-Pilot-Files: C-Pilot-Files are available for use in the sizes 06, 08, 10, 12.5, and 15 each with a 2% taper and a rectangular cross-section. They are manufactured by twisting and special hardening procedures in order to increase their stiffness and prevent early distortion and buckling of the tip, enabling the use of slightly increased apical pressure.

MMC Files: MMC files are available in ISO-sizes 06, 08, 10, and 15 with a 2% taper. They are manufactured from especially hardened stainless steel and show the same geometry as K-files.

S-Finder: The S-Finder instrument is manufactured from stainless steel and shows a geometry and design of a Hedstrom file with two cutting blades and an s-shaped cross-section. The size is ISO 10.

C+-Files: These instruments are by grinding stainless steel wires of a square cross-section. The apical 4 mm show a 4% taper to increase the stiffness of the tip; the coronal part shows a 2% taper to maintain flexibility.

RaCe: For automated preparation of a glide path, RaCe instruments size 10 with three different tapers were designed: .02, .04, and .06. These instruments present a non-cutting safety tip, a triangular cross-section, and alternating conical and parallel cutting areas, preventing a screwing effect. The surface of the instrument is electropolished, resulting in an increased resistance to torsion and fatigue.

Following hand preparation with instruments size 08, as deep as possible without pressure, a RaCe-file .06/10 is introduced one millimeter shorter. Near the apex, an instrument size .04/10 is used, and in severely curved canals a file size .02/10. These RaCe instruments are operated at 800 r.p.m.

G-files: The design of the G-files resembles that of the Revo-NiTi-instruments (Micro-Mega). Two sizes are available: .03/12 and .03/17. They are used following hand instrumentation with a size 10 file. Consequently,

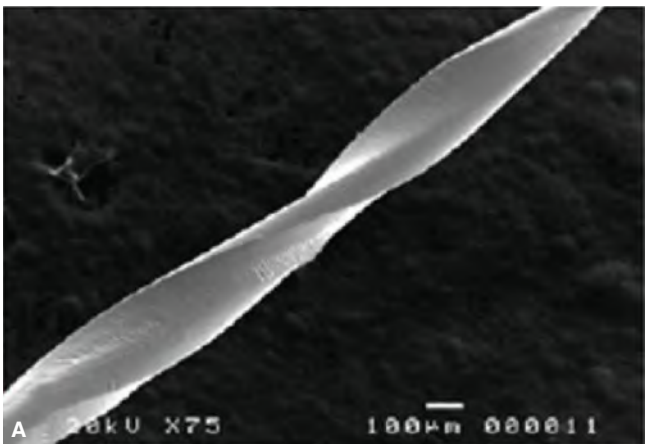
enlargement of the root canal to size 20 is facilitated. The instruments are operated at 400 r.p.m. with a torque of 1.2 N. The tip is rounded and non-cutting and the cross-section is variable across the length of the instrument (Figure 20-26).

PathFiles: PathFiles are available in sizes 13, 16, and 19 with a taper of 2% and a specially designed tip (Figure 20-27).

ProGlider: The instrument size is 016 with a 2% taper at the tip and progressive conicity up to 8.5%. The file is manufactured from M-wire and operated at 300 r.p.m. and a torque of 2 Ncm-5.2 Ncm.)

Summary

- Preparation of a glide path can reduce the incidence of fractures of rotary NiTi instruments, since reduced friction of the instruments is achieved.
- The final size of the glide path depends on the size of the first rotary instrument used for final preparation.



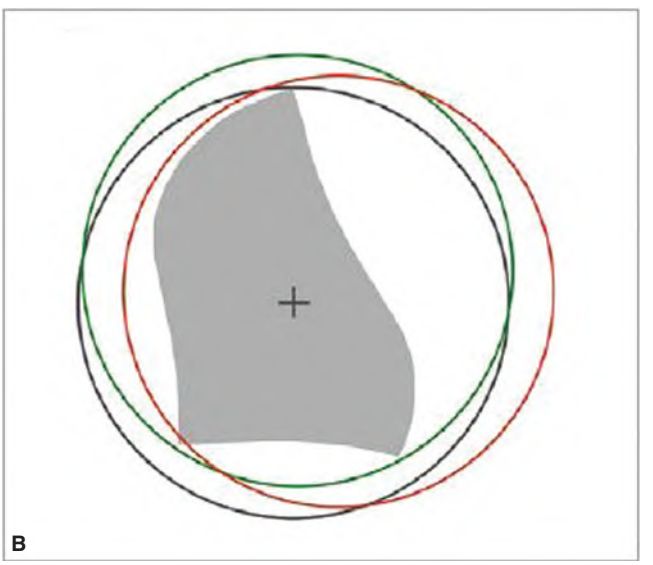


FIGURE 20-26 ***A-B.*** NiTi G-file (MicroMega) with asymmetrical cross-section.

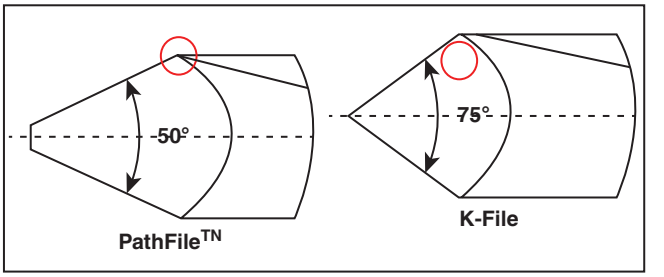


FIGURE 20-27 Differences in tip design of K-Files und PathFiles (Courtesy of Dentsply/Maillefer, Ballaigues, Switzerland.)

- Apical stiffness and high buckling resistance are important parameters for pathfinding files in order to prevent early and frequent buckling of the instrument tip.
- Stainless steel instruments can be used for preparation of the glide path as well as rotary NiTi files. NiTi instruments show slightly better results regarding straightening of curved or double curved root canals.
- Whether preparation of a glide path is mandatory before rotary preparation using NiTi instruments remains controversial.

Instruments for Root Canal Preparation

Hand Instruments

ISO standard 3630-1[53] and the ANSI/ADA Standards No. 28 and No. 58[54,55] specify general requirements of root canal instruments for manual use. These specifications cover design designations, dimensions and taper, resistance to fracture and bending, color coding, alloys, and identification symbols of the different types of instruments. In general, hand root canal instruments can be divided into three types: reamers, K-files and Hedstrom files. Today, the majority of hand root canal instruments are either made of stainless steel or NiTi alloys.

Metallurgy

Until the 1960s root canal instruments were made from *carbon steel* and since then mainly from *stainless steel*.[56] The main disadvantage of these older carbon steel alloys was their considerably low corrosion resistance. Sterilization procedures caused detrimental physical changes and severe corrosion damage.[56]

Stainless steel is considerably more resistant to sterilization hazards. Several studies failed to show any effect of sterilization on torsional and bending properties of stainless steel instruments.[57] Even up to 10 repeated sterilization cycles exerted no negative effect on the cutting efficiency of stainless steel instruments.[56,58]

Another alloy used for hand root canal instruments is *nickel-titanium* consisting of approx. 55% wt nickel and 45% wt titanium.[59,60] Due to the superelasticity of this alloy, NiTi instruments are made by grinding since it is impossible to twist a NiTi blank counterclockwise in order to produce a spiral.[61]

Design Features and Types of Instruments

According to the stipulated standards laid down in the ISO standard all ISO-sized hand instruments possess a constant taper of .02. This means that every millimeter of the cutting part the external instrument diameter increases by 0.02 mm from the tip towards the shank. Hence, engine-driven NiTi instruments with tapers different from the ISO-taper .02 are not covered in the ISO standard and are thus not ISO-sized.

In general, the following design features have a crucial impact on the clinical performance of root canal hand instruments (Figure 20-28):[62,63]

- *Cutting angle*: This angle, also called helical angle, is the angle between the instrument long axis and the tangent to the cutting edge (Figure 20-29).[61,62] The helical angle of instruments used for hand preparation indicates the most efficient working motion of the instrument. Instruments having a helical angle of less than 45° (reamers and K-files) require a rotary reaming working motion to effectively cut dentin, while those having an angle greater than 45° (e.g. Hedstrom files) require a linear filing motion.[61]
- *Tip design*: According to the ISO specification 3630-1, root canal instruments usually possess a sharp, cutting tip.[53] The ridges on the face of a cutting tip enable the tip to cut forward and produce a file-tip shaped cavity, thus creating ledges. Studies indicate that the tip configuration has a crucial impact on the shaping ability of root canal instruments. Currently, there is clear evidence, that independent of the alloy, instruments with modified non-cutting tips perform better in maintaining the original canal curvature than those having a conventional cutting tip.[64,65] These non-cutting tips are characterized by a reduction of the tip, transition angle and the incorporation of a guiding plane (Figure 20-30).[303,66] Instruments having non-cutting tips cause less canal transportation and remove material more equally at the inner and outer aspects of the curved canal compared to similar instruments having conventional tips.[67,68]
- *Pitch*: This is the distance between the edges of two cutting blades measured along the working part of an instrument (Figure 20-29).[62,69] The smaller the pitch, the greater the contact area between the instrument and the root canal walls, thereby increasing the torsional stress of the instrument.[62] The pitch may be regressive, continuous, or progressive.[21] Normally, hand instruments possess a continuous pitch. Only some Hedstrom files (e.g., those manufactured by VDW, Munich, Germany) have a progressive pitch length.
- *Configuration of cutting edges*: The cross-section of any root canal instrument determines the relevant cutting

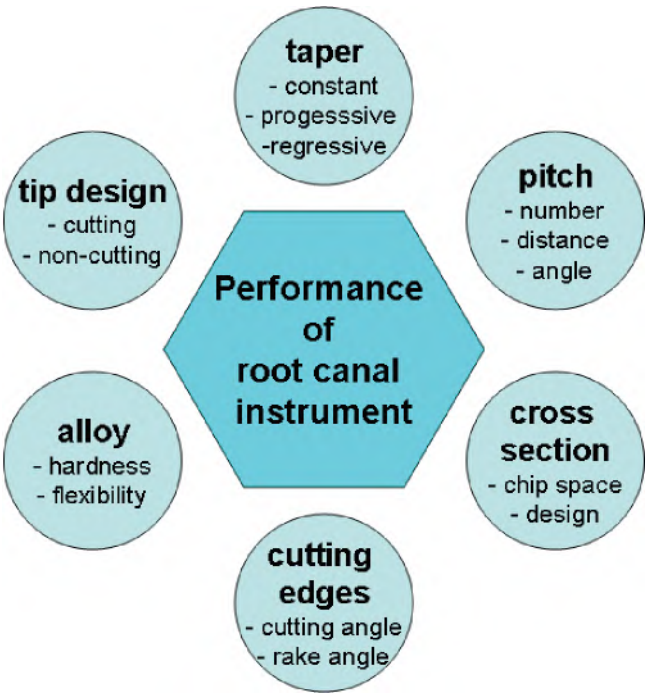


FIGURE 20-28 Factors affecting the performance of root canal instruments.

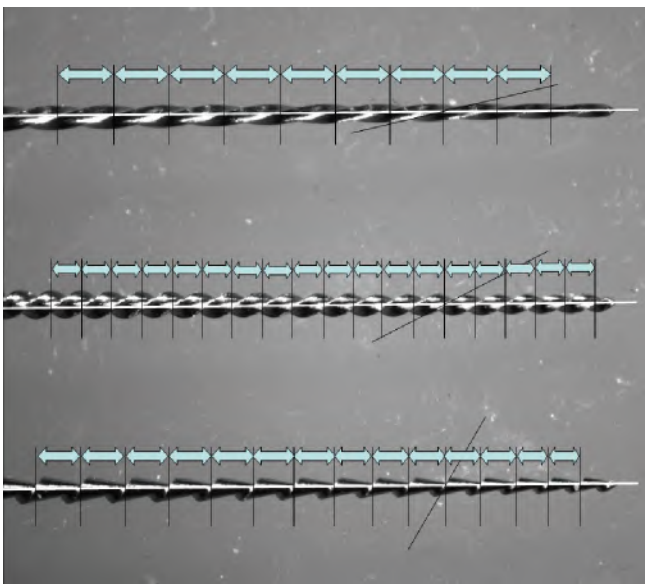


FIGURE 20-29 Helical angle of reamer, K-file and Hedstrom file with their pitch distance.

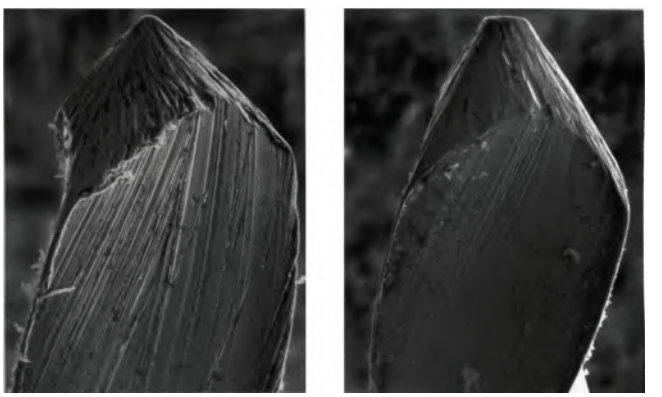


FIGURE 20-30 Scanning electron microscopy image of different tip designs. Left- conventional cutting tip; Right- modified non-cutting tip.

angles that are wedge angle, angle of cutting, clearance angle, and rake angle (Figure 20-31). Very small modifications of the cross-sectional shape may have serious consequences on instrumentation behavior. The wedge angle determines the strength of the cutting blades of the instrument. The greater the wedge angle the stronger the blade.[63] The clearance angle has an impact on the amount of the frictional force arising during the cutting process.[62] The smaller the clearance angle the less is the cutting efficiency. The sum of the wedge and clearance angle build the angle of cutting.[62] The rake angle is the angle between the cutting edge of the instrument and a perpendicular to the root canal surface at the point the cutting edge touches the canal wall.[62] The rake angle may be positive, negative, or neutral (nearly equal to the perpendicular). Instruments with a positive rake angle cut dentin very effectively by creating dentin chips.[62] A positive rake angle reduces the cutting forces. In general, Hedstrom files are characterized by a positive rake angle. However, nearly all root canal instruments requiring a rotary reaming working motion have a negative rake angle, such as reamers, K-file, and rotary NiTi instruments. Although a negative rake angle makes these instruments less effective they can be better controlled inside the root canal as their tendency to penetrate deeper into the root canal dentin is reduced (aka screw-in effect) compared to instruments having positive rake angles.[62] Moreover, a negative rake angle increases the strength and the wear resistance (longevity) of the cutting edges.

- *Chip space*: This is the difference between the area of the canal lumen and the area of the cross-section of the particular instrument used. The chip space is directly related to the cutting efficiency of any root canal instrument and its cleaning effectiveness. Simply stated, the greater the chip space the more efficient is the instrument in transporting dentin and debris out of the canal.[63,69] The greater the chip space, the smaller the cross-section of any instrument and the smaller the cross-section the greater the flexibility of the instrument.
- *Core diameter*: The core diameter of any root canal instrument affects its flexibility as well as its resistance to fracture.[61] A greater core diameter is associated with an increased resistance to fracture but at the same time with a decreased flexibility. The core diameter depends on the cross-sectional shape of the instrument. For hand instruments, the core diameter decreases from square, to triangular and further to S-shaped cross-sections. Usually, reamers, K-files and S-files are symmetrical in cross-section, while Hedstrom files are characterized by an asymmetrical cross-section.
- *Taper (conicity)*: The taper of ISO-sized hand instruments is .02 (or 2%). From the tip to the end of the working part, the diameter of the instruments increases by 0.02 mm every millimeter.[52] The taper may be regressive, continuous, or progressive.[62,70] The more tapered the instrument is, the faster the core diameter increases resulting in reduced flexibility.[70]

Reamers

Reamers are made from square or triangular blanks depending on manufacturer and ISO size.[61] In general, smaller sizes are made from square blanks to increase their core diameter thereby increasing the resistance to fracture. On the other hand, reamers of greater sizes are made from triangular blanks to increase their flexibility as the core diameter of a triangular blank is smaller than that of a square bank. Reamers made from stainless steel are twisted to give the working part of the instrument a spiral form. This is of clinical relevance as twisted instruments are *per se* more resistant to fracture than milled instruments, as the integrity of the blanks will not be damaged.[61]

Reamers have ½ to 1 cutting blades per millimeter in their working part, thus the number of cutting blades is less in the reamer than in the K-file (Figure 20-32).[61] This spiral form of the working part results in blade angles of 10° to 30°

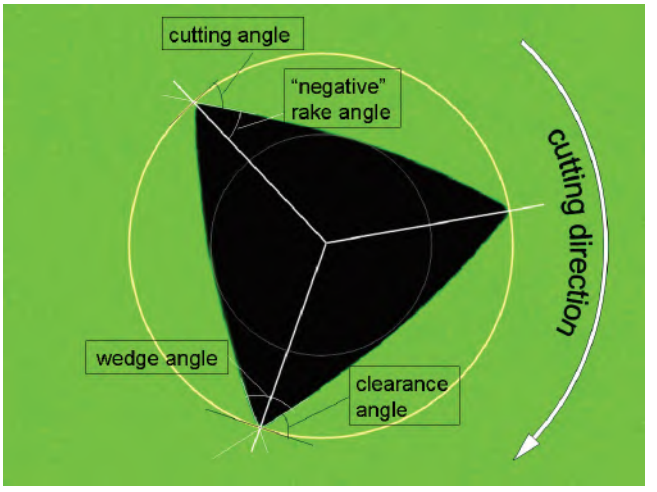


FIGURE 20-31 Cross sectional parameters illustrating the configuration of cutting edges.

FIGURE 20-32 Scanning electron microscopy image of a reamer (left) and K-file (right) showing that the K-file has more cutting blades per millimeter of the working part.

relative to the long axis (Figure 20-29).[61] Due to this angle configuration, reamers are primarily designed to be used in a reaming working motion.[61] They cut dentin by being inserted into the canal, twisted about one-quarter turn clockwise to engage their blades into the dentin, and then withdrawn.[61]

K-Files

K-files made from stainless steel are twisted instruments just like reamers. They are also made from square or triangular blanks, depending on their ISO size and manufacturer.[61] Compared to reamers, K-files show 1½ to 2½ cutting blades per millimeter of their working part, thus there are about twice the number of spirals on a K-file as on a reamer of a corresponding size.[61] The tighter spirals of a K-file establish cutting angles, greater than those of reamers. The cutting angle of K-files is about 25° to 40° (Figure 20-29), hence these instruments are, like reamers, primarily designed to be used in a rotary reaming motion.[61]

It is worth mentioning that although the term "K-files" implies that these instruments should be used in a filing working motion, however, they are by far more efficient when used according to their cutting angle in a rotary reaming motion. The term "file" denotes a cutting instrument that removes substrate by back-and-forth (linear) motions along the surface of the substrate.[62] Thus, the term "file" is incorrectly coined for this type of instruments.

On average, K-files display a greater resistance to bending than reamers. In most cases, K-files show greater angular deflection than reamers of the same brand.[66,71] The greater resistance to fracture of K-files has a direct clinical impact. The angular deflection provides some information about the susceptibility to fracture of an instrument. Hence, under clinical conditions, the risk of torsional fracture is lower for K-files as compared to reamers.[71]

Hedstrom and S-Files

Independent of the alloy used, the cutting blades of Hedstrom and S-files are milled into a round blank.[61] The flutes of the Hedstrom file constitute a spiral (screw shape) and the cross-section is nearly circular (teardrop cross sectional shape), while S-files possess a sigmoidal cross-section.[61] The flutes of S-files are less deep than those of Hedstrom files and S-files are characterized by two spiral grooves.[61]

The angle between the cutting edges and long axis of the Hedstrom files (cutting angle) is 60° to 65° (Figure 20-33) while that of S-files is generally smaller. Due to their cutting angle, both instruments are designed primarily for a linear (filing) motion.[61] Due to their positive rake angle they cut in one direction only, in a withdrawal stroke. Therefore, these instruments are in fact "files."

The manufacturing milling process of Hedstrom and S-files has two clinically relevant implications: (1) As Hedstrom and S-files are milled from circular cross-sectioned blanks, they have sharp cutting edges.[61] Used in linear back-and-forth working motion, they are by far more efficient than reamers or K-files as they cut away more root canal dentin per time unit than K-files.[72] Out of all the hand instruments designed to be used in a linear filing motion, Hedstrom files displayed the best cutting efficiency. The helical angle of Hedstrom files appears closer to 90° than that of S-files and this may explain the superior cutting efficiency of Hedstrom files in linear motion.[70,73] (2) Due to the manufacturing milling process, grinding cracks may result on the surface of the instrument. This fact, together with the relatively low core diameter of Hedstrom and S-files, explains why these instruments show an increased risk of torsional fracture compared to reamers or K-files.[71] In general, milled instruments like Hedstrom files are not recommended for use in narrow or curved canals due to their higher risk to fracture.[66]

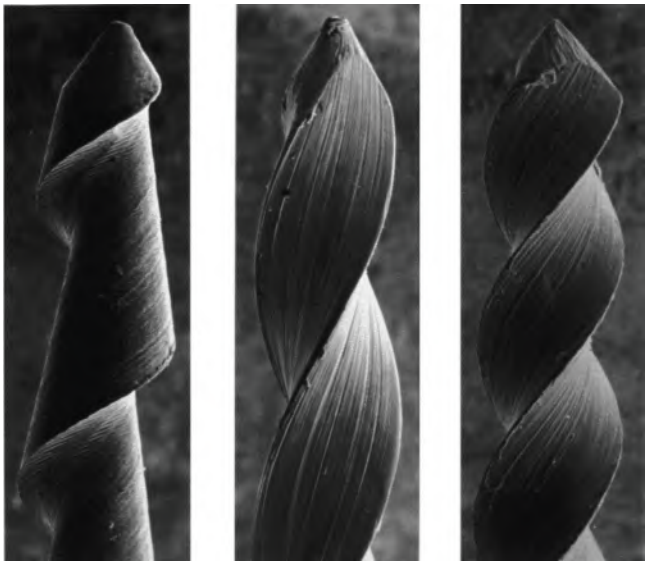


FIGURE 20-33 Scanning electron microscopy image of a Hedstrom file, reamer and K-file (from left to right) showing that the helical angle of the Hedstrom file is considerably greater than those of the reamer and the K-file.

Shaping Ability of Stainless Steel Hand Instruments

Reamers used in a reaming rotary working motion are able to produce a round, tapered preparation, when enlarging straight root canals.[74] However, in curved canals the use of reamers resulted in marked canal transportation, ledge formation and other undesirable deviations from the original path of the canal.[68,75] Currently, the sole use of reamers to prepare root canals is not recommended.[61]

Regarding the shaping ability of K-files, controversial results have been published. While some authors stated that shaping of curved canals with K-files, manipulated in a linear filing motion, resulted in good maintenance of the original canal curvature,[61,75] it was not corroborated by others. One study investigated *in vitro* the ability of K-files used in a linear filing motion to shape simulated curved canals in resin blocks.[76] K-files created pronounced zips and elbows and removed an excessive amount of resin from the outer aspect of the canal curves. These findings are in agreement with those obtained in another study that used extracted human molars.[77] Again, the use of K-files

was associated with marked canal transportation and procedural errors.[77]

Jungmann et al.[78] assessed the shaping ability of K-files when used in a reaming and linear filing motions. Results revealed that canal enlargement using a rotary reaming working motion proved to be better than a linear filing motion. In contrast, other authors stated that using K-files in a rotational cutting action was associated with distinct canal transportation.[68] Therefore, the combined use of these two types of instruments appears to be an effective technique for preparation of curved root canals.[74,79]

Clinically, the main advantage of reamers and K-files is that they can easily be pre-bent. Therefore, these stainless steel instruments are best suited for initial scouting of the root canal prior to full canal instrumentation. NiTi hand instruments are not suitable for probing and scouting due to their superelasticity and increased flexibility and since they cannot be adequately pre-bent.[80] Pre-bent reamers and K-files can be used for passive probing of the canal when used in a gentle watch-winding motion, thereby delivering sufficient tactile feedback. This tactile feedback is of clinical relevance since possible blockages of the canal (root canal branches or calcifications) can be assessed as well as the width of the canal lumen and the consistency of its contents. It is recommended that initial canal scouting and establishment of a glide path should be performed prior to canal preparation with engine-driven NiTi instruments, in order to reduce separation rate of engine-driven instruments.[81,82]

The shaping ability of Hedstrom files when used in curved root canals is controversial. While according to one study the use of pre-bent Hedstrom files resulted in good maintenance of the original canal curvature,[83] other authors recommended that Hedstrom files should not be used in the apical part of curved root canals.[76,84] This recommendation is based on the observation that in curved canals the filing motion of Hedstrom files resulted in a serve straightening of the inner canal wall as well as in excessive material removal on the outer side of the curvature.[76,84] According to extensive investigations conducted by Alodeh et al.[85] and Al-Omari et al.[84] these instruments created numerous procedural errors. Zip and elbow formations, as well as marked loss of working length, occasionally even complete blocking of the canals were common findings.[86]

On the other hand, Hedstrom and S-files are suitable and recommended whenever maximum removal of dentin in a short period of time is desired. From a clinical point of view, these instruments can be recommended for the preparation of straight root canals (occurring in 10% of cases[87]) or for initial conical enlargement of the coronal part of the canal.[61] Moreover, the use of hand Hedstrom files is beneficial following rotary NiTi preparation of root canals having an oval cross-section, or of canals with extensive isthmuses, Preparation of lateral canal extensions using either NiTi hand or engine-driven instruments is not satisfactory as these instruments are too flexible and cannot be properly directed into the extensions of the canal lumen (Figure 20-34).[80] These extensions can be easily cleaned and shaped using stainless steel Hedstrom files applying the circumferential filing technique.[88]

Flexible Stainless Steel Root Canal Instruments

Due to the unsatisfactory shaping results obtained with conventional stainless steel reamers and K-files, further developments led to the introduction of flexible stainless steel instruments characterized by higher flexibility while bending.[89,90] These instruments are similar in shape to conventional reamers and K-files but possess markedly improved properties.[85] Well-known representatives of these flexible stainless steel instruments are the Flexoreamer and the K-Flexofile (both manufactured by Dentsply/Maillefer, Ballaigues, Switzerland).

The Flexoreamer and the K-Flexofile, both having a non-cutting Batt-tip, are twisted instruments (Figure 20-35). When used in rotary reaming motion, the K-Flexofile and

FIGURE 20-34 Cross-section of a root canal showing that the extensions were not adequately cleaned and shaped.

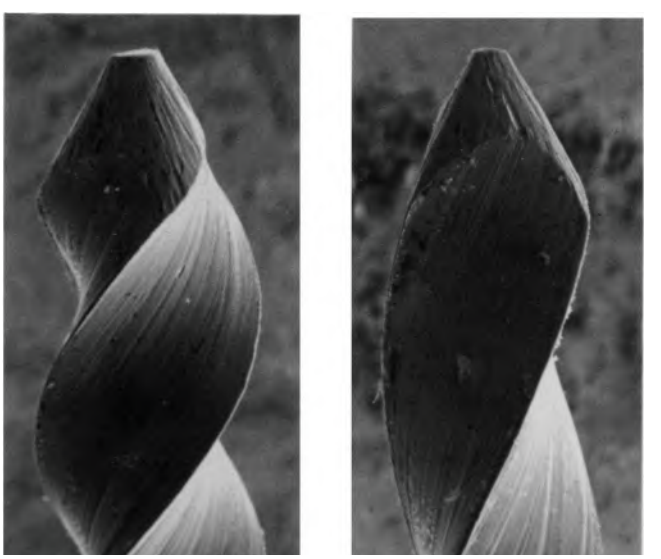


FIGURE 20-35 Scanning electron microscopy image of the flexible stainless steel Flexoreamer (left) and K-Flexofile, both having a non-cutting tip.

the Flexoreamer displayed the highest cutting efficiency of all hand instruments.[71] This increased efficiency can be explained by metallurgical improvements and by the fact that independent of the instrument size, the cross section is triangular, resulting in relatively low core diameters. It is due to the low core diameter that these instruments possess an enhanced flexibility compared to conventional stainless steel instruments.[71] Concerning resistance to fracture, flexible stainless steel instruments displayed less torque than most conventional stainless steel instruments whereas their angular deflection was similar to conventional reamers and K-files.[66,71]

Results of several studies suggested that flexible stainless steel instruments displayed less resistance to bending than conventional stainless steel reamers or K-files.[66,71] This property has a clinical impact since the tendency of flexible stainless steel instruments to straighten themselves in curved canals is much lower compared to conventional stainless steel instruments. There is evidence that these flexible instruments are superior to conventional stainless steel reamers and K-files in maintaining the original canal curvature.[68,83,84] Investigations conducted by Al-Omari et al.[84] demonstrated that shaping of curved canals with flexible instruments appeared to be superior to conventional stainless steel instruments (Figure 20-36).

Nevertheless, despite the decisive improvements associated with these flexible stainless steel instruments with non-cutting tips, satisfactory preparation of severely curved root canals using hand instruments is still challenging. Several studies pointed out that independently of the preparation technique used, even flexible stainless steel instruments with non-cutting tips failed to produce entirely satisfactory results in terms of adequate maintenance of the original curvature of severely curved canals.[68,89] Furthermore, even with these instruments, apical enlargement is limited to relatively small sizes, possibly jeopardizing adequate cleaning and sufficient irrigation of the most apical parts of the root canal.[68,83]

In summary, out of all stainless steel instruments for hand use, flexible stainless steel instruments are the most efficient and best suited to prepare curved root canals.

Nickel-Titanium Root Canal Instruments

Today, nickel-titanium instruments are mainly manufactured from a 55-NiTi-alloy, thus they consist of approx. 55% nickel and 45% titanium by weight.[59] This alloy is extremely flexible and NiTi instruments were shown to have about three times the elastic flexibility in bending and torsion as compared to corresponding stainless steel instruments.[58,69,71,90] NiTi hand instruments are available as reamers, K-files, and Hedstrom files.

The NiTi alloy exhibits superelastic behavior, thus upon unloading following deformation, this alloy returns to its original shape (Figure 20-37).[58,59,91] Superelastic alloys

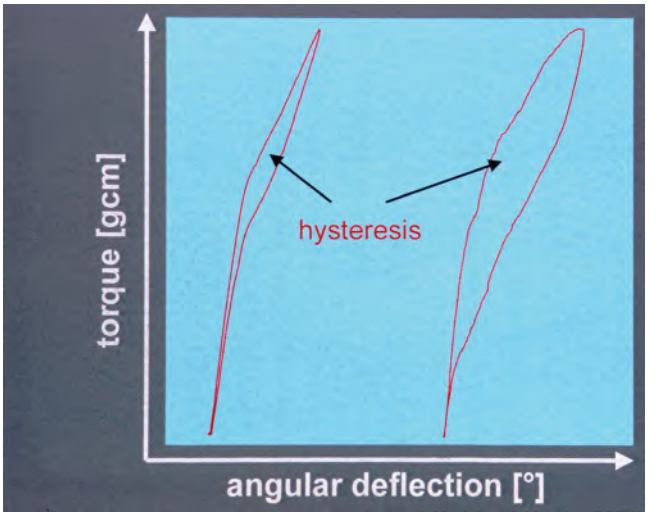


FIGURE 20-37 Nickel-titanium instruments fixed at their tips are rotated clockwise. Upon unloading, the instruments return to their original shape due to the superelastic behavior of the alloy.

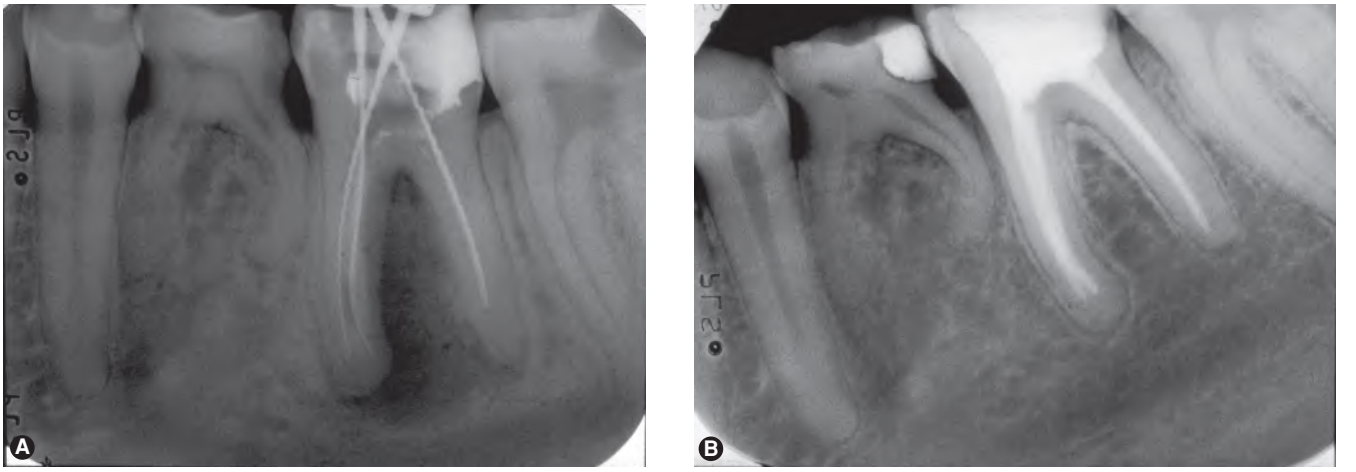


FIGURE 20-36 Flexible stainless steel K-Flexofiles were used for root canal preparation of the mandibular molar associated with peri- and intraradicular lesions. ***A.*** Working length radiograph. ***B.*** One year follow-up radiograph showing evidence of healing of the periradicular lesions.

undergo a stress-induced martensitic transformation from a parent structure that is austenite (Figure 20-38). Upon release of stress, the structure reverts back to austenite, recovering their original shape.[62] This superelastic behavior of NiTi occurs over a limited temperature range with an optimum at 37°C.[91,92] Clinically, the superelastic behavior is of relevance since NiTi instruments suffer no permanent deformation when being used in severely curved canals, whereas deformation of about 1% results in permanent deformation in stainless steel instruments (Figure 20-39). Because of their superelasticity, however, NiTi instruments cannot be pre-bent, making these instruments unsuitable to bypass ledges.[93]

As pointed out, stainless steel reamers and K-files are twisted instruments. Due to the superelasticity of NiTi, it is impossible to twist a NiTi blank counterclockwise in order to produce a spiral, since these alloys usually don't undergo permanent deformation (Figure 20-40).[50] More likely, the blank will fracture when being extensively twisted in order to produce a spiral. Therefore, all hand instruments made from conventional austenite NiTi are grinded instruments. However, grinding of titanium-based alloys is difficult since considerable wear of the milling head occurs within a short time.[63] This leads to structural defects on the surface, especially at the cutting edges of NiTi instruments (Figure 20-41).[58,59,94] These structural defects coupled with the considerable lower surface hardness, compared to stainless steel counterparts,[95,96] are probably responsible for their relatively low cutting efficiency. Cutting efficiency of NiTi K-files was found to be far lower compared to flexible stainless steel instruments.[71,73,97] Cutting efficiency in a linear filing motion of NiTi Hedstrom or S-files was only about 60% less than that of matching stainless steel files.[73]

While some studies showed corrosive tendency of NiTi instruments under simulated clinical conditions (Figure 20-42),[58,98,99] other studies failed to show it.[73,100,101] In addition, there seems to be evidence that

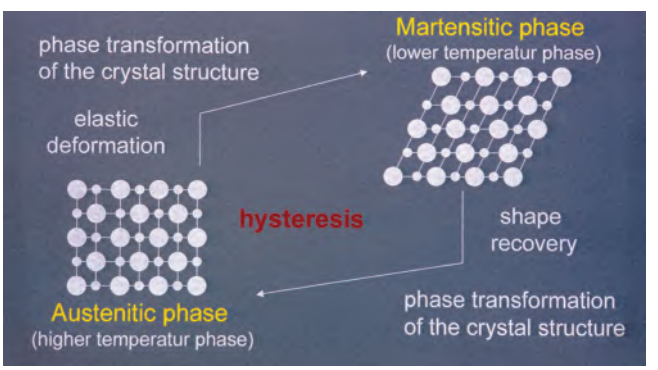


FIGURE 20-38 Stress-induced phase transformation of nickel-titanium from austenite to martensite.

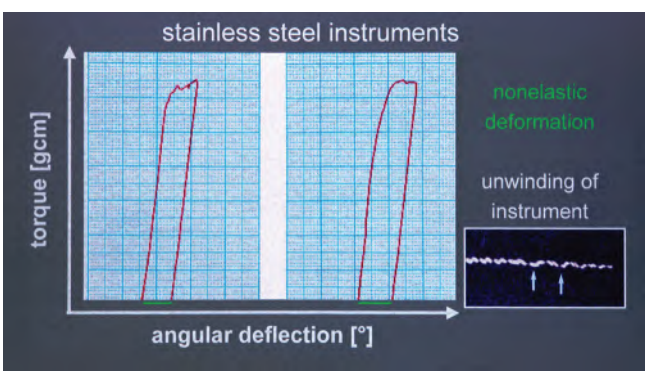


FIGURE 20-39 Stainless steel instruments fixed at their tips and rotated clockwise showing non-elastic permanent deformation.

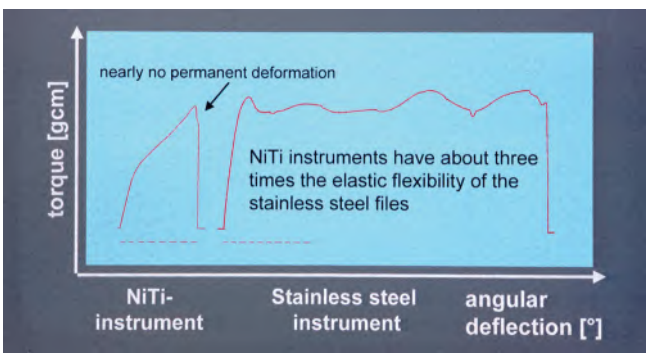
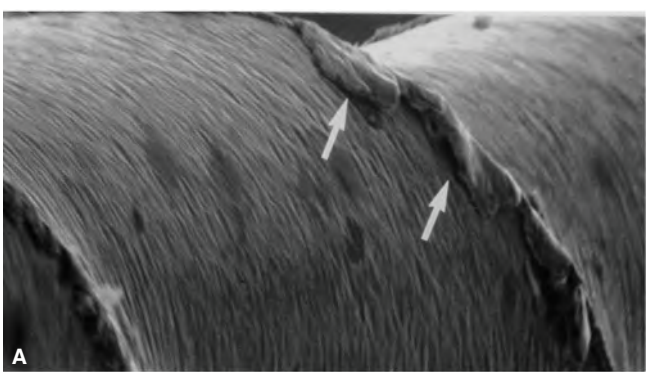


FIGURE 20-40 Nickel-titanium and stainless steel instruments fixed at their tips are rotated clockwise. While the stainless steel instrument exhibits a wide range of permanent deformation before fracture the nickel-titanium instrument shows nearly no permanent deformation.



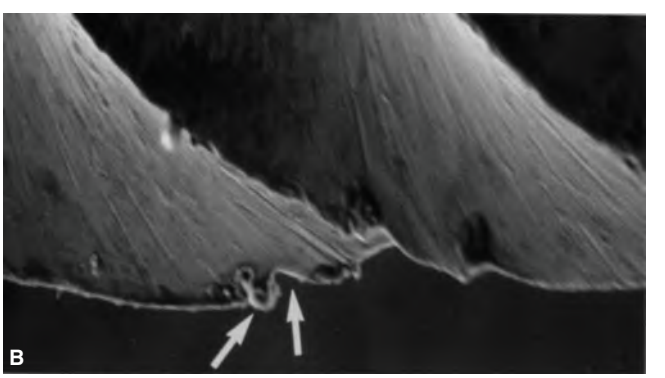


FIGURE 20-41 Scanning electron microscopic image of nickel-titanium hand instruments showing structural defects at their cutting edges (arrows).

FIGURE 20-42 Severe corrosion of a nickel-titanium hand instrument following sterilization.

repeated sterilization cycles have a detrimental effect on the cutting efficiency of NiTi instruments.

Regarding the shaping ability of NiTi hand instruments, studies using simulated curved canals in resin blocks reported a superior shaping ability for NiTi instruments in terms of increased centering ratio, less canal transportation, and less procedural errors compared to stainless steel counterparts.[102,103] These findings are in agreement with those obtained when using extracted teeth with curved root canals.[102] NiTi instruments were significantly more effective in maintaining the original path of the canal when instruments larger than size 30 were used.[104-107] NiTi K-files caused significantly less canal straightening and also the incidence of procedural errors was significantly reduced by the use of NiTi instruments.

One study, however, showed no significant difference regarding the shaping results in curved root canals when NiTi and stainless steel hand instruments were compared.[108] The curvature of 24–52°curved canals was equally maintained with either flexible stainless steel or NiTi K-files when used according to the circumferential filing technique. In this context Gambill et al.[109] stated "The reason that Ni-Ti instruments caused less transportation than stainless steel K-files when the same filing technique was used may not be due to increased flexibility of the Ni-Ti instruments, but rather to the decreased cutting efficiency of the Ni-Ti instruments."

In general, it can be stated that there is evidence that NiTi instruments, as compared to stainless steel instruments, allow for a faster, more efficient and centric preparation of curved canals (Figure 20-43).[104,109] NiTi instruments seem to prepare curved canals more centric and more circular than flexible stainless steel instruments.

Non-ISO-Sized Instruments for Manual Use

Further improvements led to the development of instruments that do not match the requirements regarding diameter and conicity laid down in the ISO and ADA specifications.[52-54]

Half sizes: The percentage increase in diameter from one ISO size to the other is considerably great in the range of smaller sizes. For instance from ISO size 10 to ISO size 15 the diameter of the instrument increases by 50%, whereas the increase from ISO size 50 to ISO size 55 is only 10% (Table 20-2). This unbalanced gradation might impede manual preparation of severely curved and/or calcified root canals. Therefore, some manufacturers introduced instruments with half sizes, having diameters that lay exactly, or nearly, in between two ISO sizes. For example, the flexible stainless steel Flexoreamer and K-Flexofile instruments (Dentsply/Maillefer, Ballaigues, Switzerland) are available in half sizes from no. 12 through no. 37, both having a non-cutting tip. These instruments are named "Golden Mediums."

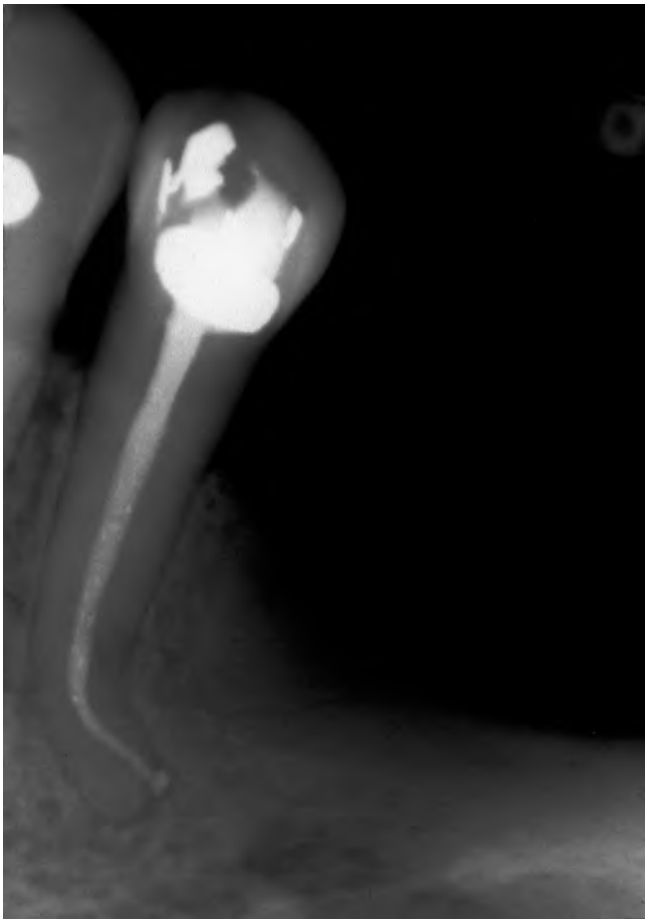


FIGURE 20-43 Severely curved root canal with a small radius of curvature enlarged using nickel-titanium hand instruments. The original curvature was well maintained.

Weine et al.[110] and Schilder[110] recommended the use of incremental instrumentation, especially in narrow and sclerotic root canals. According to these authors, to reduce undesirable instrumentation effects, intermediate increments should be employed. "*This is accomplished by cutting off one millimetre of the tip with sharp scissors and then using a diamond fingernail file to re-establish the bevel and the sharp edges from the tip.*" However, this procedure is time consuming and it is nearly impossible to produce a non-cutting tip with a diamond file.

TABLE 20-2 Gradation of ISO Sizes Showing That the Percentage of Increase in Diameter Is Considerably Greater in the Range of Smaller Instruments

diameter [mm]	increase in diameter [%]
0	
0,06	
0,08	33
0,1	23
0,15	50
0,2	33
0,25	25
0,3	20
0,35	16,7
0,4	14,3
0,45	12,5
0,5	11,1
0,55	10
0,6	9
0,7	16,7
0,8	14,3
0,9	12,5
1	11
1,1	10
1,2	9
1,3	8,3

Greater Taper Hand Instruments

With the introduction of rotary NiTi instruments having tapers greater than the ISO standard, some manufacturers also developed NiTi instruments with greater taper for manual use. For example, the ProTaper instruments (Dentsply/Maillefer, Ballaigues, Switzerland) are available as rotary and as hand instruments. Currently, studies assessing the properties of greater taper NiTi hand instruments are limited.

One study compared the shaping ability of ProTaper hand and rotary instruments used by inexperienced dental students.[111] The use of rotary instruments resulted in less canal straightening compared to hand instruments. In another study, the root canals of extracted premolars were instrumented with ProTaper hand instruments using the balanced-force technique and 3-D analysis using micro-CT was conducted to assess the percentage of unprepared canal surface.[112] The authors reported that 28%–83% of the root canal surfaces were not prepared by the instruments.[112] In another study,[113] the root canals of human mandibular molars were instrumented with ProTaper by hand, ProTaper rotary, and the rotary NiTi system RaCe (FKG, La Chaux de Fonds, Switzerland). Canal deviation in the apical third of the canals was evaluated by superimposition of pre- and postoperative radiographs. The incidence of canal deviations was 25% for ProTaper used by hand, 25% for rotary ProTaper and 20% for RaCe, respectively, with no statistically significant differences among the three instruments.[112]

In summary, it seems that the hand use of NiTi instruments with tapers greater than the ISO standard offers no significant clinical advantages and that these instruments should be preferably used as engine-driven instruments.

Conclusions

In summary it can be concluded that:

- Out of all stainless steel instruments, flexible stainless steel instruments, having non-cutting tips, show the best shaping ability. However, they do not produce completely satisfactory results, in severely curved canals, since apical enlargement is limited to relatively small sizes.
- Stainless steel Hedstrom files, used according to the circumferential filing technique, are best suited for cleaning oval canals and canal extensions.
- NiTi instruments respect the original canal curvature better than stainless steel instruments, especially in severely curved canals.
- Stainless steel pilot instruments are best suited for bypassing ledges.

PREPARATION TECHNIQUES

The endodontic literature describes many different techniques for root canal preparation, including manual (hand), automated, sonic, ultrasonic, lasers, and non-instrumental techniques (Table 20-3).[2,3] There are only a few comparative studies, making an evidence-based conclusion difficult.

Preparation and Obturation

When planning the preparation phase, the final obturation technique should always be taken into account since each obturation technique requires a specific shape of preparation. For example:

TABLE 20-3 Preparation Techniques Described in the Literature

Standardized	Ingle	1961
Step back	Clem	1969
Incremental	Weine, et al.	1970
Serial preparation	Schilder	1974
Telescopic	Martin	1974
Anticurvature filing	Abou-Rass, et al.	1980
Step-down	Marshall and Papin	1980
Crown-down	Goerig, et al.	1982
Double flare	Fava	1983
Crown-down pressureless	Morgan and Montgomery	1984
Balanced force	Roane	1985
Circumferential filing	Lim and Stock	1987
Canal master	Wildey and Senia	1989
Apical patency	Buchanan	1989
Apical box	Tronstad	1991
Progressive enlargement	Backman, et al.	1992
Modified double flare	Saunders and Saunders	1992
Non-instrumental (NIT)	Lussi	1993
Passive step back	Torabinejad	1994
Alternated rotary motions	Siqueira, et al.	2002
Reciprocal picking	Yared	2008

Single cone: The preparation shape should match the size of the Master Apical File (MAF); additional step back will reduce the fit of the cone.

Warm vertical compaction: A conicity of 7% or more is required to create a capture zone, preventing extrusion of the warm gutta-percha.

Lateral compaction: A constant conicity, exceeding the 2% taper of the instrument, is necessary to allow insertion of spreaders and thorough compaction of the gutta-percha cones.

Basic principle: Preparation should dictate the obturation technique rather than the obturation technique dictating a certain preparation technique.

Manual Preparation Techniques

Ingle[114] described the first formal root canal preparation technique known as the "standardized technique." In this technique, each instrument is introduced to working length resulting in a canal shape that matches the taper and size of the final instrument. This technique was designed for single-cone filling techniques using gutta-percha or silver cones.

Standardized Technique

This technique was first described by Ingle in 1961.[114] Starting with small instruments, all following instruments are inserted to working length. Preparation results in a root canal with low conicity that theoretically reproduces the shape of the last instrument used (Master Apical File [MAF]). The final shape of the preparation is suited, theoretically, for single cone obturation techniques as a high congruence between the MAF, the shape of the root canal and a matching gutta-percha cone is expected. Studies have shown that this congruence is rather conceptual than real.[115]

Step-Back Technique

The step-back technique[116] or similar techniques such as the telescopic technique or the serial technique aim at a higher conicity by stepwise reduction of working length, once apical preparation has been finished with the MAF (Figure 20-44). Each step back is followed by irrigation and recapitulation with a small file. The step-back technique has been for many years one of the traditional preparation techniques in endodontics. In some studies it produced cleaner root canals than standardized techniques.[116,117]

Circumferential Filing

Circumferential filing[118] aims at reducing the percentage of unprepared root canal walls by intentionally pressing the instruments against the walls. The instrument is inserted to working length, pressed against the canal dentin, and withdrawn. After the next insertion of the instrument to working length, it is pressed against the root dentin in some minor distance from the first file and again withdrawn in a shaping movement. Repetition results in circumferential preparation of the root canal dentin, theoretically touching the entire canal walls. This preparation results in a higher conicity than the techniques described above.

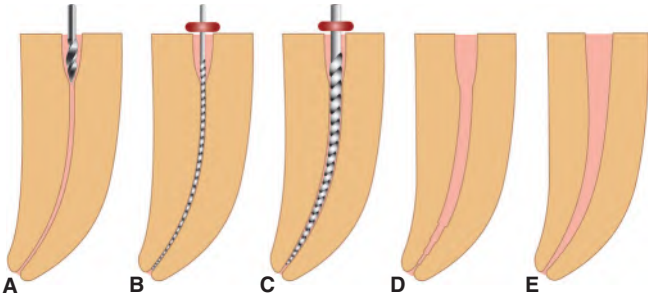


FIGURE 20-44 Sequence of instruments in the step-back procedure. After coronal pre-enlargement with Gates Glidden burs (**A**), apical preparation to the desired master apical file size commences with K-Files to determine working length (**B**) and then files of ascending size to the desired apical dimension (also called Phase I, **C**). Then, working length is progressively decreased ("stepped-back") by 1 or 0.5mm to create a more tapered shape (Phase IIa, **D**). Recapitulation with a small k-file is done to smooth canal walls and to ensure that the canal lumen is not blocked (Phase IIb, **E**). Frequent irrigation promotes disinfection and removal of soft tissue.

Anticurvature Filing

This technique[119] is designed to keep files away from the furcational zone of curved root canals thus preventing excessive removal of dentin and strip perforations at this critical zone.[120] This goal should be achieved by use of precurved instruments (files) at the outer side of the curvature.

Crown-Down Technique

This technique, first presented by Goerig et al.,[121] has gained new popularity with the introduction of rotary NiTi instruments. Following coronal flaring, large instruments are used to enter and enlarge the root canal a few millimeters apically each time. The instrument is followed by a smaller file, preparing only a few millimeters deeper than the previous instrument. This is repeated until working length is reached. As the final step, enlargement of the apical diameter completes the preparation (Figure 20-45).

The advantages of the crown-down techniques are:

- Less friction and stress on the instrument thereby reducing risk of instrument fracture
- Less canal straightening since reduced friction allows a more controlled preparation
- Less contamination of the apical zone of the root canal by reducing transportation of infected debris.
- Early coronal and mid-root enlargement allowing early disinfection of the coronal parts of the root canal system.
- Reduction of extruded debris beyond the apical foramen.

A slightly modified technique, termed as crown-down-pressureless, has been also suggested.[122]

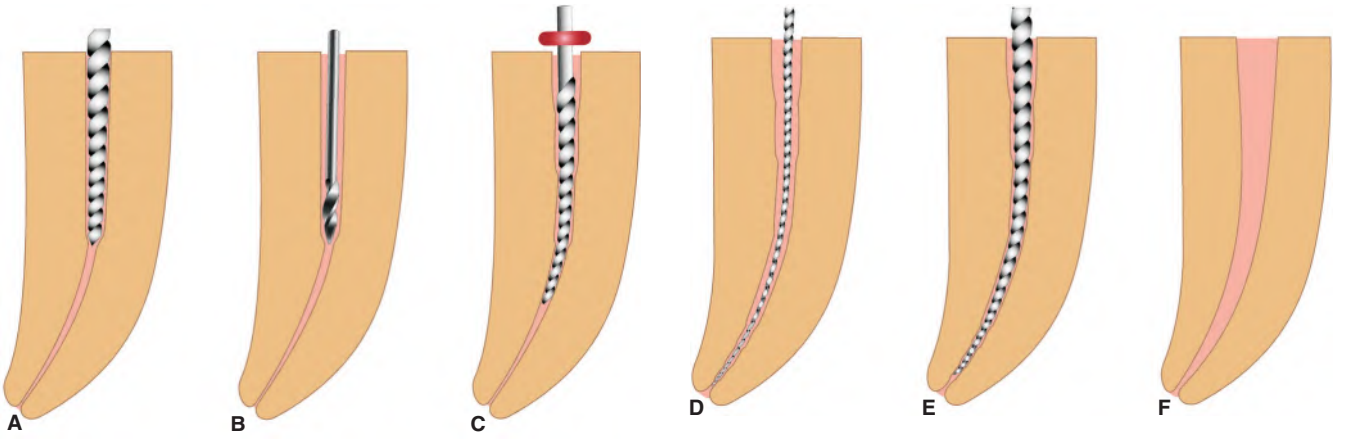


FIGURE 20-45 Sequence of instruments in the crown down approach. Coronal pre-enlargement was originally suggested to commence after determination of a provisional working length with a size #35 hand file (**A**). Then, Gates Glidden burs were used (**B**), followed by hand files starting with a large (e.g., size #60) file and progressing apically with smaller sizes (**C**). The definitive working length was determined as soon as progress was made beyond the provisional working length (**D**). Apical enlargement (**E**) and recapitulation created a homogenous shape that may be similar to the one created with the step-back approach, provided that both techniques were performed with little or no procedural errors. Both step-back and crown-down techniques may be used in conjunction with hand and rotary instruments but *in vitro* evidence suggests that a crown down approach is preferred for tapered rotary instruments. (Reproduced with permission from Blum JY, Machtou P, Micallef JP: Location of contact areas on rotary Profile instruments in relationship to the forces developed during mechanical preparation on extracted teeth. Int Endod J 1999;32:108.)

Step-Down Technique

With this technique the coronal part of the root canal is prepared first, resulting in more straight access to the middle and apical part of the root canal by early elimination of coronal interferences. Early disinfection of the coronal space is expected to reduce apical transportation of microorganisms, apical extrusion of debris and irrigant, and better control of apical preparation.[122]

Balanced Force Technique

The Balanced Force (BF) technique, described by Roane and Sabala in 1985,[123,124] uses specially designed instruments (Flex-R-Files) with non-cutting tips. The concept is to reduce the stress on the instruments thereby reducing the risk of straightening the canal and fracturing the instrument. The instruments are introduced until resistance is felt. The file is rotated at 180° in a clockwise direction (placement phase) with a slight but constant apical pressure, followed by a 120° rotation (cutting phase) in counterclockwise direction with maintained apical pressure, to prevent coronal displacement of the file (Figure 20-46). Finally the file is withdrawn in a clockwise motion (removal phase). The instrument is worked to working length in 1-2 mm steps with repeated constant clockwise and counterclockwise partial rotation, assisted by copious irrigation.

It has been claimed that BF preparation can achieve preparation sizes of 45 in curved canals and 80 in straight canals. The main advantages of the BF technique are good apical control of the file tip since the instrument does not cut over the complete length, and good centering of the instrument due to the non-cutting safety tip.

Good results for the preparation of curved canals without, or with only minimal, straightening[125] have been reported. However, incidence of procedural mishaps such as root perforations[126] or instrument fracture[127] have been described as well. The amount of apically extruded debris was lower than with stepback or ultrasonic techniques.[128] However, some earlier reports have indicated significantly more displacement of the root canal center axes, suggesting straightening.[129,130]

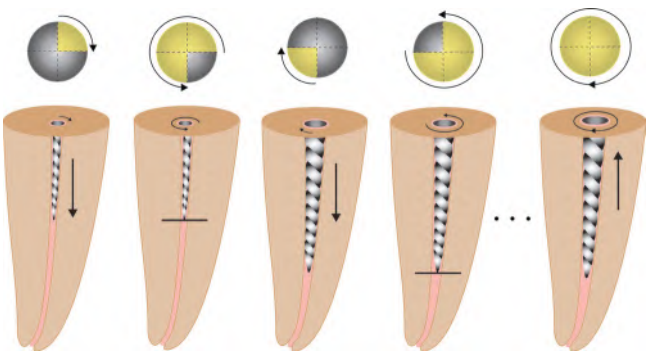


FIGURE 20-46 Principles of the Balanced Force technique. Instruments with a symmetrical triangular cross-section and pilot tips (*e. g.*, Flex-R files, Moyco Union Broach, Montgomeryville PA, USA) were originally suggested to be used in three steps in a rotational movement. A file may be advanced into the canal with a one-quarter clockwise rotation. The second movement involves adequate apical pressure to keep the instrument at this level of the canal while rotate counterclockwise for a half- to three-quarter turn. Currently, it is recommended to use the first two movements repeatedly, progressing more apically. Then the third movement pulls the instrument gently out of the canal with clockwise rotation.

Apical Box Technique

Tronstad[131] suggested preparing the root canal to a larger apical size using an apical box technique. Instead of using

a step-back approach, this technique, following preparation with a MAF, uses three or four larger instruments (K-files) in a filing and rotation action to full working length, thereby giving the apical 2-5 mm a cylindrical rather than a conical shape (shelf) (Figure 20-47).

Single Length-Technique

Single length technique has been recommended for some contemporary NiTi-systems (Mtwo, BioRaCe, Le-Chaux-de-Fonds, Switzerland). Starting with a small instrument, all instruments with increasing sizes are introduced to working length, followed by irrigation and recapitulation with a smaller instrument to working length. Instead of a step back, some NiTi systems use instruments with increasing taper thus increasing the taper of the canal preparation. This technique resembles the standardized technique.

Non-Instrumental Technique (NIT)

The non-instrumental technique (NIT), developed by Lussi et al.,[132] uses a vacuum pump and an electrically driven piston, generating alternating pressure and bubbles in the irrigation solution, inside the root canal. Finally, the root canal may be obturated with a sealer introduced by the vacuum pump.[133]

Studies demonstrated an equal or even better cleanliness compared to hand instrumentation in root canals of extracted teeth.[134] Some intraoperative complications such as severe pain, under-extension and apical extrusion of sealer or breakdown of vacuum have been reported.[135]

Since long-term clinical studies are not available yet, the NIT system cannot be considered a viable alternative to mechanical root canal instrumentation at this time.

Step-Back vs. Step-Down

Surprisingly, there are only few comparative studies assessing these approaches. Indeed, there is no evidence that step-down techniques are superior to step-back techniques or vice versa with the exception of the BF technique, that has been shown to result in less canal straightening.[127,136] In a comparative study of four preparation techniques, no difference between step-back and crown-down was detected in terms of straightening, but crown-down preparation produced more ledges.[137]

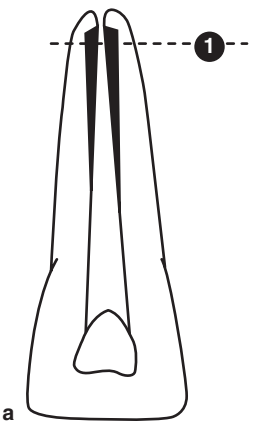


FIGURE 20-47 Schematic drawing of an apical box preparation.

Reciprocal Technique

In 2008, Yared[138] presented a technique using a ProTaper F2 file in a reciprocating motion. It is discussed later in this chapter.

AUTOMATED ROOT CANAL PREPARATION

Conventional Rotary Systems

The group of conventional rotary systems for root canal preparation comprises a number of endodontic handpieces differing in their working motion (e.g., rotation, partial rotation, reciprocal rotation, vertical movement, or a combination of rotation and up and down movement). In most of these handpieces, the motion of the file is rigid and cannot be influenced by the operator. The operator can vary the rotational speed and the frequency and amplitude of vertical strokes by changing the up-and-down movement of the handpiece. All these conventional handpieces were designed for use with stainless steel instruments, reamers as well as Hedstrom files.

In the 1980s, several handpieces with a more flexible working mode were introduced, namely the Excalibur handpiece (W&H; Bürmos, Austria) and the Canal Finder System (SET, Gröbenzell, Germany). Some of the new devices were equipped with integrated permanent supply of irrigant and permanent electronic determination of working length during preparation. Studies demonstrated that preparation of curved root canals often resulted in canal straightening.[139-145] Additionally, insufficient cleanliness[2,146] as well as apical blockages, loss of working length, perforations and instrument fractures also have been reported.[147,148]

In summary, these systems lost popularity after the introduction of rotary NiTi systems.

PREPARATION WITH ROTARY NiTi INSTRUMENTS

Development of Rotary NiTi Systems

Since their introduction, metallurgy, design, shape and philosophy of use of NiTi systems have changed frequently and to a considerable degree. Haapasalo and Chen[149] defined five distinct generations of NiTi instruments related to the evolution in design:

- Generation 1: These instruments include rotary NiTi systems with a constant taper and radial lands and a neutral or negative rank angle. Representative systems of this generation are ProFile, LightSpeed, Quantec, Greater Taper, BioRace, and EndoSequence.

- Generation 2: These instruments are characterized by having actively cutting blades without radial lands. Representative systems are ProTaper, ProTaper U, K3, M*two*, BioRaCe, FlexMaster, and S5.
- Generation 3: These instruments include systems such as Twisted Files, Typhoon, HyFlex, and Vortex Blue. The classical NiTi alloy was replaced withy improved, thermally pre-treated alloys such as M-wire or CM-wire, increasing the flexibility and durability of the files.[150]
- Generation 4: The mode of action of these NiTi systems was altered from a 360° rotation to reciprocal movements with Reciproc and WaveOne being the best-known systems of this generation.
- Generation 5: This most recent group of rotary NiTi instruments shows an irregular cross-sectional design. It includes LightSpeed LX, OneShape, SAF, Revo-S, and ProTaper Next. (Table 20-4).

NiTi systems cannot always be clearly assigned to a specific generation. Further developments in geometry and metallurgy, mode of rotation, and philosophy of use are expected in the near future.

NiTi Systems

NiTi instruments may differ significantly in design, treatment concept, and in their mode of use. Therefore, it seems reasonable to use the term NiTi systems rather than NiTi instruments. Except for the recently introduced single file systems, the majority of NiTi instruments are used in clearly defined (and mathematically designed) sequences. Some typical examples are presented in this chapter. Regarding the treatment concept, two approaches can be identified: crown-down and single length techniques.

Recently, single file systems have been introduced with the aim to of simplifying preparation, shortening time, and reducing costs.

TABLE 20-4 Generations of NiTi Systems According to Haapasalo and Chen[150]

1st generation	ProFile, GT
• 1992 McSpadden	Quantec
• 1994 B. Johnson	LightSpeed
.02, .04 and .06 taper, radial lands, pass, flutes	
2nd generation	ProTaper, K3
active flutes without radial lands	FlexMaster, BioRaCe
→ less instruments needed	
3rd generation	HyFlex, K3XF, Vortex
• 2007 M-wire	Vortex, Vortex blue
• 2010 R-phase	Twisted Files
• 2010 CM wire	
4th generation	Reciproc, SAF
• 2008 Yared	WaveOne
• 2011 Wave One, Reciproc reciprocal motion	
5th generation	OneShape
asymmetrical cross-section	Revo-S, PT Next

Single File Systems

Systems, with a single instrument designed to prepare the entire root canal, are Reciproc, WaveOne, F6 Sky Taper, SAF, One Shape, and OneEndo. These systems are discussed in a separate section of this chapter.

Single Length Systems

Single length systems are compatible to the standardized technique, where each instrument is introduced to working length, after preparation of the glide path. Some systems of this group are LightSpeed, M*two*, and BioRaCe. Each instrument is used to working length starting with small instruments and followed by files of higher taper or increasing diameter. Some systems such as BioRaCe have instruments with cutting tips each second instrument, whereas LightSpeed or M*two* have non-cutting tips.

Crown-Down Systems

Crown-down systems use a different preparation approach. Instead of inserting each instrument to working length at the beginning of the preparation, instruments with large diameter and high taper are used first. The following instruments gradually get smaller in taper and/or diameter (Figure 20-48). This technique, already known from manual root canal preparation, reduces the friction of the instruments inside the root canal. Each instrument has to enlarge only small parts of the root canal, resulting in shorter working time, reduced stress on the file and reduced risk of instrument fracture.

From a biological point of view, necrotic and infected material are removed first from the coronal part of the endodontic cavity and replaced by disinfecting irrigants, before the instrument is inserted deeper into the root canal. When working length has been reached, apical enlargement follows. Typical systems of this group are ProFile 04 and 06, ProTaper, and FlexMaster.

It should be noted that there is no evidence indicating that crown-down techniques, using NiTi instruments are superior, compared to single length techniques, with regard to relevant clinical parameters (shaping quality, safety of use, extrusion of debris, post-operative pain, clinical success). For hand instruments, it has been shown that this technique reduces the extent of straightening curved root canals,

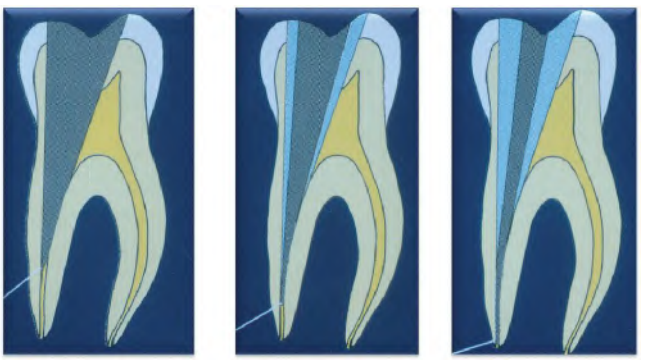


FIGURE 20-48 Systematic crown-down preparation.

reduces the risk of apical blockage and apical extrusion of infected debris.[151]

FIRST GENERATION SYSTEMS

LightSpeed, LightSpeed LX (LightSpeed Technology, San Antonio, Tx, U.S.A.)

Originally, LightSpeed was designed similarly to a Gates Glidden with a small non-cutting tip, radial lands and a long shaft.[152,153] Manufactured from NiTi, LightSpeed instruments were the most flexible instruments at the time. Each working tip showed an individual shape with a slow increase in tip length (0.25 mm – 2 mm), diameter and conicity from one instrument to the next. The instruments were initially used at 1,000 r.p.m. – 2,000 r.p.m. Later, the manufacturer has recommended a slower speed (Figure 20-49).

The principal of the LightSpeed technique was to minimize, as much as possible, the contact between instrument and dentin. Consequently, intermediate sizes were included, that further reduced the work load for each file. This philosophy resulted in a high number of instruments (more than 20), with an extremely short working time for each file. Due to its unique design, larger apical preparation could be achieved, even in severely curved canals.

Following apical preparation, a step-back procedure had to be performed in order to create sufficient conicity along the complete length of the canal allowing proper obturation. It has been recommended to obturate the apical part of root canals prepared with LightSpeed with a tapered 5 mm long gutta-percha cone attached to a metal carrier (SimpliFill).[152]

In a later version, called LightSpeed Xtra, the tip design was changed and the number of intermediate size instruments was reduced, making the file set considerably smaller. LightSpeed Xtra can be regarded as the first system of the fifth generation with an asymmetrical geometry.

FIGURE 20-49 Each LightSpeed file shows a unique tip with tip size, conicity and length increasing slightly from one instrument to the next.

ProFile .04 and .06 (Dentsply/Maillefer, Ballaigues, Switzerland)

ProFiles have been among the first systems to use crown-down preparation with rotary NiTi systems.[149,153] All instruments show a constant taper of 4% or 6%, respectively, and are used in a crown-down sequence at 300 r.p.m. (Figure 20-50).

K3 (Kerr/SybronEndo, Orange, CA, U.S.A.)

K3 instruments mark the transition from first to second generation. The file presents with radial lands as well as with actively cutting blades and a positive rake angle, a non-cutting safety-tip and variable helical flute angle preventing a screw-in-effect.[154]

SECOND GENERATION SYSTEMS

Mtwo (VDW Antaeos, Munich, Germany)

M*two* was one of the first NiTi systems with instruments designed as Hedstrom files. The instruments show two actively cutting flutes making the system very effective for dentin removal. All files of the set (04/10, 05/15, .06/20, .05/30, .04/35, .04/40) are used to working length with the largest instrument in size being .04/40 (Figure 20-51). Two additional sets .04/45, .04/50, .04/60 and .06/30, .06/35, and .06/40 are completing the system. If thermoplastic obturation is used, a final .07/25 instrument is also available.

RaCe and BioRaCe (FKG, La Chaux-de-Fonds, Switzerland)

The design of RaCe, an acronymon for **R**eamers with **a**lternating **C**utting **e**dges, shows alternating conical and parallel sections along the axis of the instrument, a convex triangle cross-section, sharp cutting edges and a chemically treated surface.[155] The system was followed by the more popular BioRace system. All instruments, except an orifice shaper, are used to working length: .05/15 (non-cutting tip) and .04/25

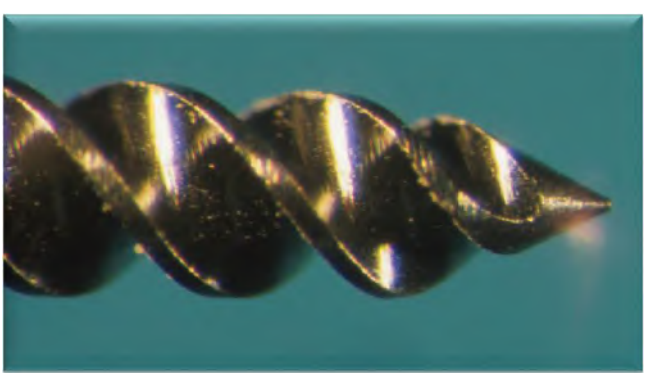


FIGURE 20-50 ProFile .04 showing radial lands and a non-cutting tip.

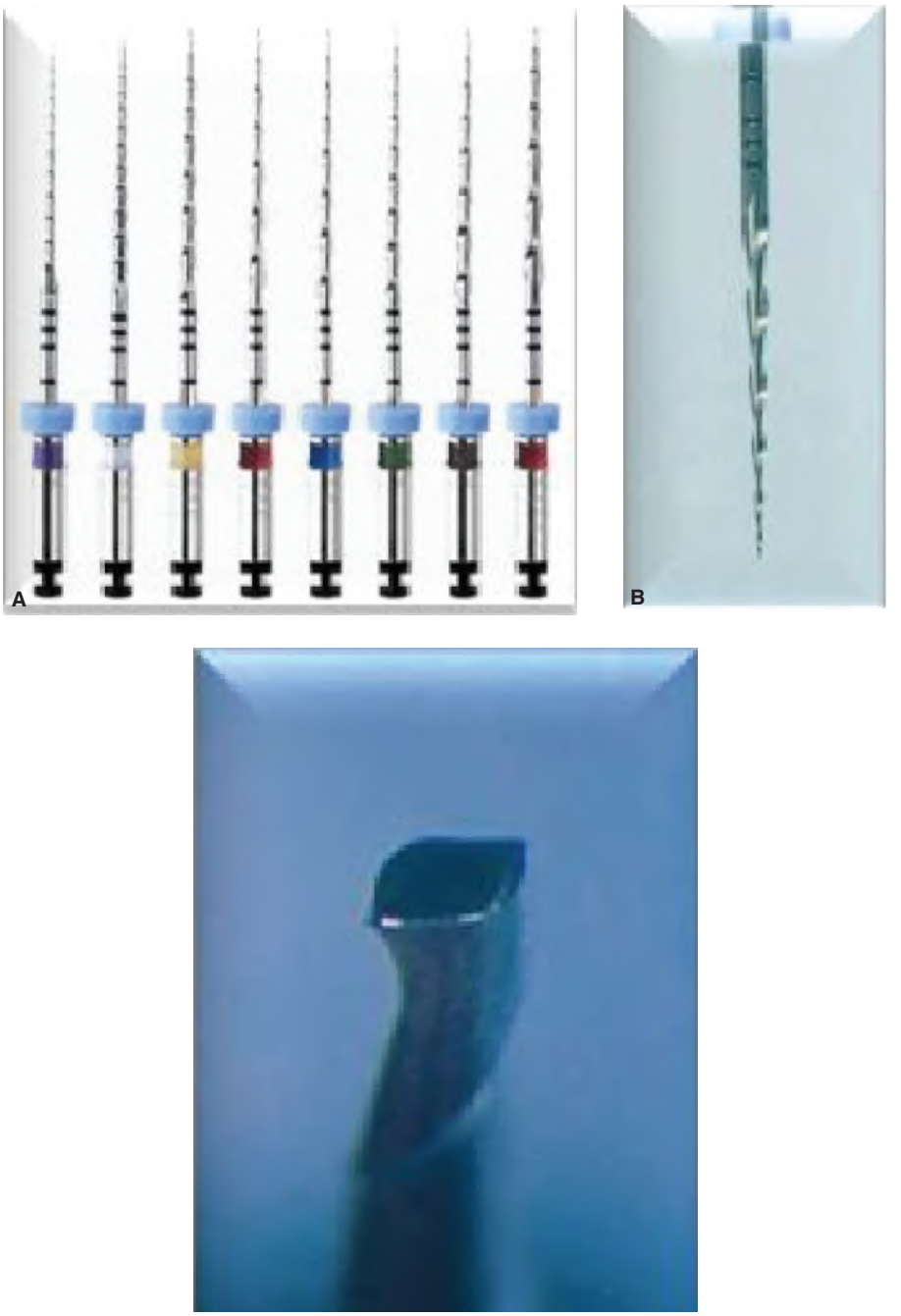


FIGURE 20-51 ***A.*** The set of *Mtwo* instruments. ***B.*** The design of *Mtwo* is similar to a Hedstrom file. ***C.*** Cross-section of *Mtwo*-files with two actively cutting blades.

(cutting tip) working in the most apical area; .06/25 (non-cutting tip) working in the middle and coronal third, 04/30 and .04/40 (cutting tips), working apically. In severely curved root canals, following size .06/25, two flexible instruments sizes .02/35 and .02/40 can be used (Figure 20-52). For large preparations additional sizes .04/50 and .02/60 are available.

BT RaCe (FKG, La Chaux-de-Fonds, Switzerland)

Booster Tip (BT) RaCe is a modified tip with a low taper. Three files with an atypical sequence of instruments are used for preparation: first a .06/10, followed by a non-tapered .00/35 and finally a .04/35 instrument. For larger root canals, .04/40 and .04/50 instruments are also available (Figure 20-53).

FlexMaster (VDW Antaeos, Munich, Germany)

The FlexMaster system comprises of instruments with three different tapers: 2%, 4%, or 6%, non-cutting tips and a triangle, slightly convex cross-section[156] and instrument sizes from 15 to 70, making it one of the systems with the largest variety of instruments (Figure 20-54).

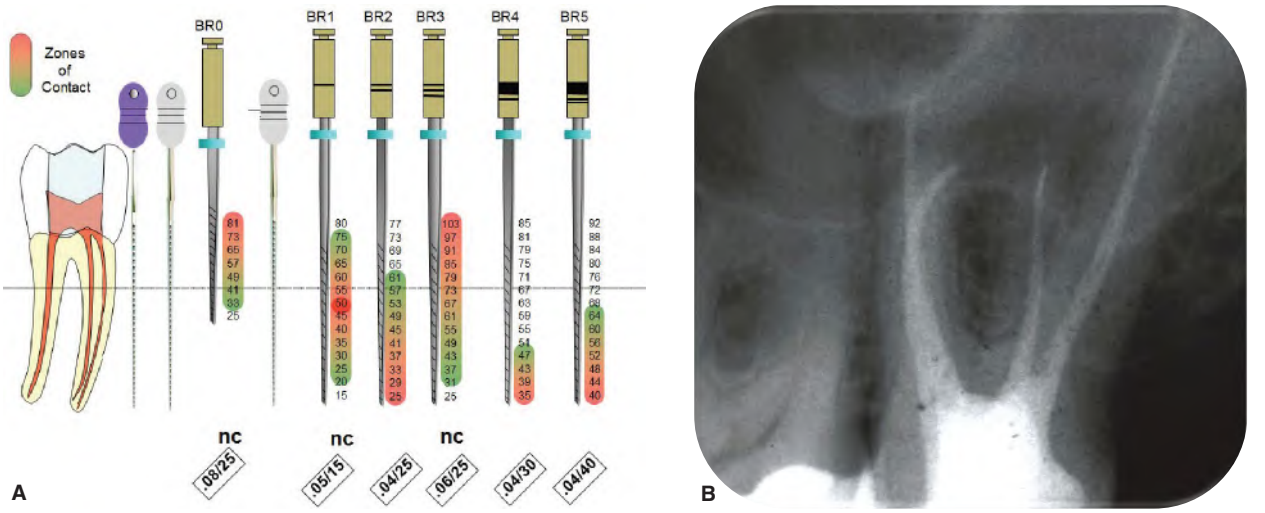


FIGURE 20-52 ***A.*** Regular sequence of BioRaCe instruments (ns: non-cutting tip). The red color marks the regions inside the root canal where the respective file is most active. ***B.*** Maxillary molar prepared with BioRaCe instruments to size .04/40.

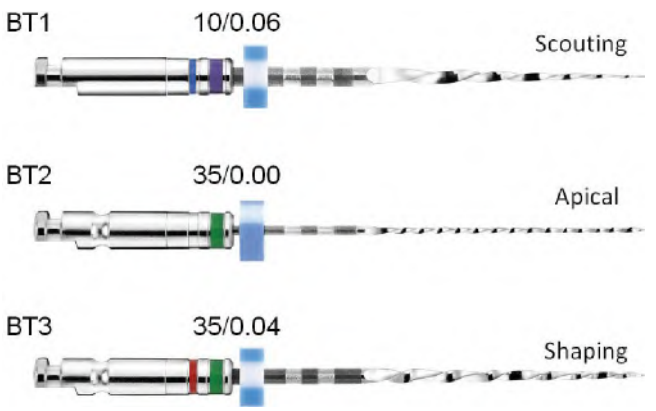


FIGURE 20-53 Sequence of the BT system. Note that the second instrument has a 0% taper.

The instruments are used in a crown-down-sequence starting with an Intro-File (9 mm cutting length, 11% taper) followed by instruments with a 6% conicity. Following each instrument, size or taper are decreased allowing a stepwise deeper penetration of the root canal. Size .02/ 20 has to reach working length, followed by 2% instruments with increasing diameter for apical enlargement. The instruments are used at approximately 300 r.p.m.

ProTaper and ProTaper Universal (Dentsply/ Maillefer, Ballaigues, Switzerland)

ProTaper instruments were the first to show different conicities along the shaft in one instrument. The system consists of six instruments:

- An Orifice shaper with a high conicity and a short cutting part;
- Two Shaping Files with small taper and small size at the tip and a high conicity (9%) near the shaft; and
- Three Finishing Files with increasing sizes (20, 25, and 30) and tapers up to 9% at the tip and 5% towards the handle (Figure 20-55).

This design results in limited contact areas between instrument and root canal dentin. In a later version, called ProTaper Universal, the design was changed. The cross-section was altered, making the file more flexible, the tip was rounded, making the instrument tip non-cutting, and additional sizes (35, 40, and 50, all with a 5% taper) were offered.[157]

THIRD GENERATION SYSTEMS

Third generation NiTi systems present modifications in the composition of the alloys that are thermally pretreatment, making the files more flexible[149,150] (see sub-chapter on metallurgy of instruments).

ProTaper Gold (Dentsply/Maillefer, Ballaigues, Switzerland)

The manufacturer's latest development, ProTaper Gold, shows increased flexibility due to undeclared alterations in the metallurgy of the alloy. The file design is the same as the PT Universal.

Twisted files (Kerr/SybronEndo, Orange, CA, U.S.A.)

Twisted files are manufactured by twisting pretreated M-wire NiTi alloy. The system includes five instruments with varying taper (.04 to .12) and a uniform tip size of 25.

TF adaptive are also manufactured from M-wire NiTi and produced by twisting thermically pretreated alloy.

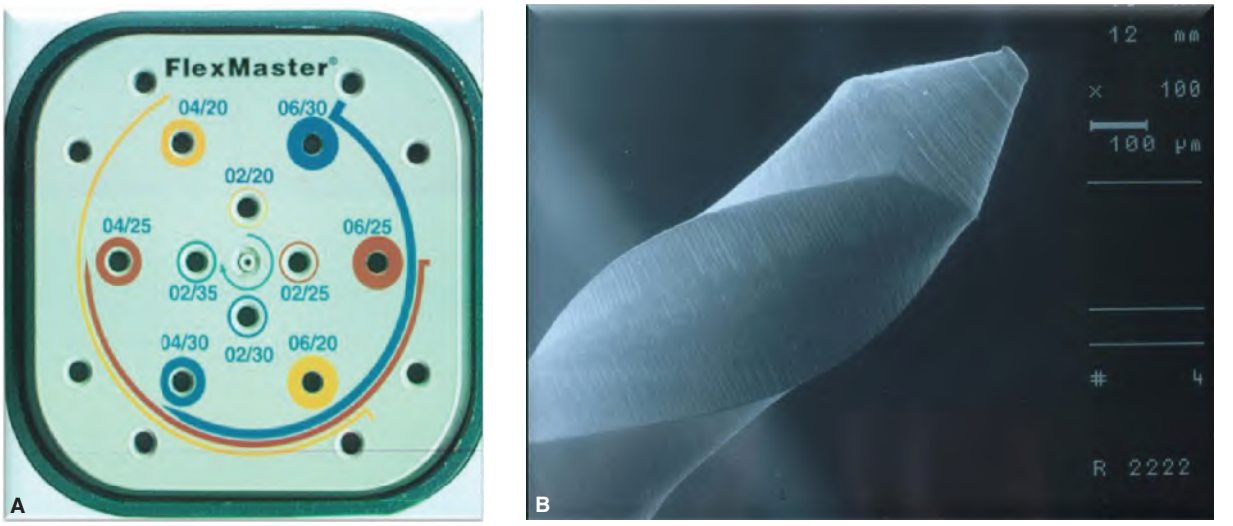


FIGURE 20-54 ***A.*** Basic box of FlexMaster with 10 instruments. More than 10 additional sizes (up to .02/70) are available. ***B.*** The instrument shows a non-cutting tip and a triangular, slightly convex cross-section.

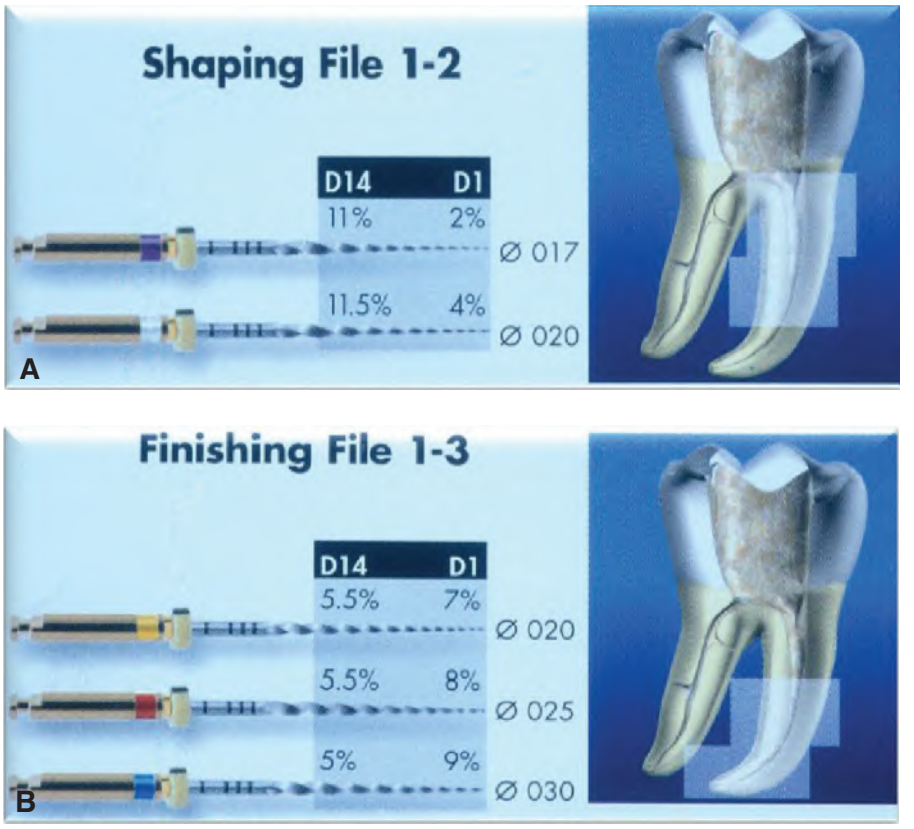


FIGURE 20-55 ***A.*** Sequence of ProTaper instruments. Shaping files have small tips, but a large taper and size at the upper shaft. ***B.*** Finishing files show increasing size and taper at the tip, with a smaller shaft. Instrument no. 6 is an orifice opener with a great taper and reduced working part.

Typhoon (TYP, Clinicians's Choice Dental Products, New Milford, CT, USA)

These instruments are fabricated from Controlled Memory (CM) wire, a thermally pretreated NiTi alloy that significantly increase the resistance to torsional failure.[158]

F 360 (Komet, Lemgo, Germany)

This system consists of two files sizes: .04/25 and .04/35, with two additional sizes for larger root canals (.04/45 and .04/55). The design is of double helix, similar to M*two*-instruments (F360). The instruments, manufactured from

conventional NiTi alloy, are used in a single-length technique with 300 r.p.m. A root canal can usually be prepared with two or three instruments.

F6 Sky Taper (Komet, Lemgo, Germany)

The company has introduced a corresponding One File-System: F6 Sky Taper.

The file has a taper of 6%, two blades, a Hedstrom file design and is used in full rotation (Figure 20-56).

FOURTH GENERATION SYSTEMS

TF adaptive (Kerr/SybronEndo, Orange, CA, U.S.A.)

The system includes two sets of instruments made from R-phase NiTi alloy: for small canals instrument sizes .04/20, .06/25 and .04/35, for larger root canals .06/25, .06/35, and .04/50 are available. Unfortunately, the color code does not keep to the ISO-code: green for the smallest file, yellow for the middle, and red for the largest one, resembling a traffic-light sequence. The instruments rotate in a clockwise direction up to 600°. In case of resistance, the forward rotation is reduced to 370°, followed by a 50° backward motion (Figure 20-57).

FIFTH GENERATION SYSTEMS

These instruments show a modified cross-section.

Revo-S (MicroMega, Besancon France)

These instruments show different cross-sections along the length of the instrument with three cutting blades at the tip and two blades towards the shaft. Files are available in sizes .04/25, .06/25, .06/30, .06/35, and .06/40.

FIGURE 20-56 F6 Sky Taper-instrument.

One Shape (MicroMega, Besancon France)

One Shape instruments also show different cross-sections along the length of the instrument with three cutting blades at the tip and two blades towards the shaft. The main instrument for preparing of the majority of root canals is a size .06/25. Additional two larger instruments (One Shape Apical) with sizes .06/30 and .06/37 are available for preparation of larger root canals.

ProTaper Next (Dentsply/Maillefer, Ballaigues, Switzerland)

ProTaper Next (PTN) presents with a rectangular cross-section and a non-cutting tip. The irregular cross-section increases space for dentinal debris and reduces contact of the instrument with the root canal wall (only a three-point contact is possible). The rectangular cross section directs the file into its optimal position in the root canal. The movement resembles a snake`s movement ("swaggering movement"). PTN is fabricated from M-wire. Instrument sizes are .04/17, .06/25, and for optional use .07/30, .06/40, .06/50. For glide path preparation, the manufacturer recommends PathFiles .03/13 and .02/15.

Mani Silk (Mani, Tohichi, Japan)

The Mani Silk system consists of three size instrument with an asymmetrical teardrop-shaped cross-section, fabricated from thermally pretreated NiTi. Three different sequences can be selected: .08/25, .06/25, and .06/30 for straight root canals; .08/25, .06/20, and .06/25 for moderately curved; and .08/25, .04/20, and .04/25 for severely curved canals.

The selection of the systems presented does not cover the entire range of available systems. Development of new systems with improved designs and metallurgy still continues.

Studies on Root Canal Preparation Using NiTi Systems

It has been demonstrated that NiTi instruments are safe to use provided attention is given to some basic principles:

FIGURE 20-57 TF Adaptive instruments work with a full rotation of maximum 600 degrees if no friction is present. Under friction, the rotation is changed into a 370° clockwise and 50° counterclockwise motion.

Cleaning Ability

Studies showed that the majority of NiTi systems are unable to completely instrument and clean the root canal walls.[159] No NiTi system (SAF, K3, WaveOne) was able to sufficiently clean and prepare all the isthmi.

Shaping

The majority of studies describe good maintenance of curvature even in severely curved root canals. Also, good centring ability with only minor deviations from the main axis of the root canal has been reported.[159-163] No difference in transportation of the foramen was detected between single file WaveOne system and multi-instrument Twisted Files.[27] Care should be taken using large tapered and large-sized instruments such as ProTaper F3 and F4 in curved root canals.[164]

No significant difference in straightening of curved root canals was detected between several NiTi systems (OneShape, ProTaper Next, Reciproc, Twisted Files, ProTaper Universal),[165] and no uniform results were reported suggesting superiority of any particular system.[166]

No difference could be detected between instruments made from conventional NiTi alloy (Revo-S, RaCe, Mtwo, ProTaper Universal) and instruments manufactured by innovative processes (Twisted File, GT series X).[167]

Instrument Fractures and Working Safety

Anatomical conditions such as radius and angle of root canal curvature, frequency of use, torque setting, and operator experience are important factors that affect instrument fractures. Selection of a particular NiTi system, sterilization and rotational speed, when confined to specific limits, seem to be less crucial. Apical blockage and loss of working length have been described in some cases, whilst perforations only rarely occurred.

Working Time

Working time with NiTi systems was significantly shorter with rotary preparation than with hand preparation. Use of single file systems further shortened the time required to complete the preparation.[162]

Conclusions

In summary, it can be concluded, that

- The use of NiTi instruments results in less straightening and better centered preparations, even in severely curved canals, than when stainless steel instruments are used.
- In order to reduce the risk of instrument fractures, a motor with constant speed and controlled torque should be used.
- Good preparation shapes can be achieved with any rotary NiTi system presently in the market.
- Some progress has been made in machining instruments, flexibility, maximum frequency of use, number of instruments needed, decrease of working time and some other technical details.
- Until now, it has not been demonstrated that new file systems, or advanced generations of NiTi systems, resulted in increased clinical success rates.

SINGLE-FILE SYSTEMS

The latest generation of engine-driven root canal instruments are single-file systems. These systems can be subdivided into those using a full clockwise rotary motion (F360 and F6 Sky Taper [both Brasseler, Lemgo, Germany] and OneShape [Micro Méga, Besançon, France]) and those using a reciprocation working motion (Reciproc [VDW] and WaveOne [Dentsply Maillefer]) (Figure 20-58). These single-file systems are claimed to be able to completely prepare and clean root canals with only one instrument. All single-file instruments have a non-cutting tip (Figure 20-59).

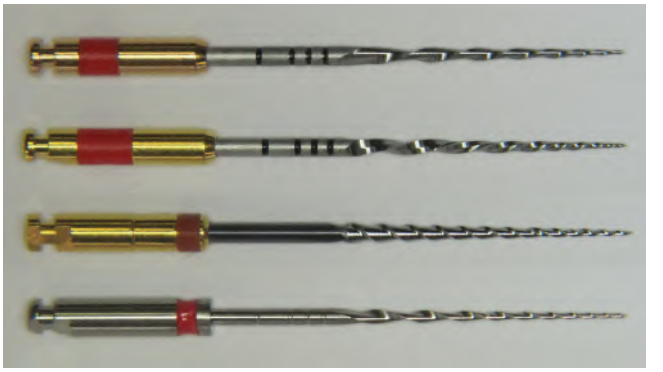


FIGURE 20-58 Different single-file instruments. From above: Reciproc, WaveOne, OneShape and F360.

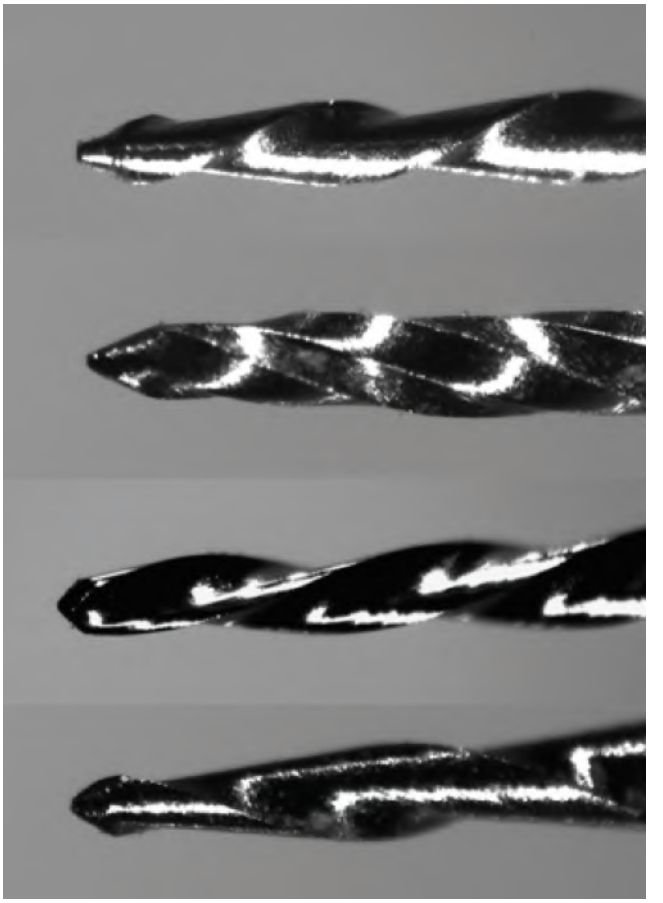


FIGURE 20-59 Tip configuration of different single-file instruments. From above: Reciproc, WaveOne, OneShape and F360.

Reciprocating Single-File Systems

Two instruments, both made from M-wire alloy, belong to this group: Reciproc (VDW) and WaveOne (Dentsply Maillefer). Reciproc instruments are available in different sizes 25.08 (R25), 40.06 (R40), 50.05 (R50), and WaveOne are available in the sizes 21.06 (small), 25.08 (primary), and 40.08 (large) (Table 20-5). Out of these instruments, only the WaveOne size 21.06 has a constant taper while all other instruments are characterized by a regressive taper. Only the first three millimetres of the working part from the tip of the instrument have a constant taper of .08 followed by a continuously decreasing taper. The final tapers near the shaft are .055 for the WaveOne primary, .045 for the WaveOne large, .043 for Reciproc R25 and .04 for Reciproc R40 and R50, respectively.

Reciproc instruments have an S-shaped cross-section along the entire working part of the instruments (Figure 20-60).[168] This cross-sectional design results in sharp cutting edges and a small core diameter with a comparable great chip space. In contrast, WaveOne instruments are characterized by different cross-sectional designs. Near the tip, the cross section shows radial lands, while in the middle part of the working length and near the shaft the cross-sectional design changes from a modified triangular convex cross section with radial lands to a neutral rake angle with a triangular convex cross section (Figure 20-60).[168]

The reciprocating working motion consists of a counter-clockwise (cutting direction) and a clockwise motion (release of the instrument), whereby the angle of the counter-clockwise direction is greater than the angle of the reverse direction (Figure 20-61). The actual angle of

TABLE 20-5 Characteristics of Single-File Systems

	Working motion	Tip design	Cutting edges	Cross-section	Alloy	Torque (Ncm)	Taper	Sizes	Glide path
Reciproc	reciprocating	non-cutting	2	S-shaped	M-wire	NA	regressive	25.08 40.06 50.05	not required
WaveOne	reciprocating	non-cutting	3	#	M-wire	NA	21.06: constant regressive	21.06 25.08 40.08	Size 10
F360	full rotary	non-cutting	2	S-shaped	austenite NiTi	1.5	.04 constant	25.04 35.04 45.04 55.04	Size 15
F6 Sky Taper	full rotary	non-cutting	2	S-shaped	austenite NiTi	1.5	.06 constant	20.06 25.06 30.05 35.06 40.06	Size 10
OneShape	full rotary	non-cutting	tip and middle 2; near the shaft 2	tip and middle: modified triangular; shaft: S-shaped	austenite NiTi	4	.06 constant	25.06 30.06 37.06	Size 15

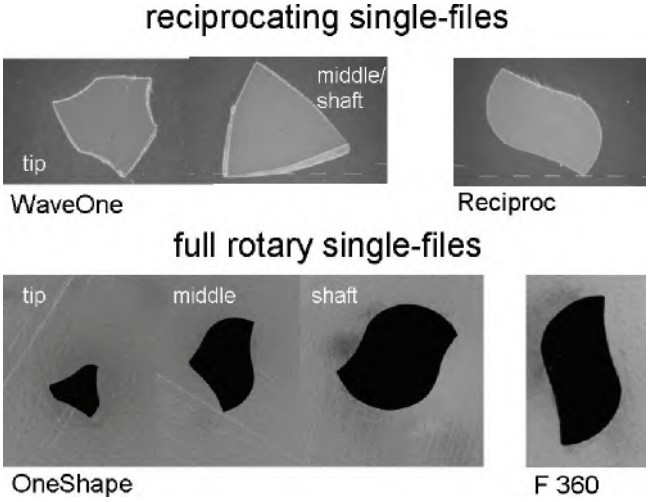


FIGURE 20-60 Cross-sectional designs of reciprocating and full rotary single-file instruments.

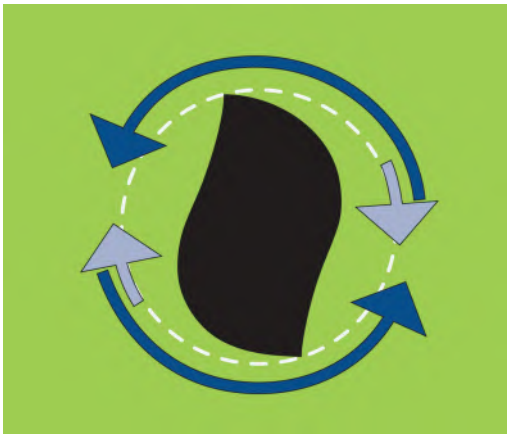


FIGURE 20-61 Schematic illustration of the reciprocating working motion. The angle of the counter-clockwise direction (cutting action) is greater than the angle of the reverse direction (release of the instrument).

counter-clockwise rotation is 158.68° for Reciproc and 159.85° for WaveOne, while the angle in clockwise direction is 34.65° for Reciproc and 41.44° for WaveOne.[169]

The actual cycle rotational speed is 282.92 r.p.m for Reciproc and 343.36 r.p.m for WaveOne.[169] However, these values strongly depend on the type of motor used. It should be noted that the actual values may differ from those set by the manufacturer.[169] Due to the fact that the angle of the counter-clockwise motion is greater than the angle in clockwise direction, the instrument progresses continuously towards the apical end of the root canal. It is important to note that the angles of reciprocation are specific to the design of WaveOne and Reciproc instruments and are programmed in special electronic motors.[169] It is therefore mandatory to use a motor that is able to generate the required counter- and clockwise angles. Reciproc and WaveOne instruments are not suited for sterilization processes since they do not fit into the handpiece again. Therefore, these instruments can only be used for one patient.

Clinically, it is important to consider that these instruments should not be used in a single straightforward motion up to the apical end of the root canal. Instead, the instruments should be used in a slow in-and-out pecking motion with amplitude of less than 3 mm. One in-and-out movement is defined as one peck. Following three pecks, the instruments should be removed, the canal copiously irrigated, the flutes of the instrument cleaned and the canal scouted with a small hand instrument, to avoid canal blockage with debris or dentin chips. Thus, the handling of these reciprocating instruments should be based on a sequence of three pecks where with every peck the instrument progresses deeper into the canal.

Some studies suggest that the use of reciprocating single-file systems is associated with little to nearly no canal transportation, even when preparing severely curved canals (Figure 20-62).[168,170-172] A micro-computed tomographic evaluation found that the use of Twisted Files Adaptive in severely curved root canal of extracted human mandibular molars resulted in less canal transportation and better centering ability than Reciproc and WaveOne.[173]

It appears that these systems considerably reduce the working time for canal preparation compared to other engine driven NiTi systems.[168,170,174] The use of these reciprocating instruments was safe, and fractures were not noticed.[168,171] Data about the cleaning efficiency of reciprocating instruments is controversial.[170]

Regarding cyclic fatigue of reciprocating single-files, several studies suggested that the reciprocating single-file instruments displayed greater flexural fatigue resistance than full-sequence rotary systems.[175,176] In general, Reciproc showed better torsional resistance and less bending resistance than WaveOne.[177,178] The superior behavior of Reciproc might be attributed to the smaller core diameter of these instruments compared to WaveOne and the fact that for Reciproc, the angles of reciprocation are smaller than those of WaveOne. Similarly, the speed of reciprocation is lower for Reciproc than for WaveOne.[168,170] However, the damage that these instruments may cause to dentin is still controversial.

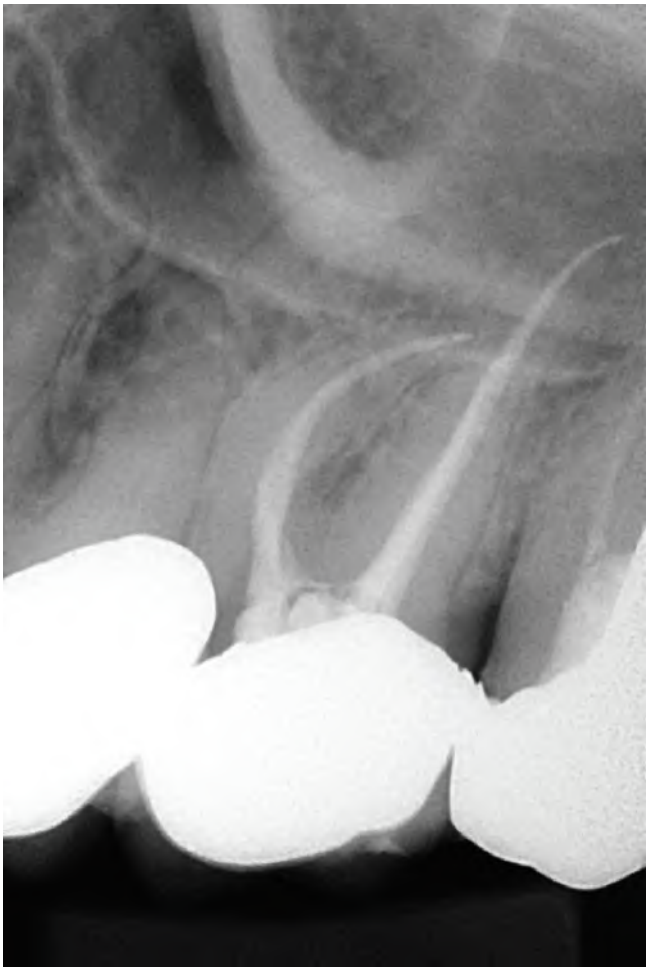


FIGURE 20-62 Adequate root canal preparation without canal straightening. The canals were prepared using a reciprocation single-file system.

Full Rotary Single-File Systems

The single-file systems F360 and F6 Sky Taper (both Brasseler, Lemgo, Germany) and OneShape (Micro Méga, Besançon, France) operate in a full clockwise rotational motion and can be used in every torque-controlled motor and handpiece (Table 20-5). Currently, no data are available regarding the F6 Sky Taper instruments, since these instruments were launched only recently.

All F360 instruments possess a constant taper of .04 and the following sizes are available: 25, 35, 45, and 55.[179] A modified S-shaped cross-sectional design is used for the entire working part of the instruments (Figure 20-60) and the instruments are made of a conventional austenite NiTi alloy.[179] OneShape instruments are available in sizes 25, 30, and 37 with a constant taper of .06. The instruments are made of conventional austenite NiTi and are characterised by different cross-sectional designs over the entire length of the working part (Figure 20-60).[179] In the tip region, the cross section represents three cutting edges while in the middle, the cross-sectional design progressively changes from a three-cutting-edge design to two cutting edges. Near the shaft, the instruments possess an S-shaped cross section with

two cutting edges.[179] OneShape instruments have variable pitch lengths along the working part (Figure 20-58). It is claimed that this design minimizes threading and binding of the instrument in continuous rotation.

According to comparative investigations, F360 and OneShape single-file instruments prepared curved canals equally well compared to reciprocating single-file systems and full-sequence rotary NiTi instruments (Figure 20-63).[172,179] The original canal curvature was well maintained and hardly any procedural errors were noted. The instruments were found to be safe. One study[171] found that the use of OneShape instruments required less time to prepare curved canals in extracted teeth compared with Reciproc and WaveOne, while another study[179] found no difference. However, preparation of severely curved canals was significantly faster with F360 and OneShape compared to the full-sequence Mtwo system (VDW).[179]

Two investigations evaluated the shaping ability of F360 and OneShape instruments compared with the two reciprocating single-file systems Reciproc and WaveOne in simulated S-shaped canals.[180,181] Both studies agreed that the lower tapered rotary single-file systems (F360 and OneShape) maintained both original canal curvatures better than the reciprocating instruments having greater tapers (Figure 20-64).

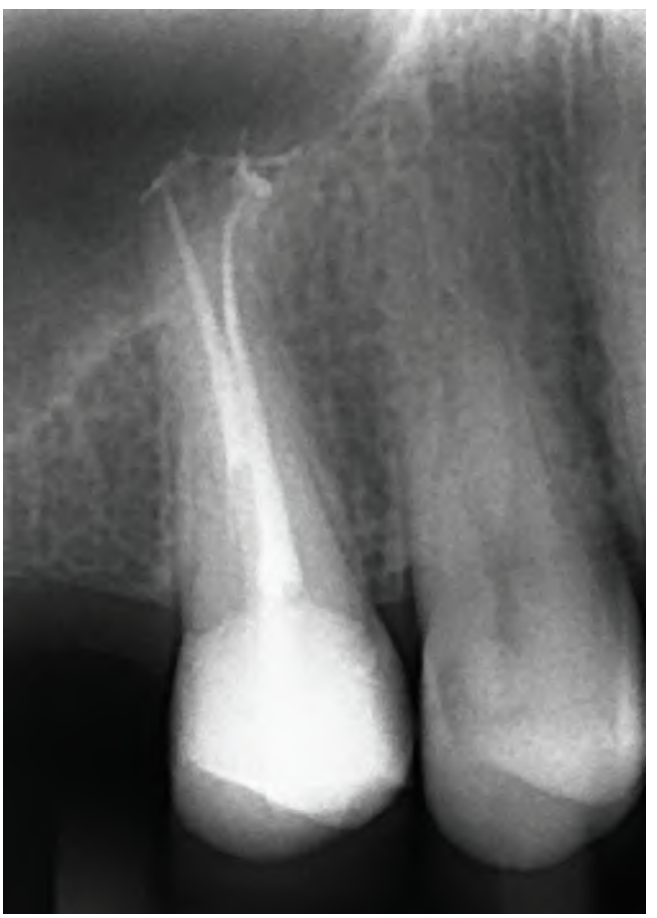


FIGURE 20-63 No visible canal transportation after preparation with a full rotary single-file system.

OneShape prepared these S-shaped canals significantly faster compared to WaveOne and F360 instruments.[181]

Regarding the tendency of rotary single-file systems to extrude debris apically and to cause dentinal damages, the available data is inconclusive.

Clinical Consequences of Faster Canal Preparation

Preparation time using automated NiTi systems is shorter. It can be decreased by up to 60% when using the single-file systems.[168] Consequently, the time available for irrigation and disinfection of the root canal system is substantially shorter. This must be taken into consideration by increasing the volume of the irrigants used. Passive ultrasonic activation of the irrigant may also be advantageous.

Conclusions

In summary it can be concluded that

- Single-file systems maintain well the original canal curvature, even of severely curved canals, and are safe to use.
- Single-file systems are faster than full-sequence rotary NiTi instruments.
- Due to the faster preparation time with single-file systems, special measures to improve canal cleanliness and disinfection (e.g., passive ultrasonic activation of irrigants, longer contact time) may be required.
- All single-file systems, except Reciproc, require a glide path preparation.
- Currently, controversy exists whether the use of reciprocating single-file systems is associated with increased risk of apical debris extrusion and dentinal damages (cracks).
- For complex canal anatomy such as S-shaped canals, rotary single-file systems with less taper seem to be better than reciprocating single-file systems.

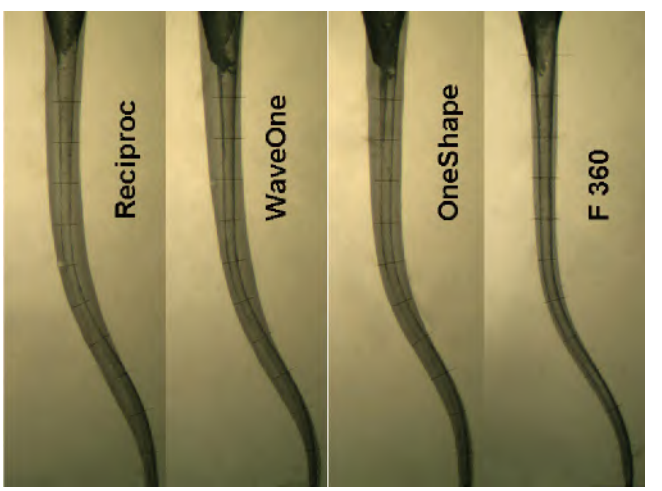


FIGURE 20-64 Canal transportation using different full rotary and reciprocating single-file instruments in simulated S-shaped canals. Preparation was performed to an apical size 25. The full rotary single-file instruments maintained both canal curvatures better than the reciprocating instruments.

SELF ADJUSTING FILE (SAF)

The SAF shows a new conceptual approach to instrument design. It is constructed of two broad lattices of NiTi that are connected by several small wires forming a connective mesh. This principle of construction allows the file to be compressed in small and narrow canals and to extend in root canals with large diameters. If the correct file size is selected, the instrument is designed to touch as much root canal surface as possible. The instrument operates in a special handpiece with slight vibrations in a frequency of 5,000 Hz.[182]

Since the inner part of the instrument is hollow, an irrigant of choice can be introduced via this empty space resulting in continuous irrigation during preparation. The irrigant can be delivered at a maximum rate of 1–10 mL per minute. The two lattices show a rough surface without blades. The file does not cut dentin like conventional instruments that are equipped with cutting blades, but rather removes dentinal hard tissue by brushing or scrubbing and immediately irrigating the shavings and tissue remnants out of the root canal. The tip of the file is asymmetrically designed, allowing the instrument to vibrate itself into the best position, hence the term Self Adjusting File.

The compressibility of the instrument enables a good adaption to the cross-sectional shape of the canal. When inserted into an oval canal with a 0.2 mm mesio-distal diameter, a 1.5 mm SAF will be compressed mesio-distally and thus spread bucco-lingually as far as 2.4 mm (Figure 20-65).[182]

The manufacturer recommends using the SAF for four minutes per canal with a 4 mL per minute flow rate of irrigant in small up and down movements. Ideally, the canal will be uniformly enlarged along its entire circumference, resulting in an undefined, non-ISO shape, magnification of the original canal shape.

Regarding obturation, lateral compaction does not seem to be suitable for the SAF technique since the prepared root canal does not show a specific geometry. Thermoplastic obturation techniques may be difficult since the apical taper is not clearly defined.

The SAF represents an interesting and novel approach of root canal preparation. It is able to save dentin by maintaining the original root canal cross-sectional shape and provides good canal cleaning. However, the SAF system is not widely used. Further studies are still necessary.

MOTORS AND TORQUE CONTROLLED HANDPIECES

Following the improvement in instrumentation systems, endodontic motors have also undergone enhancements in torque control and adjustable kinematics.[183] Modern automated devices can be classified into:

- Torque-limited handpieces
- High- and low-torque electric motors
- Open and closed reciprocating motors

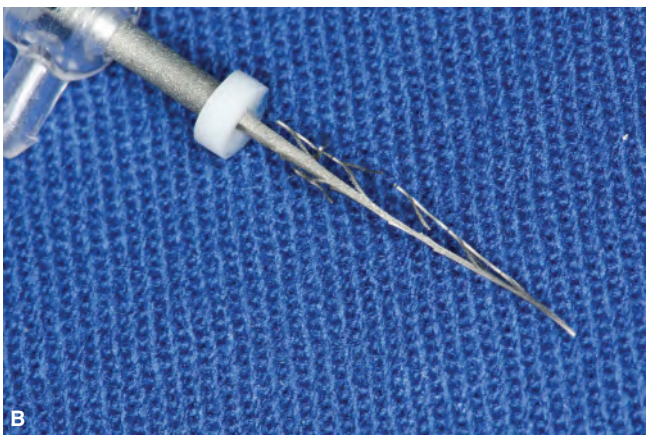


FIGURE 20-65 ***A&B, C.*** Instrument fractures are prevented by the connecting mesh.

- Motors combining rotary and reciprocating motion (adaptive motion)
- Vertical vibration (see "Self Adjusting File")

Torque-Limited Handpieces

These handpieces possess an integrated mechanical torque control. The different torque settings can be chosen by a swivel type adjustment ring of the handpiece (Table 20-6).[184] One of the first torque-limited handpiece was the ENDOadvance (KaVo, Biberach, Germany), that has a maximum speed of 40,000 r.p.m. Due to a 120:1 reduction, instruments are rotating with a speed of 333 r.p.m. The torque setting can be adjusted by a glide clutch to 0.25, 0.50, 1.0, and 3.0 Ncm. A clicking sound and a vibration are noticed as soon as the clutch is activated.[184]

The torque-limited rotation contra-angle air-driven handpiece SiroNiTi (Sirona, Bensheim, Germany) set the instruments in rotation with a maximum speed of 350 r.p.m due to a reduction of 115:1. This handpiece allows the selection of five different torque settings (between 1 Ncm and 3 Ncm) (Figure 20-66). Retro-rotation prevents the instruments from becoming locked in the root canal if excessive torque is reached.[184] The successor model SIRONiTi Air+ (Sirona) can be used either with an air motor or with an electric motor. When used with an electric motor, even instruments requiring higher rotational speeds such as RaCe, BioRaCe, and BTRaCe (all FKG, La-Chaux-de-Fonds, Switzerland) can be used, as the SIRONiTi Air+ has a maximum rotational speed of 600 r.p.m at a motor speed of 40,000 r.p.m.[184]

The Anthogyr NiTi Control handpiece (Dentsply Maillefer, Ballaigues, Switzerland) is another contra-angle handpiece that can be connected to the dental unit with a reduction ratio of 64:1 for pneumatic dental units and 128:1 for electrical ones, respectively. This handpiece permits a maximum speed of 312 r.p.m., and the torque level can be adjusted between 0.7 Ncm and 4.5 Ncm.[184] Especially for Mtwo instruments (VDW, Munich, Germany), the air-driven torque-limited rotation handpiece Mtwo direct (Sirona, Bensheim, Germany) was introduced (Figure 20-67). The torque settings of this handpiece are specially adjusted for Mtwo instruments and can only accommodate these files. (Table 20-7).[184]

A study investigated the influence of a torque-limited handpiece (SiroNiTi; Sirona, Bensheim, Germany) and an electric stepper motor using various full-sequence NiTi instruments in simulated curved canals in resin blocks with curvatures ranging from 0° to 30°.[185] It was found that while the use of the torque-limited handpiece was associated with significant shorter preparation times, no significant differences in canal straightening and loss of working

TABLE 20-6 Characteristics of Some Torque-Limited Rotational Handpieces

	ENDOadvance Lux NT	SiroNiTi Air+	Anthogyr NiTi control	Mtwo direct
Manufacturer	KaVo	Sirona	Dentsply Maillefer	Sirona
Suitable for	rotary NiTi instruments	rotary NiTi instruments	rotary NiTi instruments	Mtwo
Torque levels	4	5	4	8
Reduction	120:1	115:1	66:1 and 128:1	115:1
Rotational speed (rpm)	333	350 – 600	312	350
Reverse rotation	No	Yes	No	yes

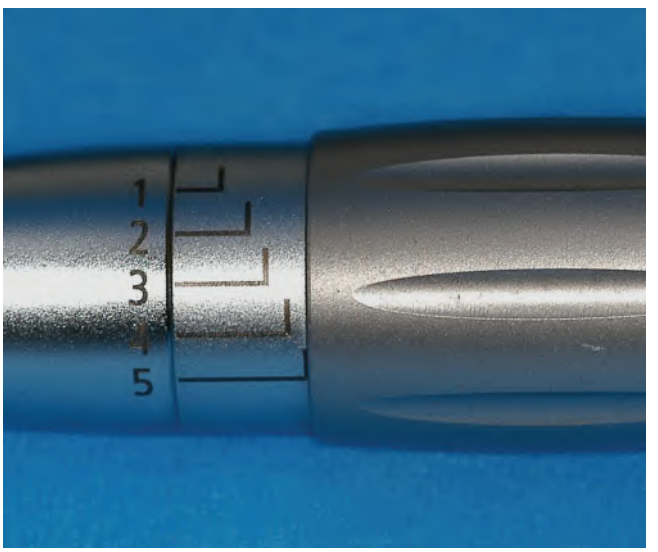


FIGURE 20-66 SiroNiTi handpiece with ring for selection of the five different torque setting.

FIGURE 20-67 The torque-limited Mtwo direct handpiece with a swivel type adjustment ring for the selection of the different torque setting. The eight different torque settings are adjusted to the particular size of the rotary NiTi Mtwo instruments.

TABLE 20-7 Characteristics of Some Selected Low-Torque Electric Motors

	torque (Ncm)	torque levels	rotational speed (rpm)	individual programming	updates
Dentaport ZX (Morita)	0.3 – 4.9	11	0 – 400	yes	possible
Endo IT (VDW)	15 – 500 gcm	individual for each instrument	150 – 1860	yes	possible
TriAuto ZX (Morita)	0.3 – 2.6	8	260 – 300	no	No
VDW.Gold (VDW)	20 – 500 gcm	Individual for each instrument	200 – 2000	yes	possible
VDW.Silver (VDW)	20 – 410 gcm	15	250 – 1000	yes	possible
X-Smart (Maillefer)	0.6 – 5.2	9	120 – 800	yes	possible

length were detected. These results were supported by another study indicating that both systems respected well the original root canal curvature and were safe.[186]

Similar results were obtained when using the full-sequence FlexMaster instruments (VDW) in a crown-down approach.[187] The automated devices compared in this study included two torque-limited handpieces (SiroNiTi and ENDOadvance) and a low-torque electric motor (Endo IT professional). Preparation of simulated curved canals in resin blocks was significantly faster with the electric motor than with the torque-limited handpieces, and ENDOadvance was significantly faster than SiroNiTi. In severely curved canals of extracted teeth, preparation with Endo IT and ENDOadvance was significantly faster than with SiroNiTi and the use of electric motor resulted in less canal straightening as compared to SiroNiTi. Regarding all other parameters, no significant differences were observed.[187]

High- and Low-Torque Electric Motors

Although high-torque motors allow very efficient root canal preparation, the use of these motors was found to be associated with an increased incidence of file deformation and separation.[188] In low-torque motors with torque levels beneath 1 Ncm, torque settings below the estimated torque failure can be selected (Table 20-8). The reduction of cyclic fatigue of NiTi files seems to be more reliable with low-torque motors.[189,190] However, the torque value should not be set too low because a minimum torque is required for any engine-driven instrument to work against friction inside the root canal and to cut-off dentin.

Electric motors usually have an auto-retro-rotation function to loose locked instruments (Figure 20-68). To minimize the risk of instrument separation, in some systems an acoustic warning tone can be adjusted by the operator when a critical torque limit is reached.[184] In the library of these motors, pre-settings of optimal rotational speed and maximum torque values are stored (Figure 20-69) and for most motors, these values can be updated for newly introduced system by an interface (Table 20-7).

For most rotary NiTi systems, the recommended rotational speed is in the range of 150 r.p.m - 600 r.p.m. Data regarding the influence of the rotational speed on the safety and efficiency of the instruments used is inconclusive.[183]

TABLE 20-8 Set Values of Different Closed Reciprocating Motors (CCW=counter-clockwise; CW=clockwise)

Motor	CCW angle (°)	CW angle (°)	Cycle rotation speed (r.p.m.)
VDW.Silver – Reciproc ALL mode	150	30	300
VDW.Silver – WaveOne ALL mode	170	50	350
ATR Technika	144	72	400

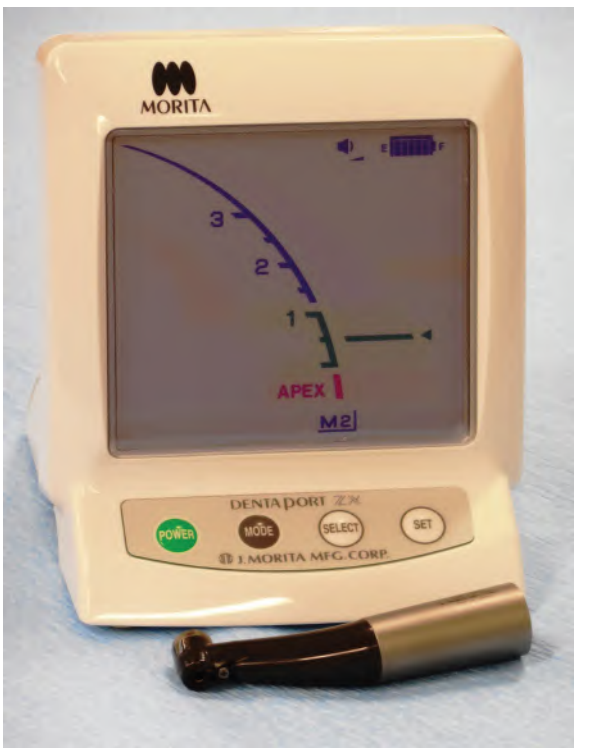


FIGURE 20-68 The Dentaport ZX Set ORT motor (Morita, Japan). The ORT (Optimum Torque Reverse) mode is claimed to relieve the stress on the instrument during preparation. When reaching a specific torque value, the full clockwise rotation of the instrument changes direction to a 90° counter-clockwise rotation and then reverts back to a clockwise rotation. If the instrument is still under stress, it will turn another 180° in a clockwise direction before reversing back to relieve.

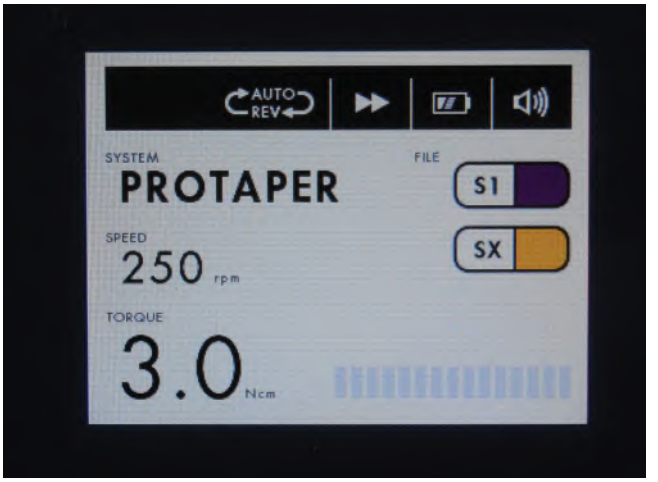


FIGURE 20-69 Display of the X-Smart Plus motor showing the pre-set torque value and the rotation speed.

FIGURE 20-70

Some studies reported that increasing the rotational speed resulted in increased efficiency of the instruments[191,192] and was not associated with an increased risk of instrument deformation, higher torque or higher risk of instrument fracture.[193,194] However, one study reported that an increase in the rotational speed resulted in higher risk of instrument fracture.[195] It appears that the combination of the rotational speed of the instrument and the degree and radius of the canal curvature is mainly responsible for the increased risk of instrument breakage.[196]

While low-torque motors were found to be safer when used by inexperienced operators,[197] no such differences were found for experienced operators.[81,198] However, it should be noted that in some motors, the actual torque values differ from the values indicated on the motors. These can be even higher than the torque causing failure of some rotary NiTi instruments.[183,199]

Reciprocating Motors

Reciprocating motors are available with fixed values of angles and speed of reciprocation (representatives are: ATR Technika, Pistoia, Italy; X-Smart Plus, Dentsply Maillefer, Ballaigues, Switzerland); VDW.Silver Reciproc and VDW. Gold Reciproc (both from VDW, Munich, Germany) (Figure 20-70) (Table 20-8).

In contrast to these closed-system motors, other motors are designed to allow modification of reciprocation.[183] The ATR Vision (ATR, Pistoia, Italy) and iEndo Dual (Acetone, Merignac, France) are representatives of open-system motors.

It is noteworthy that the actual values of reciprocating differ markedly from the set values given by the manufacturers.[169] As long as the actual values are below the set ones, the clinical consequences are negligible as decreasing the reciprocating values results in longer preparation times but simultaneously in increased cyclic resistance of the instruments and less canal transportation during preparation.[200] However, increasing the range of reciprocation was found to reduce the cyclic fatigue resistance of the instruments.[201] Thus, the operator should be aware of the actual reciprocating values of the particular motor used, in order to avoid undesirable effects like instrument fracture during the prolonged used of reciprocating instruments.

Regarding the impact of the reciprocating working motion on the torsional and cyclic fatigue, as well as the life span of the instruments used, the results of the available studies agree that the reciprocating motion is associated with a higher mean life span and an increased cyclic fatigue resistance.[183,202,203] The incidence of instrument fracture has been reported to be considerably low for reciprocating instruments.[204,205] The reciprocating movement seems to be relatively safe as it is assumed that deformation of the instruments during the counterclockwise rotation can be recovered upon unloading.[206]

Most of the torque-controlled and reciprocating endodontic motors are also available with an integrated electronic apex locator (Figure 20-71). Clinical studies showed that the accuracy and reliability of working length determination using these apex locating endodontic motors was comparable to independent apex locators.[207-210]

Reciprocating Handpiece

Recently, a wireless handpiece has been introduced, generating reciprocating working motion and full clockwise rotation. The VDW.Connect Drive (VDW) is a handpiece that can be connected via Bluetooth with an iPad (≥ iOS 8) (Figure 20-72). A special App (VDW.Connect App) offers the opportunity to select the required working motion, either reciprocating or full clockwise rotation. The required angles of reciprocation and the torque and rotational speed values for the different instrument sizes are stored in the library of the App. Additional functions, such as electronic apex locator, can be selected via the App. As customary for Apps, the system can be updated via regular downloads offered by

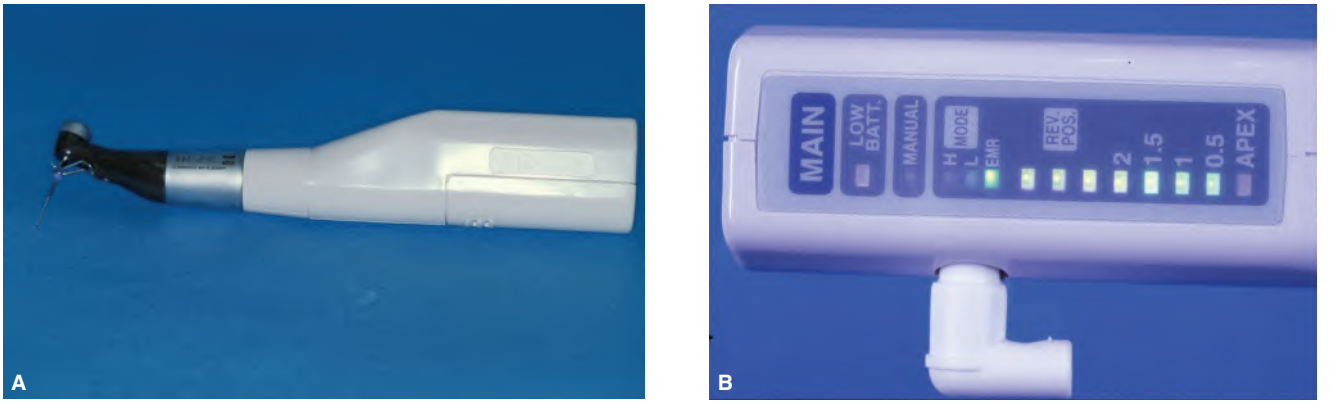


FIGURE 20-71 ***A.*** The torque-control motor TriAuto ZX (Morita, Dietzenbach, Germany). ***B.*** The display showing two different torque settings and the integrated electronic apex locator.

FIGURE 20-72 ***A.*** The VDW.Connect Drive handpiece generates reciprocating working motion and a full clockwise rotational working motion. ***B.*** The VDW.Connect App offers the opportunity to select different working motions.

the manufacturer. Another similar wireless handpiece called X-Smart iQ (Dentsply/Maillefer) has also been recently launched. It has the same features as the VDW.Connect Drive and is also connected via Bluetooth. Studies assessing these handpieces are not yet available.

Motors Combining Rotary and Reciprocating Motion (Adaptive Motion)

The latest developments in the field of automated devices are electric motors that combine the rotary and the rotational reciprocating motion (i.e., adaptive motion). Originally, this adaptive working motion was developed for the use of Twisted File Adaptive instruments and the Elements Motor (both from Kerr/SybronEndo, Orange, CA, U.S.A.).

This motion aims to combine the advantages of the rotary and the reciprocating motion.[183] When the instrument used is not stressed, or only minimally stressed, the motor generates a rotary clockwise motion of about 600° inside the canal and then stops. Thereafter, the clockwise rotation restarts. Whenever the instrument is exerted to intracanal stress (in curved and narrow canals, or during removal of filling materials for retreatment) the motion of the instrument is changed to reciprocal.[183] The angles of reciprocating are not constant but depend on the intracanal stress of the instrument and can be modified in the range of 0°-600° for the clockwise and 0°-370° for the counterclockwise motion.[183]

Data regarding the properties of this new generation of endodontic motors is scarce. However, preliminary results indicate that under cyclic fatigue testing, the adaptive motion increased the time to fracture of TF Adaptive instruments (Kerr/SybronEndo) when compared to full rotary motion.[211]

In summary it can be concluded that

- Torque-limited handpieces seem to be safe and allow a rapid preparation of even severely curved root canals without increased risk of procedural errors. The main advantages of these handpieces in comparison to electric motors are their lower cost and that no separate dental equipment is necessary.

- For electric motors, actual torque values may differ markedly from the pre-set values. Occasionally, actual values may be higher than those values reported at fracture. Selection of rotational speed is crucial as instrumentation speed directly affects instrument failure.
- For reciprocating motors, actual angles of reciprocation as well as speed may differ from the set values of the particular motors.
- The reciprocating motion seems to increase the fatigue resistance of instruments. Also, the incidence of instrument fracture is lower for reciprocating instruments when compared to rotary instruments.
- The adaptive motion seems to have a beneficial effect on cyclic fatigue of the instruments.
- Currently, no evidence exists suggesting that any specific automated device has any impact on treatment outcome.

ULTRASONICS

In 1976, Dr. Martin reported on the use of ultrasonics for root canal preparation and termed the technique "endosonics." Nevertheless, the first report on the use of ultrasonics for root canal treatment was published almost 20 years earlier by Richman in 1957.[212] Cavitation has been discussed as a main mechanism, however, acoustic streaming should be regarded the main mode of action.[213]

Ultrasonic devices are driven by piezoelectricity or magnetostriction, resulting in high-frequency oscillation (25 kHz - 40 kHz) of the inserted file that initiates acoustic microstreaming in the irrigation fluid. This action has been reported to result in excellent cleaning ability exerting antimicrobial activity by destruction of cell membranes.[214]

Shaping

Most studies report unsatisfactory results concerning the quality of preparation,[215,216] due to the high-frequency oscillations resulting in poor control of the file and especially the file tip. Since ultrasonic units are used with stainless steel instruments, the outer aspect of curved root canals is easily straightened or shows severe zipping and ledging. Longitudinal grooves in the root dentin following the use of ultrasonically activated files have been detected[217,218] (Figure 20-73). Nevertheless, when used carefully, ultrasonic instruments can be helpful in removal of obstacles, opening root canal orifices, and widening narrow isthmi.

Cleaning

The cleaning and disinfecting capacity of ultrasonics is still controversial. Excellent or good root canal cleanliness, removal of debris and smear layer compared to conventional irrigation techniques have been reported.[219-222] (Figure 20-73)

Working Safety

Apical blockages, ledging, and increased amount of apically extruded debris[223,224] as well as instrument fractures have been associated ultrasonic preparation.

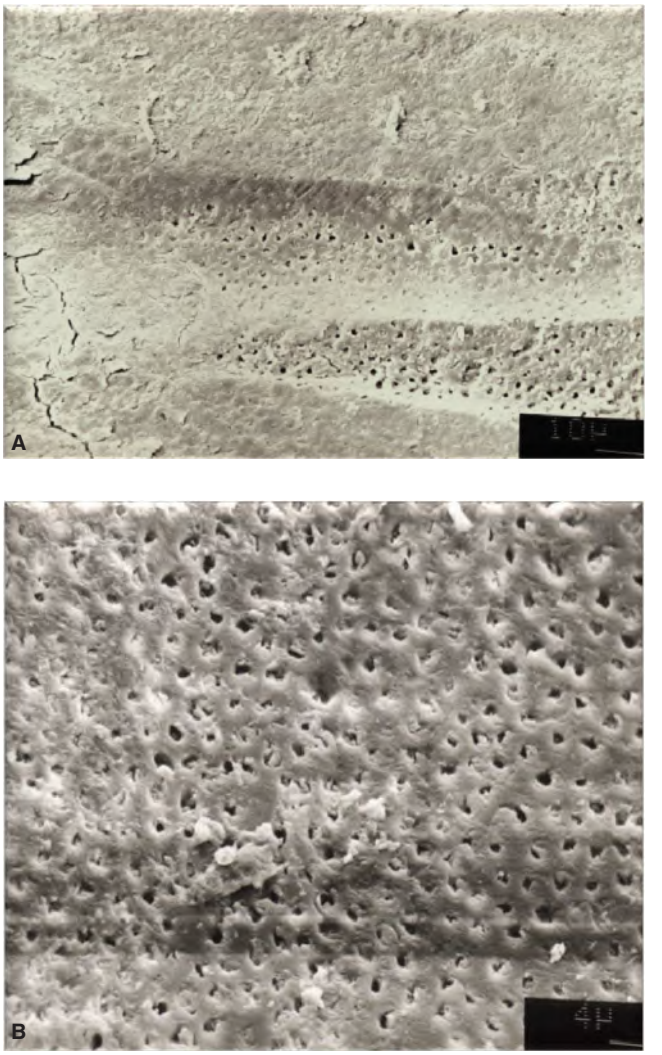


FIGURE 20-73 ***A.*** Longitudinal grooves in the root dentin following the use of ultrasonically activated files. ***B.*** Root canal wall following use of ultrasonics. Smear layer and debris have been removed completely.

Conclusions

It can be concluded that ultrasonics is a good adjunct for irrigation, but cannot be recommended for routine root canal preparation. Ultrasonics can be helpful in selected situations.

CONTROVERSY I: DENTINAL DAMAGE

With the introduction of reciprocating single-file systems, a new controversy has recently been rekindled regarding the impact of mechanical root canal preparation on dentinal damage. During root canal instrumentation and irrigation the structure, composition, and physical properties of dentin can be altered.

The following effects of mechanical root preparation and disinfection on root canal dentin were documented:

- Reduction of fracture resistance
- Preparation marks
- Dentinal cracks

Reduction of Root Strength

Any loss of tooth substance during preparation of the coronal access cavity is associated with a decrease in fracture resistance.[225,226] In general, the fracture resistance of root canal treated teeth is directly related to the amount of remaining sound tooth structure.[227] Currently, hand root canal preparation, using ISO standardized instruments with a constant taper of .02, appears to be associated with less weakening of the tooth compared to engine-driven NiTi instruments with tapers greater than the ISO standard.[228,229] When treating non-infected root canals, the use of greater taper instruments should be reconsidered and kept to a minimum. In cases of vital extirpation, there is no need to excessively enlarge such canals since they are not infected.[226] This will also reduce the risk of cracks or fractures (Figure 20-74).[227]

Preparation Marks

Evidence from several studies indicates that mechanical root canal preparation may cause preparation marks at the canal walls. Such defects have been shown following hand instrumentation as well as following engine-driven canal preparation (Figures 20-74 and 20-75).[2,86]

Dentinal Cracks

Dentinal cracks or root fractures occur when the tensile stress in the root canal wall exceeds the tensile stress of dentin.[228] During root canal preparation, the cutting edges of the instrument used make contact with the dentin walls creating momentary stress concentrations in dentin.[228] The highest tensile and comprehensive stress concentrations occur at the external root surface in the apical part of the canal. The authors demonstrated that the stiffer the instrument is (or the greater the taper of the instrument) the higher the stress concentration in the apical root dentin.[228] These stress concentrations may cause either complete or incomplete dentinal cracks, short- or long-term[230] (Figure 20-76).

It is currently debatable whether or not the use of single-file systems is associated with an increased risk of creating dentinal defects. Data regarding this issue are scarce at the moment. One study also assessed one rotary single-file system (OneShape, Micro Méga, Besançon, France).[231] Results showed dentin cracks in 50% of canals prepared with the full-sequence ProTaper systems (Dentsply Maillefer), in 5% of canals enlarged with Reciproc (Dentsply Maillefer) and in 35% of canals instrumented with OneShape.

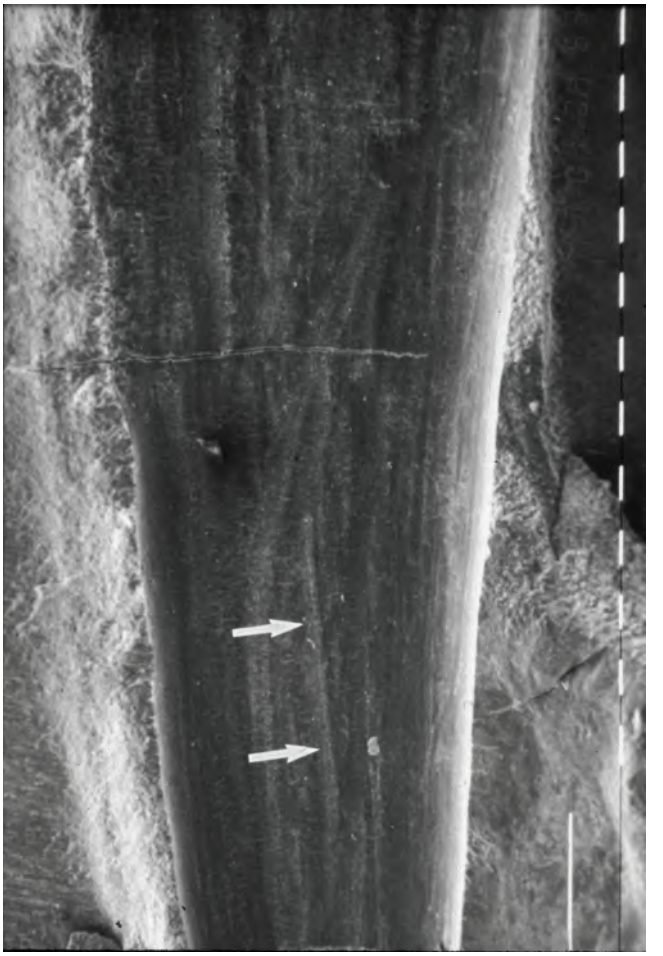


FIGURE 20-74 Scanning electron microscopic image showing grooves at the canal wall (arrows) running from coronal to apical following canal preparation with stainless steel Hedstrom files, used in a linear filling motion (magnification x160).

Definite appraisals of the clinical consequences are not possible and mere speculative at the moment.

Conclusions

In summary it can be concluded that:

- The different steps of root canal treatment are associated with a reduction of fracture-resistance of teeth.
- The use of tapered instruments has a crucial impact on root strength.
- Root canal preparation may cause preparation marks on the canal walls.
- Engine-driven preparation may cause dentinal defects such as complete and incomplete dentin cracks.
- Whether or not reciprocating instruments cause more dentinal defects than full-sequence rotary NiTi instruments is not clear.

CONTROVERSY II: APICAL PATENCY

One of the controversies that existed for some years has been the discussion on the benefits and shortcomings

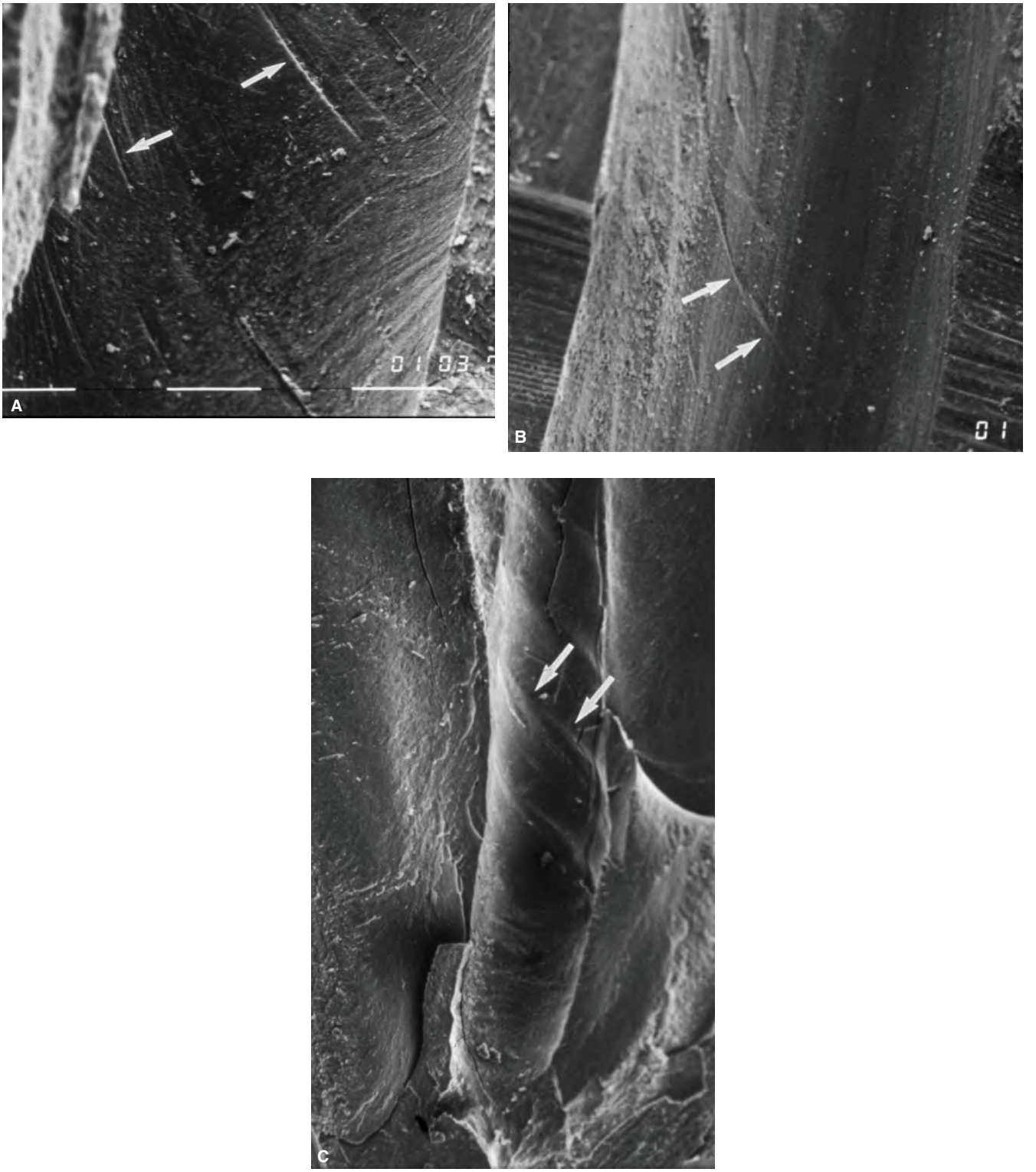


FIGURE 20-75 Scanning electron microscopic images of preparation marks. ***A & B.*** Slight grooves (arrows) following hand instrumentation with stainless steel K-files used in a reaming motion (x160). ***C.*** Clearly defined wave-shaped pattern (arrows) after using stainless steel K-files (x160).

of preparation techniques including "apical patency."[232] Buchanan[233] was credited as the first author introducing the patency concept. The patency file must be a small flexible K-file that should be used passively through the apical constriction without widening it.[234] The definition suggested by the American Association of Endodontists (AAE) is[235]: "A preparation technique in which the apical region of the root is kept clear of debris by recapitulation through the physiological foramen (apical constriction) with a fine file" (Figure 20-77).

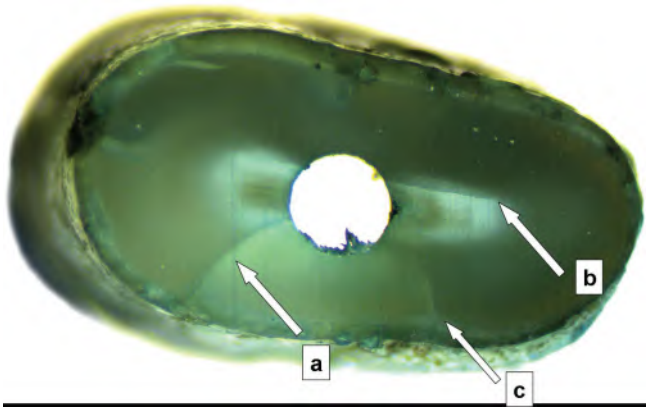


FIGURE 20-76 Cross-section of a root showing complete (a) and incomplete (b & c) dentinal cracks (x25).

It should be noted that some debris could be extruded beyond the apical foramen potentially carrying microorganisms into the periapical tissues and causing post-operative pain.[236,237] Also, only the major foraminal opening, located in the straight axis of the root canal, can be treated with the patency technique. Lateral openings and ramifications remain mechanically untreated and at best can be disinfected chemically by the irrigant (Figure 20-77). For more details, see Chapter 21. Nonetheless, when the patency technique was performed, with a file contaminated with bacteria in root canals filled with 5.25% sodium hypochlorite, no contamination of the periradicular tissue was found[238] (Figure 20-77).

The key concept when applying this technique is the use of very small size files (#8, #10 or #15). Larger files may carry the risk of opening the apical constriction or creating a "zipping effect."

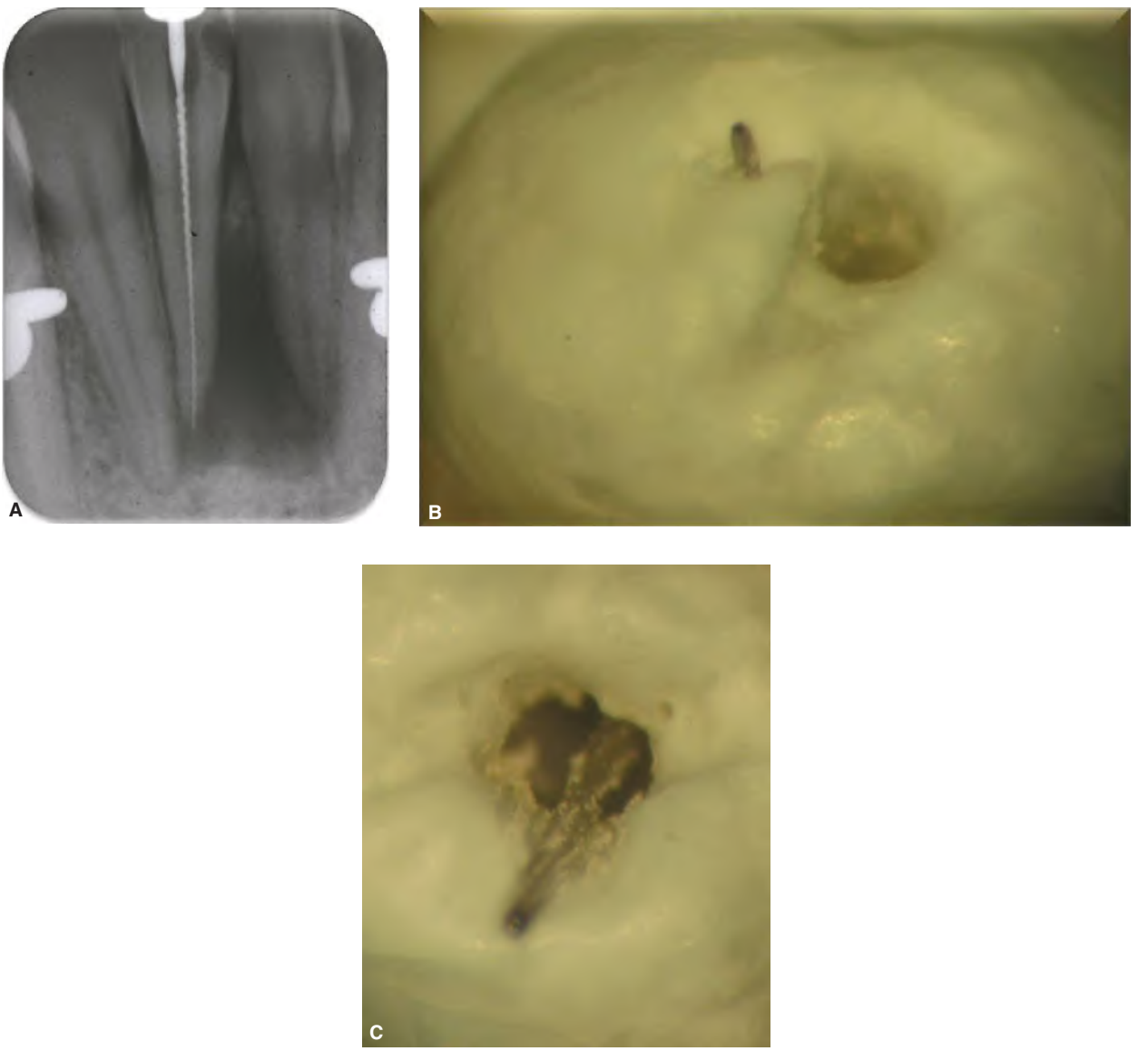


FIGURE 20-77 ***A***. Apical patency aims at keeping the foramen free of debris by intentionally extruding the file tip slightly beyond the apical foramen. ***B***. Patency has been achieved for one foramen, but the larger one remains uninstrumented. ***C***. Even with a small patency file size 010 debris is pushed beyond the foramen. Note that the circumference of the foramen is still covered with debris that could not be removed by use of a patency file.

When patency was assured with sizes 10 and 15 files, all teeth showed apical extrusion of irrigant.[239] Delivery of irrigant into the apical part of large root canals was superior when patency was maintained.[240,241] Also, the presence of gas bubbles inside the irrigant was minimized.[242]

In non-vital teeth, the frequency of posttreatment pain was lower when patency was maintained. Nevertheless, in some cases (mandibular teeth, teeth with pre-existing pain) duration of postoperative pain was extended in teeth with patency.[243]

General Health

In some studies, it has been demonstrated that overinstrumentation results in bacteremia.[244,245] Therefore, apical overinstrumentation or extrusion of infected debris should be avoided in patients with compromised health.[232,246,247]

Conclusions

- Infected debris, extruded through the apical foramen into the periapical tissues may cause periradicular inflammation.
- Apically extruded material may play a role in the etiology of post-treatment pain.
- Only major apical foramina can be treated using the patency technique.
- Controlled clinical trials and prospective studies validating the benefits or disadvantages of patency techniques are lacking.
- Patency techniques should not be used in medically compromised patients.
- When treating teeth with vital pulps, there is no indication or justification for intentional overinstrumentation.

From the limited evidence-based data in the current endodontic literature it is hard to solve the debate on apical patency.

CONTROVERSY III: APICAL EXTRUSION OF DEBRIS

Avoiding extrusion of some debris during root canal preparation is virtually impossible[248,249] (Figure 20-78). During root canal preparation, necrotic or vital pulp tissue, dentin chips, microorganisms, irrigants and in the case of retreatment even certain filling materials may be extruded into the periapical tissues.[248]

The most relevant consequences of apical debris extrusion during root canal preparation are post-treatment symptomatic apical periodontitis and interappointment flare-ups.[236] Thus, from a clinical and biological points of view, it is mandatory to make every effort to minimize the amount of apically extruded debris during root canal preparation.

When comparing different hand preparation techniques to engine-driven rotary preparation, it has been reported that hand preparation techniques using crown-down approach, or employing cervical flaring, were associated with less debris extrusion than techniques involving a linear manual filing motion.[248,250-252] On the other hand, some studies reported that, when properly used, rotary engine-driven preparation does not produce significant debris extrusion.[253,254]

FIGURE 20-78 Debris extruded beyond the apical foramen during root canal preparation.

Single-File Systems

Currently, controversy exists regarding the tendency of reciprocating single-file systems (WaveOne [Dentsply Maillefer, Ballaigues, Switzerland]) and Reciproc [VDW, Munich, Germany]) to extrude debris into the periapical tissues.[255] While one study found that the use of reciprocating single-file systems was associated with greater amounts of apically extruded debris,[256] other investigations reported contradictory results.[257-259] However, concerning the tendency of rotary single-file systems (F360 [Brasseler, Lemgo, Germany] and OneShape [Micro Méga, Besançon, France]) to extrude debris apically, the available data indicate that these systems caused less debris extrusion than the reciprocating single-file systems.[179,260] Although the currently available data are limited, the use of rotary single-file systems does not appear to be

associated with an increased risk of debris extrusion.[261] On the other hand, the impact of reciprocating canal preparation with particular single-file systems is currently debatable.

In this context, a recent meta-analysis study showed a trend that the use of reciprocating single-file systems was associated with greater amounts of extruded debris compared to full-sequence systems.[261] The reciprocating movement could have favored apical packing of debris and dentin, that might result in transportation of this debris beyond the apical foramen.[249,261] However, the cross-sectional design of the instruments was found to be the most relevant parameter that affected debris extrusion.[249] Instruments having greater cross-sectional areas extruded more debris apically and caused greater expression of neuropeptides (substance P and CGRP) than instruments having an S-shaped cross-sectional design.[249] Clearly, a sufficient chip space is a crucial design feature of root canal instruments that seems to have a direct impact on the amount of apically extruded material. Finally, this meta-analysis showed that the number of instruments used has an impact on apical debris extrusion, although current evidence supporting this conclusion is very limited.[249]

The results of this meta-analysis are at least partially supported by the findings of two clinical studies[262,263] Gambarini et al.[262] assessed post-treatment pain after root canal treatment using three different engine-driven instruments: the reciprocating single-file system WaveOne, Twisted Files instruments (Kerr/SybronEndo, Orange, CA, U.S.A.). A statistically significant difference was found between WaveOne and the two other instruments.

Following canal preparation with WaveOne, about 26.6% of patients experienced severe pain. None of the patients of the Twisted File group and only about 6.6% of the Twisted File adaptive group reported pain.

In a randomized clinical trial, root canals of 42 patients were prepared using the full-sequence rotary ProTaper systems and the reciprocating WaveOne instrument (both from Dentsply/Maillefer).[263] Treatments were performed in two appointment and postoperative pain was assessed using a numerical rating scale (NRS) score. Duration of pain and analgesic consumption were also evaluated. After both appointments, the NRS scores and the duration of pain were significantly higher in the WaveOne group. After the first appointment the analgesic consumption was significantly higher in this group as well. The authors explained these findings by the fact that the single-file WaveOne instrument may create a "*piston effect*" inside the root canal thereby extruding more debris apically than the instruments used in a continuous clockwise rotation.

Conclusions

In summary it can be concluded that

- Currently, the use of any types of instrument or preparation technique is associated with some debris extrusion.
- Full-sequence rotary NiTi instruments seem to extrude less debris than hand instruments.
- Full rotary single-file systems seem to extrude less debris than reciprocating single-file systems.
- Reciprocating single-file systems seem to extrude more debris than rotary full-sequence systems.

PREPARATION OF COMPLEX ROOT CANAL SYSTEMS

Confluence of Root Canals

If a confluence of two root canals is not detected, a high probability of over-enlargement and/or straightening exists (Figure 20-79). If the angle of insertion of the

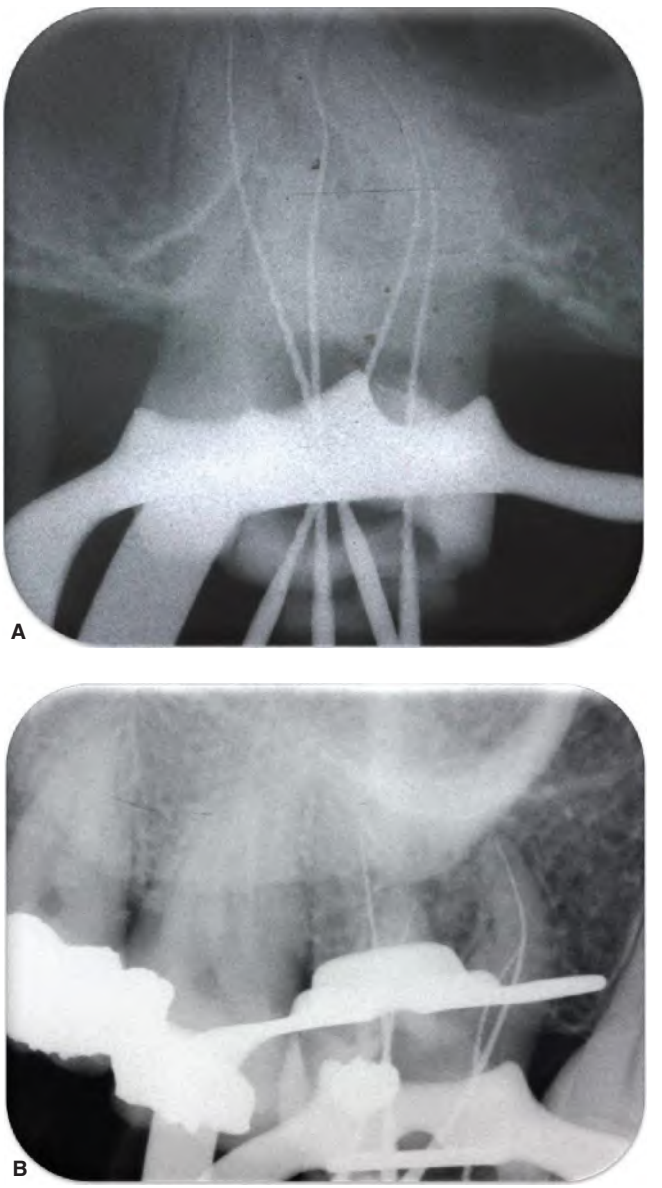


FIGURE 20-79 ***A & B.*** Radiographs demonstrating different locations and angles of confluence.

second canal is too large, there is a high risk of ledging or instrument fracture. Additionally, confusion during obturation may occur, as the estimated working length cannot be reached in the second canal when the first already has been obturated. Radiographically, in many cases, a confluence cannot clearly be diagnosed. Clinically, several methods have been proposed to identify confluencing root canals[80]:

- Endodontic instruments are inserted into both root canals. If only one can be inserted to working length at a time, confluence may be present.
- A gutta-percha point is placed in one root canal and an endodontic instrument into the other one. If joining, the instrument will leave a mark in the gutta-percha at the point of confluence (Figure 20-80).
- Both root canals are filled with an irrigant. When the level of irrigant is sinking in the second canal during drying of the first one, the presence of a confluence is likely. The same is true when calcium hydroxide is placed in one root canal and emerges from the second (Figure 20-81).

It should be remembered that confluences may not always be detected on radiographs.

Deep Splitting of Root Canals

The main challenges with deep splits of a root canal are detection and accessibility to the split. Thorough probing of the canal with a fine and pre-curved instrument is the best way of diagnosing a split. CBCT may also be helpful. For preparation and obturation, a good access cavity is mandatory, allowing accurate placement of gutta-percha, spreaders, or pluggers (Figure 20-82).

S-shaped Root Canals

Preparation of root canals presenting with a double, S-shaped curvature (also termed as bayonet-shaped), presents a major challenge for root canal instruments, whether

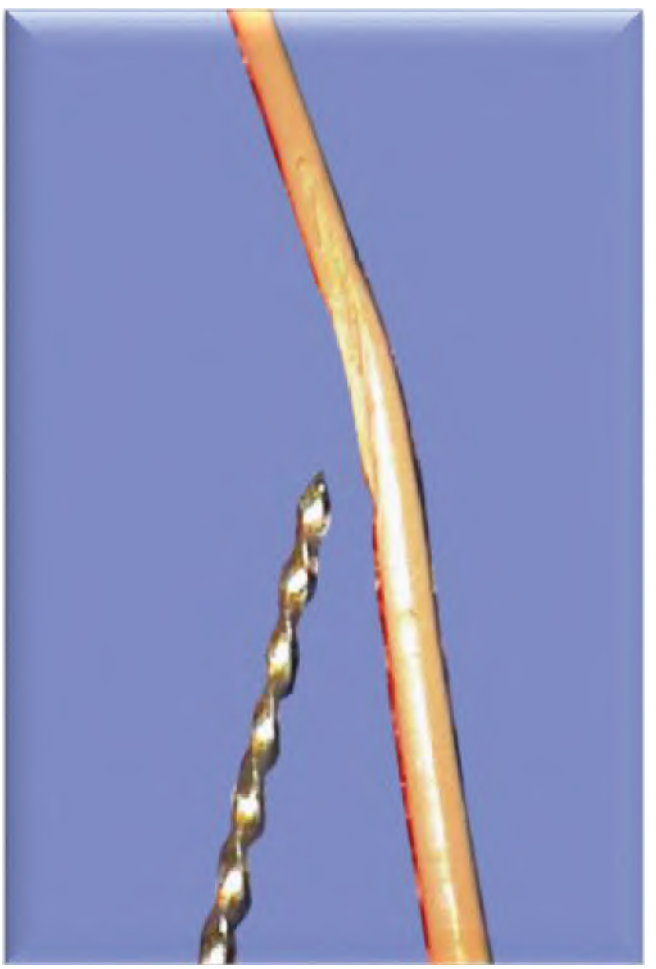
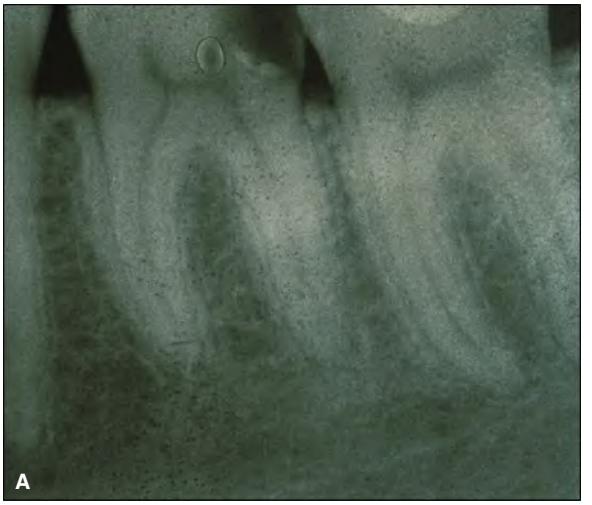
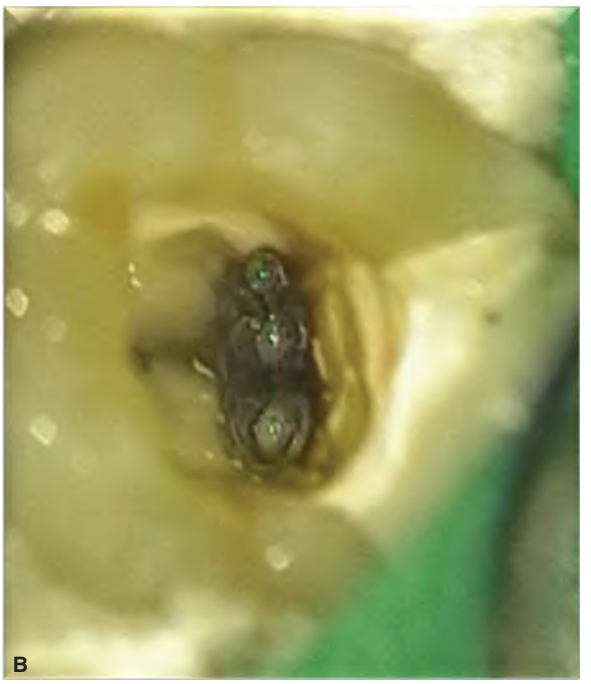


FIGURE 20-80 A gutta-percha cone is inserted in the "main" root canal. If an instrument is inserted in a joining canal, the impression on the cone will indicate location and angle of confluence.

FIGURE 20-81 If calcium hydroxide or an irrigant are placed into one root canal and emerge from the second, it is indicative of a confluence.





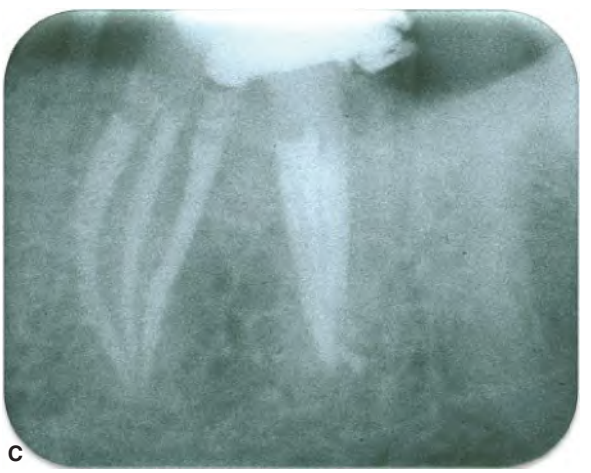


FIGURE 20-82 ***A-C,*** Deep splitting of root canals in the distal root of a mandibular molar. Detection and preparation were performed under the operating microscope.

stainless steel or nickel titanium. The severity of the problem depends on the radius and angle of the curvatures. It should be assumed in many cases, that an additional curve exists.

It has been demonstrated that complex anatomies such as double curvatures, S-shaped curvatures, abrupt curvatures or C-shaped root canals are associated with a higher risk of instrument fracture and unprepared root canal areas as well as of perforation.[264] Unfortunately, double curvatures in many clinical cases cannot be recognized well in a two-dimensional radiograph (Figure 20-83). For more details, see Chapter 26, Endodontic Therapy in Teeth with Anatomical Variations.

Cyclic behavior of the instruments is extremely challenged during preparation of such root canal configurations. Unfortunately, the vast majority of studies have been conducted on simulated root canals in plastic blocks since this is a way to work on standardized anatomy. It should be noted that such studies do not represent clinical reality and the results should be interpreted with caution. Micro-CT seems to have an advantage for this type of research.

Conceptually, it has been recommended to prepare S-shaped root canals in three main steps[265]: Following preparation of a sufficiently large access cavity, the coronal part of the root canal is enlarged upto the beginning of the first curvature. Next, the coronal curvature is shaped to allow access to the apical curve. Finally, the apical part of the root canal with the second curve, is enlarged with small precurved, or even slightly over-curved, hand instruments or flexible low-tapered NiTi instruments (i.e., ProFile .04). This procedure includes slight straightening at the inner aspect of the second apical curve, and the opposite side of the coronal curve.

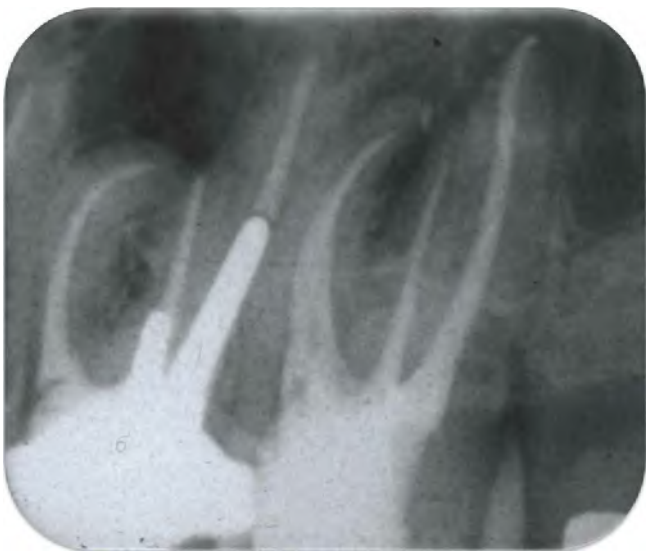


FIGURE 20-83 Maxillary molar with an S-shaped palatal root canal. The double curve in this tooth became visible only in the distal-excentric radiograph.

When multiple curvatures exist, rotary NiTi instruments are undergoing an additional stress. When compared to artificial root canals with single curves, during preparation of double-curved canals, the number of rotations till fracture (NRF) in the apical curve, dropped from 633 to 105 for ProFile .06/25, and from 548 to 93 for Vortex NiTi instruments.[266]

The results and conclusions from studies on preparation of S-shaped canals, using NiTi, instruments are controversial. Preparation of S-shaped root canals, in some studies, has shown less convincing results, regarding canal straightening and increased incidence of instrument fractures.[267,268] One study found that the SAF performed better than a few conventionally designed systems made from Controlled Memory Wire or M-Wire (Typhoon, Vortex).[269]

A final conclusion based on these findings cannot be drawn. However, it is recommended to:

- Prepare a straight-line access to the first curvature, eliminating any interferences in this part of the root canal.
- Prepare the first curvature before addressing the second one. Instruments with a larger taper can be used for this purpose.
- Start apical preparation only when enlargement of the first curvature has been performed.
- Use flexible instruments with a small taper (.05) for apical preparation, preferably hand instruments, and limit preparation to a size 25 or 30 (Figures 20-84).

Oval Root Canals

Depending on the extent of irregularity oval and C-shaped root canals also present some major challenge for preparation, disinfection, and obturation[270] (Figure 20-85). Under-preparation, leaving lateral extensions of the root canal walls untouched, and over-preparation, increasing the risk of strip-perforations, are among the major problems (Figure 20-86). Using hand instruments in a circumferential filing technique is recommended for oval and C-shaped root canals in order to adequately enlarge and clean both lateral extensions without over-preparation in the middle of the root canal.

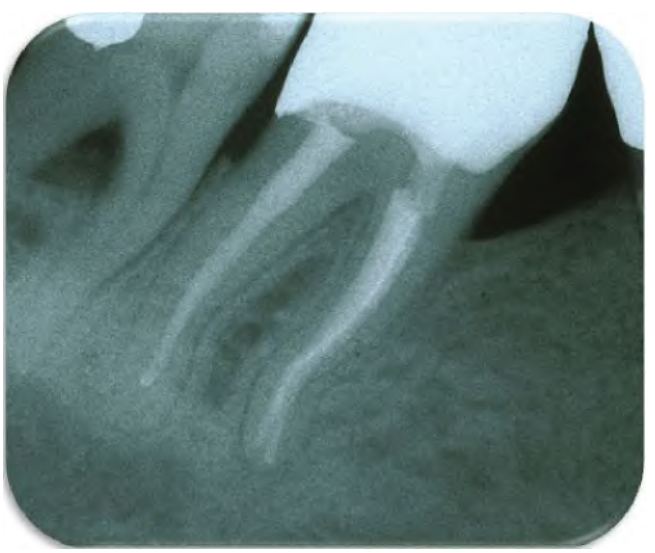


FIGURE 20-84 Well centered preparation with good maintenance of the curves in this first mandibular molar after preparation with FlexMaster .02/35. (Courtesy of Dr. Sabine Nordmeyer, Berlin, Germany)

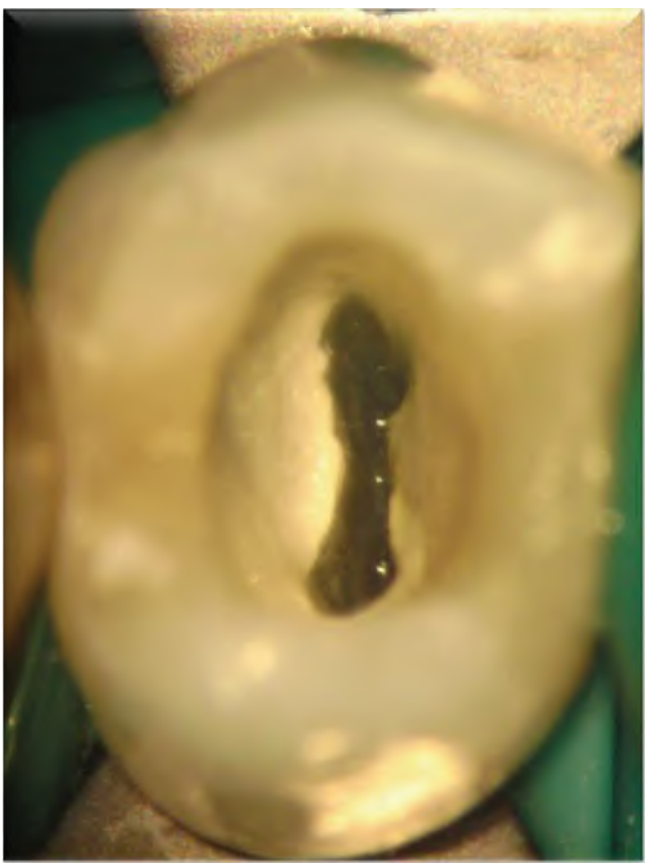


FIGURE 20-85 Long oval root canal system in a maxillary premolar.

FIGURE 20-86 Overpreparation on the inner concave side of the root canal may result in a strip-perforation.

In contrast to stainless steel instruments, flexible NiTi instruments are difficult to manipulate in lateral extensions. These instruments tend to penetrate along the path of least resistance and the file tip cannot be directed exactly into one direction by manipulation of the handpiece. This results is insufficient cleaning using single file, reciprocating, or full sequence NiTi rotary file systems.[271] ProTaper F2 left 24% of the pulp tissue in oval root canals, and the SAF 9.3%.[272]

No technique or system could completely prepare and clean oval canals.[273-275] When reciprocating instruments were used for preparation of oval root canals, no significant differences between the systems in cross-sectional shape were detected once the instruments were used in brushing motions.[275] Good results were achieved with the SAF that prepared long oval root canals with no gross preparation errors and minimal straightening, leaving 23% of the canal wall unprepared.[271] Peters at al.[276] showed that the percentage of untreated canal walls could be reduced from 63% (SAF 1.5 mm, two minutes working time) to 8.6% (using an SAF 2.0 mm for five minutes).

To optimize cleanliness and disinfection of oval root canals, ultrasonically activated irrigation directing the oscillating file into the lateral recesses has been advocated[277] (Figure 20-87).

C-Shaped Root Canals

C-shaped root canals present similar challenges as root canals with oval cross-sections. During preparation, the clinician should consider that the concavity is much more pronounced than in oval canals, increasing the risk of over-preparation, weakening the root, or even perforation. For more details, see Chapter 26, Endodontic Therapy in Teeth with Anatomical Variations.

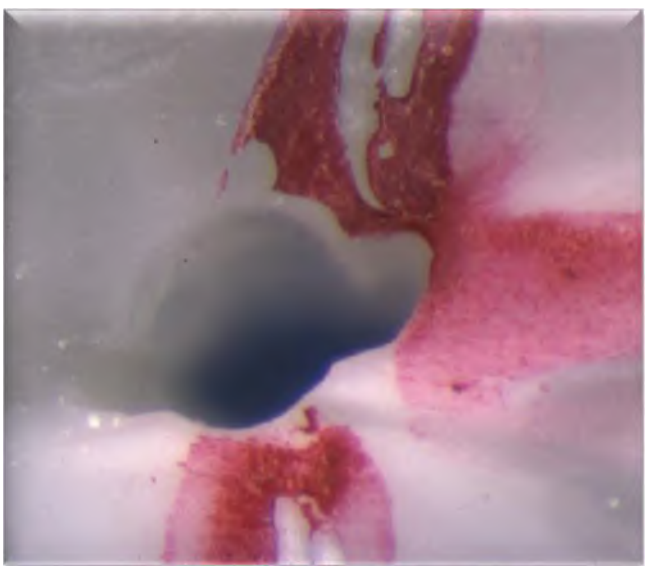


FIGURE 20-87 Postoperative image showing uninstrumented canal extensions that were cleaned by ultrasonic irrigation. The ultrasonic file was actively directed into the extensions.

In summary, it can be concluded that

- Preparation of oval, C-shaped and S-shaped root canal presents a challenge for preparation and disinfection.
- Complete mechanical preparation of the endodontic system in roots with anatomic irregularities may not always be possible.
- SAF NiTi system may offer some advantage in preparing a greater percentage of the root canal walls without excessive weakening of the root.
- Proper chemical disinfection is necessary to compensate for the drawbacks of mechanical preparation in these cases.

CONTROVERSY IV—APICAL SIZE OF THE PREPARATION

There still is some debate on the desirable final size of apical preparation with two opposite concepts: maintaining the apical integrity of the root by preserving as much dentin as possible on one side vs. circumferential removal of the infected dentin on the other side.

Traditionally, it has been suggested to prepare at least three sizes beyond the first file that binds at working length. Based on findings that the preoperative diameter of the apical foramen widely varies[278-289] (Table 20-9). Apical preparation has been recommended to ISO-sizes of at least 25 to 35 or even higer.[278-289]

Coldero *et al.*[290] found no significant differences in reduction of intracanal bacteria following crown-down preparation to size .04/35 and crown-down with GT to size .04/20 followed by stepback to .04/35. With both techniques, the majority of the root canals were rendered bacteria-free. Shuping *et al.*[291] reported on decreasing numbers of intracanal bacteria with increasing instrumentation sizes. In contrast, in a comparative study of five preparation techniques with the size of the final apical file varying from size 25 to size 40, no differences in remaining pulp tissue or debris were found.[292]

Regarding irrigation, not only the apical diameter but also the conicity of the preparation has to be considered. Boutzioukis et al.[293,294] suggested a size .04/40 as appropriate to achieve good exchange of irrigants even in the most apical part of the root canal.

The second concept aims to keep the apical diameter small and includes scouting of the apical third, establishing apical patency with a size 10 instrument passively inserted 1mm through the foramen, gauging and tuning the apical third and finally finishing the apical third at least to size 20.[295] Yared and colleagues[296] in an *in vivo* investigation in 60 single-rooted teeth with apical periodontitis found no significant differences in bacteria reduction following preparation to size 25 or 40, respectively. Bacterial counts were reduced significantly in both groups when compared to the initial number of microorganisms. Card *et al.*[297] could demonstrate *in vivo*

TABLE 20-9 Measured Apical Diameters and Suggested Preparation Sizes for Mandibular and Maxillary Teeth, Taken from Representative References Over the Last 60 Years

Maxillary Teeth										
Reference	**Kuttler (279)**	**Green (280)**	**Kerekes (281–283)**	**Morfis (284)**	**Wu (285)**	**Briseno (286)**	**Grossman (287)**	**Sabala (288)**	**Tronstad (289)**	**Glickman (290)**
Type	**268 teeth *in vitro***	**110 teeth *in vitro***	**220 teeth *in vitro***	**213 teeth *in vitro***	**180 teeth *in vitro***	**1097 teeth *in vitro***	**Suggestions**	**Review**	**Suggestion**	**Suggestion**
Centrals	25–35		45	30	34		80–90	80	70–90	35–60
Laterals	25–35		60	30	45		70–80	80	60–80	25–40
Canines	25–35		45		31		60–90	80	50–70	30–50
1st premolars	25–35	20	50	21	37		30–40	60	35–90	25–40
1 canal			70		30			60		
2 canals			20		23					
2nd premolars	25–35			21	37		50–55		35–90	25–40
1 canal			70		30			80		
2 canals			35		23			45–60		
Molars	25–35						30–55			
mesiobuccal		25	60	24	43	11–73		45	35–60	25–40
1 canal			40		19	09–60				
2 canals			40		19	11–44				
distobuccal		25	40	23	22	08–73		45	35–60	25–40
palatal		35	40	30	29	08–69		60	80–100	25–40
Mandibular Teeth										
Centrals	25–35		70	26	37		40–50	60	35–70	25–40
Laterals	25–35		70		37		40–50	60	35–70	25–40
Canines	25–35		70		47		50–55	80	50–70	30–50
1st premolars	25–35		35	27	35		30–40		35–70	30–50
1 canal					20			80		
2 canals					19			45–60		
2nd premolars	25–35		40	27	35		50–55		35–70	30–50
1 canal					20			80		
2 canals					13			45–60		
Molars	25–35						30–55			
mesial root		25	60	26	38–45	10–64		45	35–45	25–40
distal root		30	60	39	46	12–64		60	40–80	25–40

a significantly increased reduction of intracanal bacteria after apical enlargement to larger sizes with NiTi instruments. Preparation with NiTi instruments to size 50 achieved significantly larger reduction of bacteria than preparation to size 35.[298]

Conclusions

Mechanical preparation of the root canal results in a significant reduction of microorganisms independently from the preparation size. Complete microbial-free root canals cannot be achieved.

- Although there seems to be some evidence that larger apical preparation sizes may result in more reduction of intracanal microorganisms, the significance of the final apical preparation size remains to be clarified.
- Preparation technique, instruments and final preparation size should be defined individually for each root canal system.
- Introduction of a gauge 30 irrigation needle to working length minus 1-2mm seems to be the best criterion for apical preparation size. An apical size of .04/40 has been suggested to produce effective irrigation.

COMPLICATIONS DURING PREPARATION

Canal Straightening and Ledging

The American Association of Endodontists (AAE) defines root canal transportation as:[299] "*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; may lead to ledge formation and possible perforation.*" Thus, the characteristic appearance of canal transportation is an asymmetrical dentin removal in the apical curved part of the root canal. As a result, the long axis of the curved root canal is displaced and the angle of curvature is decreased, resulting in a straightening of the original curvature of the root canal (Figure 20-88).

The etiology of canal transportation, as pointed out in the definition of the AAE, is based on the fact that independent of the alloy used, any root canal instrument tends to straighten itself inside the root canal.[69,300] Due to this tendency, an uneven distribution force of the cutting edges of the instrument in certain contact areas along the root canal wall results, leading to an asymmetrical dentin removal.[69,300] These contact areas

are located in the apical part of the canal at the outer side of the curve (convexity) and in the middle or coronal thirds at the inner side of the curve (concavity). As a result, apical canal areas tend to be over-prepared toward the convexity of the canal, whereas more coronally greater amounts of dentin are removed at the concavity, leading to canal transportation or straightening.[69]

In the apical part of curved root canals straightening is associated with further preparation errors[6,52]:

- Damage to the apical foramen: When the curved canal is deviated from the original long axis it is often associated with loss of the apical stop.[2] Clinically, it may result in mechanical irritation of the periapical tissues and increase the risk of extruding irrigation solutions, debris and root canal filling materials.
- Perforation: This procedural error represents a communication between the root canal space and the external root surface. It may be caused from use of instruments with sharp cutting tips in reaming or rotary working motions[2,3] (Figure 20-89). In many instances, a perforation will be located at the apical part toward the outer side of the canal.
- Strip perforation: In contrast to perforation located at the apical part of the canal, strip perforation results from over-preparation along the inner side of the curvature in the middle or coronal third of the canal.[2,42] Strip perforations mainly occur in mesial roots of mandibular molars at the furcal aspect, also know as the "danger zone" (Figure 20-90).
- Zip: The curved part of the canal adopts an elliptical or "teardrop" shape at the apical end point.[52] This is caused because the axis of the curved canal is deviated towards the outer aspect and inadvertently more dentin is removed from the outer side. Due to this irregular cross-sectional shape, the quality of canal obturation is markedly reduced when employing the cold lateral compaction technique.[300]

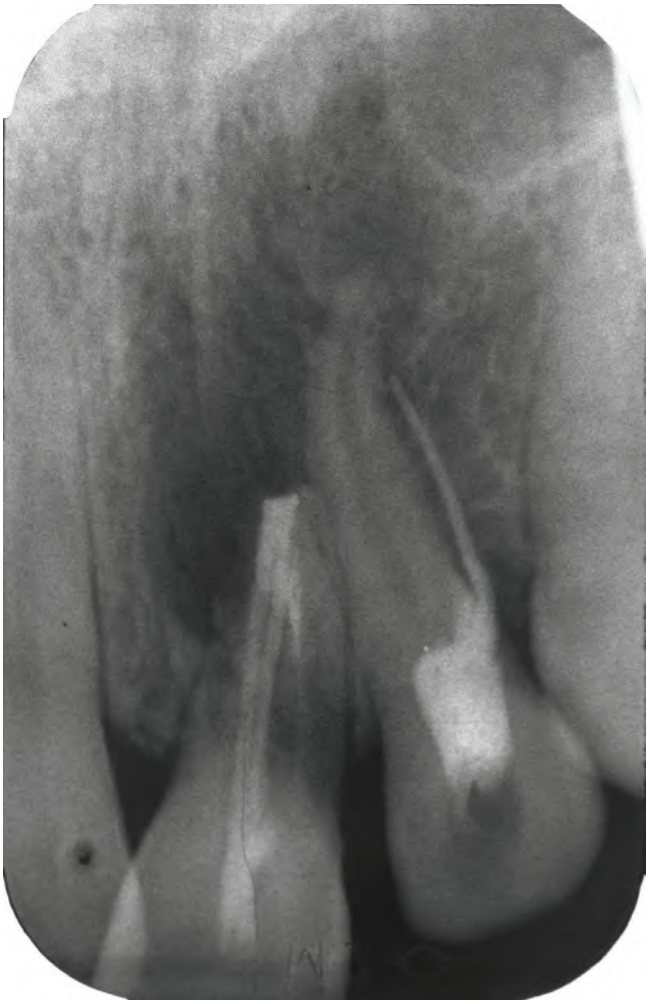


FIGURE 20-89 Uncommon perforation in a maxillary incisor.

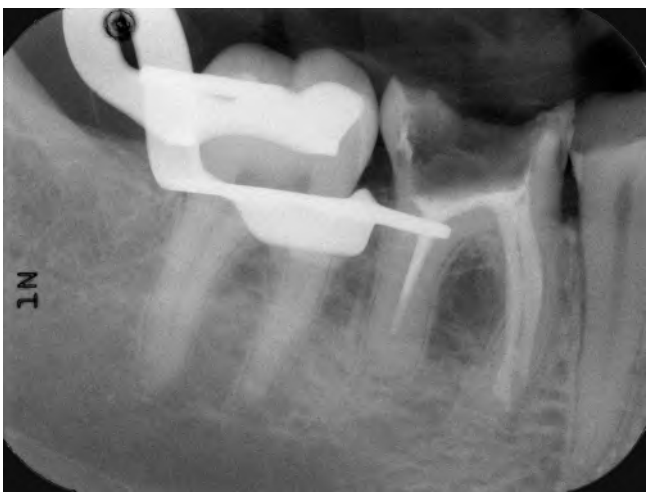


FIGURE 20-88 Root canal filling of a right mandibular molar demonstrating marked straightening of the mesial root canals with ledge and zip formation.

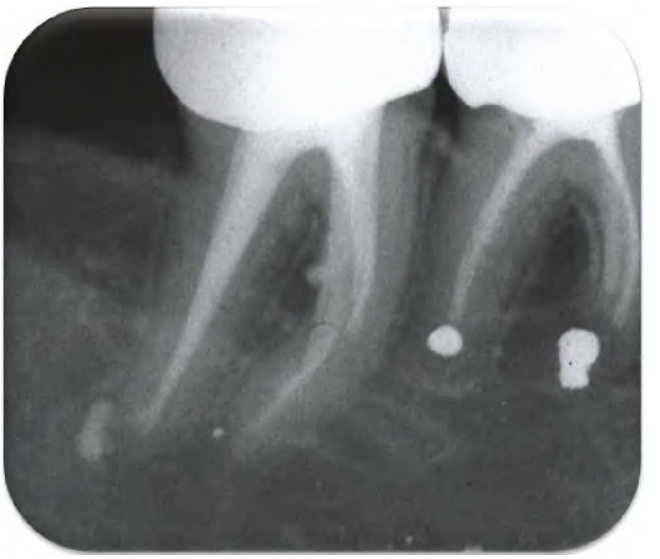


FIGURE 20-90 Strip perforation at the furcational aspect of the curve.

- Elbow-formation: The typical picture of canal transportation is over-preparation along the outer side of the curvature at the apical part and along the inner side more coronally. The elbow-formation usually occurs between two areas of excessive dentin removal and a narrow portion of the canal. Usually, the elbow is located at the point of maximum curvature. The clinically relevant adverse effects of this procedural errors are insufficient debridement and obturation of the canal part located apically to the elbow-formation.[5]
- Ledging: A ledge is a platform created at the beginning of the outer side of the curvature that hinders the instruments from reaching the working length.[2,65] (Figures 20-91 and 20-92). Usually, ledge formations are located in the middle or apical part of root canals.

There is evidence in the literature that the following factors impact the incidence of canal transportation:[69]

- Design of the access cavity
- Experience of the operator[92,301]
- Insufficient irrigation during mechanical root canal preparation[300]
- Properties of the instruments used for canal preparation[302]
- Use of instruments with sharp cutting tips[127,303]
- Instrumentation technique[2,147,304]
- Degree and radius of canal curvature. The greater the degree of curvature and the smaller the radius of curvature, the greater is the risk of canal transportation.[76,305] Preparation of root canals with a great angle and an abrupt short curve is challenging. These canals can hardly be enlarged without any transportation, independently whether rotary NiTi or stainless steel hand instruments were used[306] (Figure 20-93).

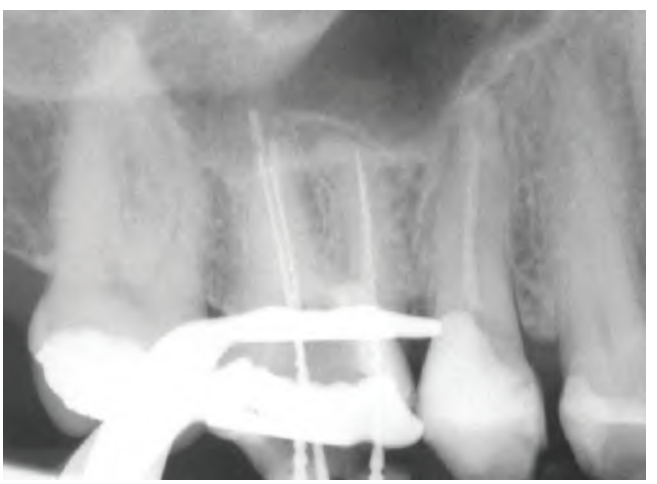


FIGURE 20-91 Ledge formation in the apical part of the mesial root canal in a maxillary molar.

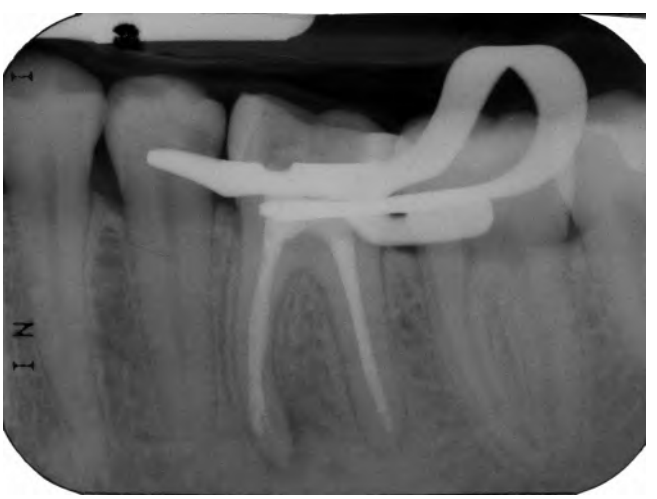
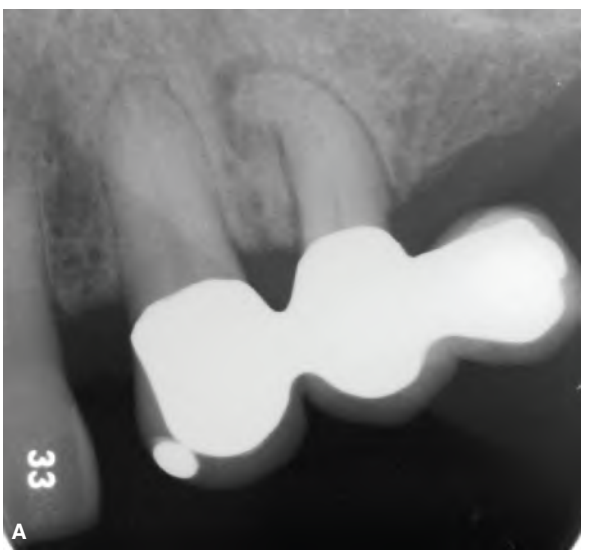


FIGURE 20-92 Root canal filling of a left mandibular molar with ledge formation, loss of working length and canal straightening in the mesial canals.



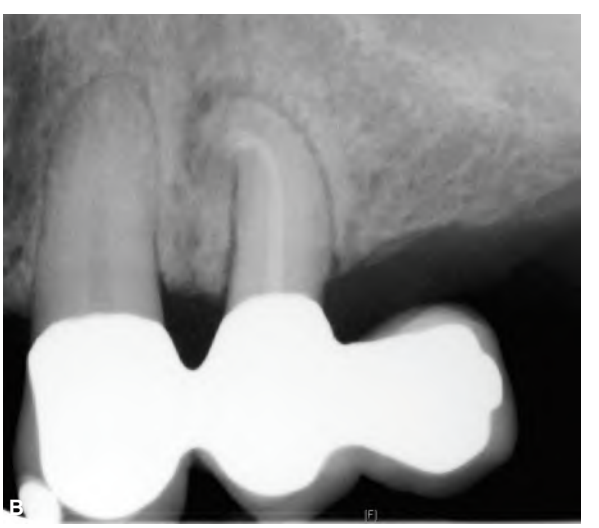


FIGURE 20-93 ***A.*** Maxillary premolar showing an abrupt curve of the root canal in the apical part. ***B.*** Postobturation radiograph. The canal was prepared using engine-driven nickel–titanium instruments.

- Radiographically unseen canal curvatures. In general, proximal curvatures cannot be detected using two-dimensional radiographs. Most often such curvatures are found in the mesial root of mandibular molars. These undetected curvatures may cause unexpected challenges during canal preparation, resulting in canal transportation or strip perforation.

From a clinical standpoint, the most relevant consequences of canal transportation and ledging are insufficient cleaning of the root canal system and over-reduction of radicular dentin (Figure 20-94).

Prevention of Canal Transportation and Ledging

The following is recommended to minimize the risk of canal transportation and ledging:

- The access cavity should be prepared so that unrestricted access of the instruments to the apical foramen is available (Figure 20-95).
- A glide path should be created prior to canal preparation. When using stainless steel hand instruments (e.g., Pilot instruments) for canal scouting, the tactile feedback provides relevant information regarding the consistency of the canal content, possible blockages (e.g., root canal branches) and radiographically unseen curvatures.
- Severely curved canals should be prepared using engine-driven NiTi instruments having non-cutting tips. When using NiTi hand instruments, preference should be given to the balanced-force technique.
- Instruments should not be forced into the root canal. Skipping instrument sizes should be avoided. Frequent recapitulation of the canal using small hand instruments is recommended.
- Copious irrigation should be carried out during canal preparation to avoid any blockage of the canal lumen. Instruments should never be used in dry canals.

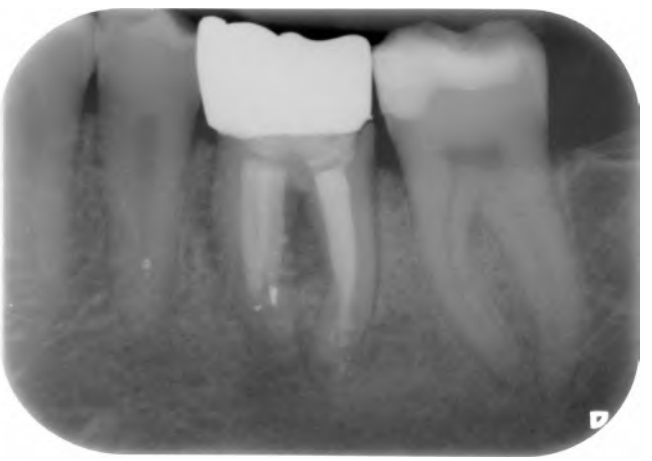


FIGURE 20-94 Root canal preparation of this mandibular molar resulted in over-reduction of radicular dentin and a strip perforation at the furcal side of the mesial root.

Management of Canal Transportation and Ledging

Canal Straightening

The extent of apical canal transportation can be classified as follows (Figure 20-96)[307]:

- Type I: minor movement of the position of the apical foramen, resulting in only slight iatrogenic relocation.
- Type II: moderate movement of the physiologic position of the foramen, resulting in a considerable iatrogenic relocation on the external root surface. The resulting communication between the root canal and the periapical tissues is much greater than in Type I. Moreover, it might be problematic to create a more coronal shape as this attempt might weaken or perforate the root.
- Type III: the physiologic position of the root canal is severely moved, resulting in a relevant iatrogenic relocation of the physiologic foramen.

Consequently, apical canal transportation may be managed by different specific treatment strategies:

In general, whenever transportation is noticed during root canal preparation, paper points and an electronic

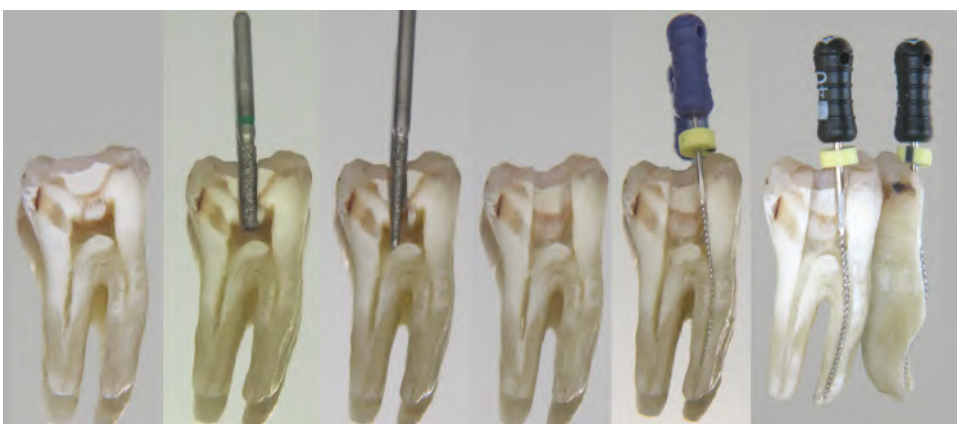


FIGURE 20-95 Adequate design of an access cavity. On one hand, this minimally invasive access cavity maintained healthy tooth structure but on the other hand allowed straight-line access of the instruments to the apical portion of the root canals (instrumentation: mesial, size 40, taper 0.06; distal, size 70, taper 0.02).

apex locator should be used to determine whether perforation has already occurred. In case a perforation is probable, the localization of the suspected perforation should be depicted radiographically with an inserted gutta-percha point or a sterile root canal instrument (Figure 20-97). If possible, the perforation should be sealed using mineral trioxide aggregate (MTA) and pre-bent stainless steel instruments may be used to bypass this area to regain access to the apical canal system (Figure 20-98).

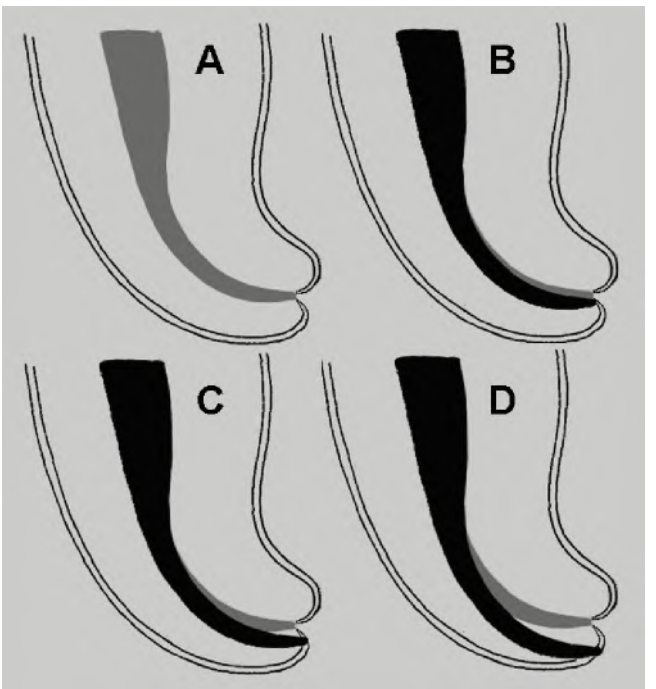


FIGURE 20-96 Schematic illustration of different types of root canal transportation. ***A***. Physiological appearance of a curved root canal. ***B***. Type I canal transportation (minor movement of apical foramen). ***C***. Type II canal transportation (moderate movement of apical foramen). ***D***. Type III canal transportation (severe movement of apical foramen).

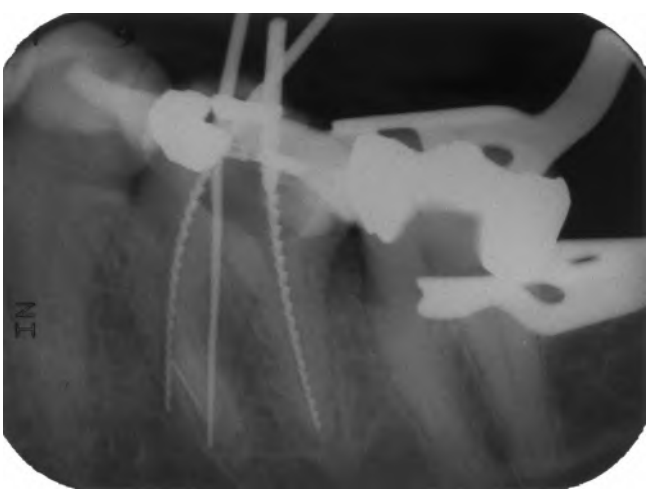


FIGURE 20-97 An attempt to bypass a separated instrument resulted in perforation in the mesial root.

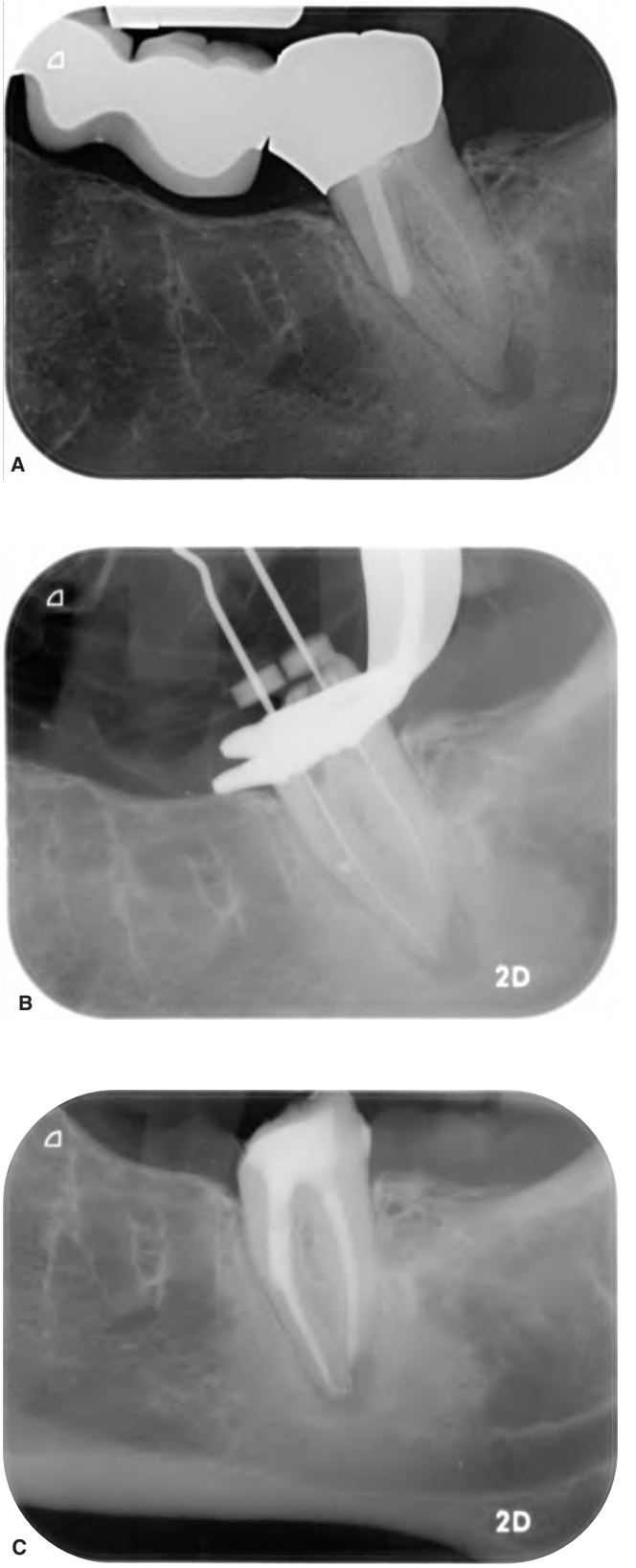


FIGURE 20-98 ***A***. Marked ledging with potential perforation in the mesial resulted in an inadequate post placement; ***B***. Following removal of the bridge and the post the ledge was sealed using MTA and access to the original root canal was regained; ***C***. Post-obturation radiograph.

Since canal transportation is associated with insufficient cleaning of the entire root canal, the canal will need to be irrigated copiously and using passive ultrasonic irrigation in order to remove tissue residues, microorganisms and debris from the non-instrumented canal areas. A thermoplastic obturation technique is preferable in any root canals showing marked transportation (Figure 20-98). For more details, see Chapter 23.

Ledge

The characteristic feature of canal ledging is the sensation of hitting against a hard structure with a non-binding instrument tip when probing the canal.[51] Frequently, ledging is associated with blockage of the canal. This compaction of debris and dentin in the adjoining canal system jeopardizes bypassing of the ledge and regaining access to the canal lumen located apically to the ledge.

In the case of canal ledging, the coronal canal part, up to the ledge, should be enlarged in such way that instruments can be used up to the ledge without any friction. This allows a more targeted use of pre-bent instruments that should be used to try to bypass the ledge. (Figure 20-99). NiTi instruments are less suitable for this procedure than stainless steel instruments as they are considerably more flexible. For more details, see Chapter 23, Endodontic Orthograde Retreatment and Managements of Mishaps.

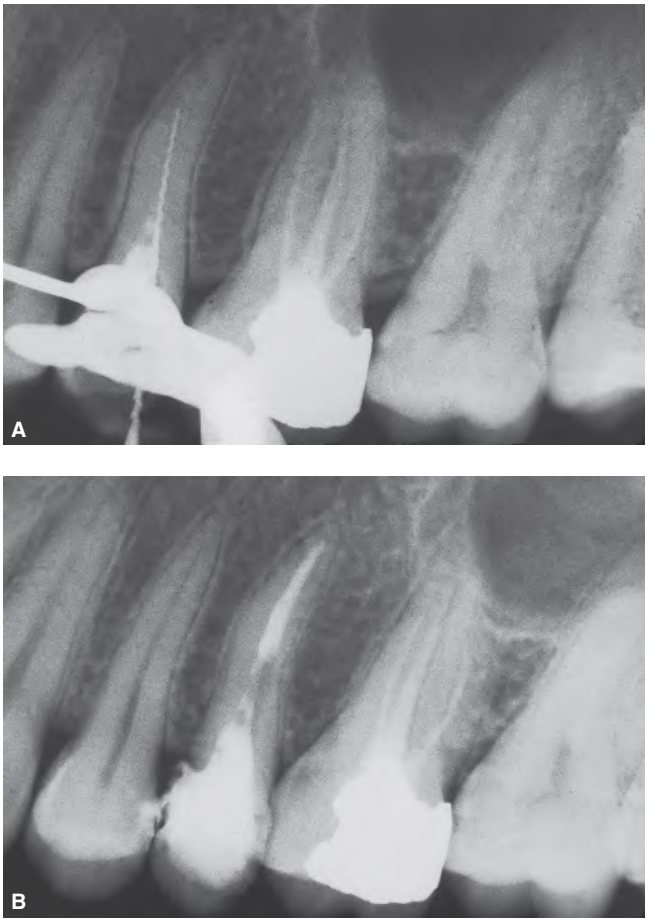


FIGURE 20-99 ***A***, Ledge on the outer side of a slightly curved apical part of the root. ***B***, The ledge was bypassed and rounded off with manual stainless steel instruments.

In summary it can be concluded that:

- Transportation may destroy the integrity of the root and lead to apical perforation.
- Transportation may result in insufficient cleaning of the root canal system.
- Transportation may result in over-reduction of radicular dentin, that may lead to a decreased fracture resistance of the entire root and to strip perforation along the inner side of the curvature.
- Root canals enlarged with instruments of greater taper are more prone to fracture than those prepared with instruments with a smaller taper.
- Straightened root canals should be copiously irrigated.
- Straightened root canals should be obturated using thermoplastic techniques.

Fracture of Instruments

> "The dentist who has not fractured the tip of a reamer, file, or broach has not treated many root canals. When one accepts the challenge of a curved, narrow or tortuous canal, one also assumes the risk of instrument fracture… Considering the delicate diameter of the instrument tip that is expected to cut a substance as hard as dentine, it is remarkable that so few instruments are broken….Who has not felt the pang, the anguish, the mortification caused by the breaking of an instrument? That moment of remorse lives on for days until it is faded out by time" (*L. I. Grossman 1969*)[308]

The fracture of endodontic instruments is not an uncommon mishap during root canal preparation. It may occur in 1%–6% of cases for stainless steel instruments and approximately 0.4%–3.7% for rotary NiTi instruments.[309] Instrument fracture is an inherent risk of root canal preparation. The patient has to be informed of this risk and its consequences before starting root canal treatment in order to avoid (or at least minimize) medico-legal consequences.

Basically, there are two modes of fracture: cyclic fracture and torsional fracture.

Cyclic fracture occurs when an instrument is used for too long. Unfortunately many manufacturers focus on the number of canals treated rather than the duration. A single torturous canal may, often, require a duration exceeding that of several non-complicated root canals.

Torsional fracture occurs when the tip of an instrument is firmly engaged inside the root canal and the motor continues to rotate. The torque will exceed the elastic limit and the instrument will separate. Single overload of NiTi instruments has been identified as a major reason of fractures.[310] It can also occur when the flutes of an instrument are packed with dentinal debris thereby increasing the torque above a critical limit.

The following factors were associated with file fracture: [311]

- Frequency of use
- Number of rotations
- Preflaring and preenlarging
- Angle of the curvature
- Radius of the curvature

Non-contributing factors are:

- Torque
- Sterilization

Controversial factors:

- Rotational speed
- Operator's experience

It has also been reported that larger instruments show faster deterioration of quality than smaller ones.[312,313] Crown-down approaches[314] and preparation of a glide path[38] seem to reduce the number of fractures.

Prevention of Fractures

The basic recommendations to prevent fractures of instruments are:

- Straight-line access (Figures 20-100).
- Proper preoperative radiographic and tactile evaluation of degree and radius of the curvatures and adjustment of size and taper according to the instruments (Figure 20-101).

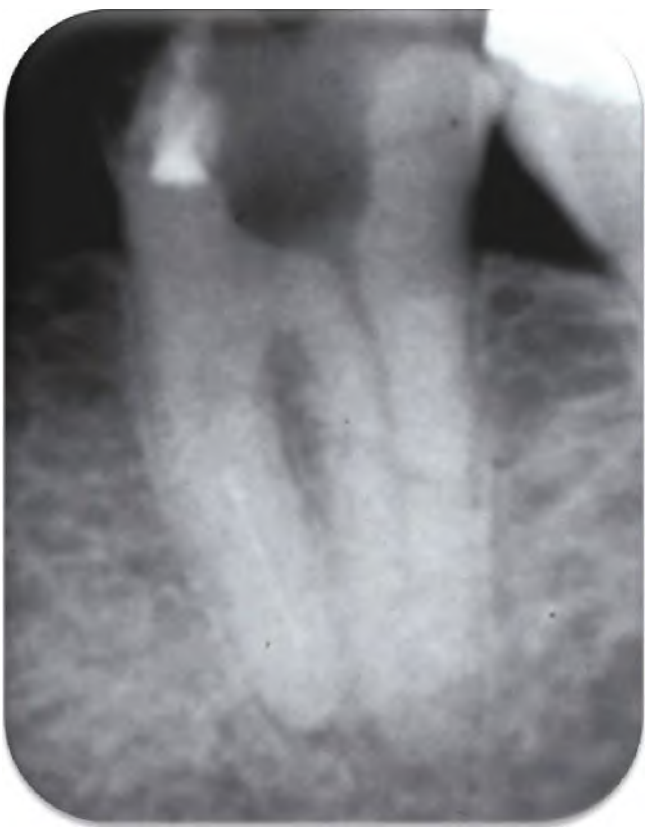


FIGURE 20-100 Incorrect access cavity resulting in instrument fracture.

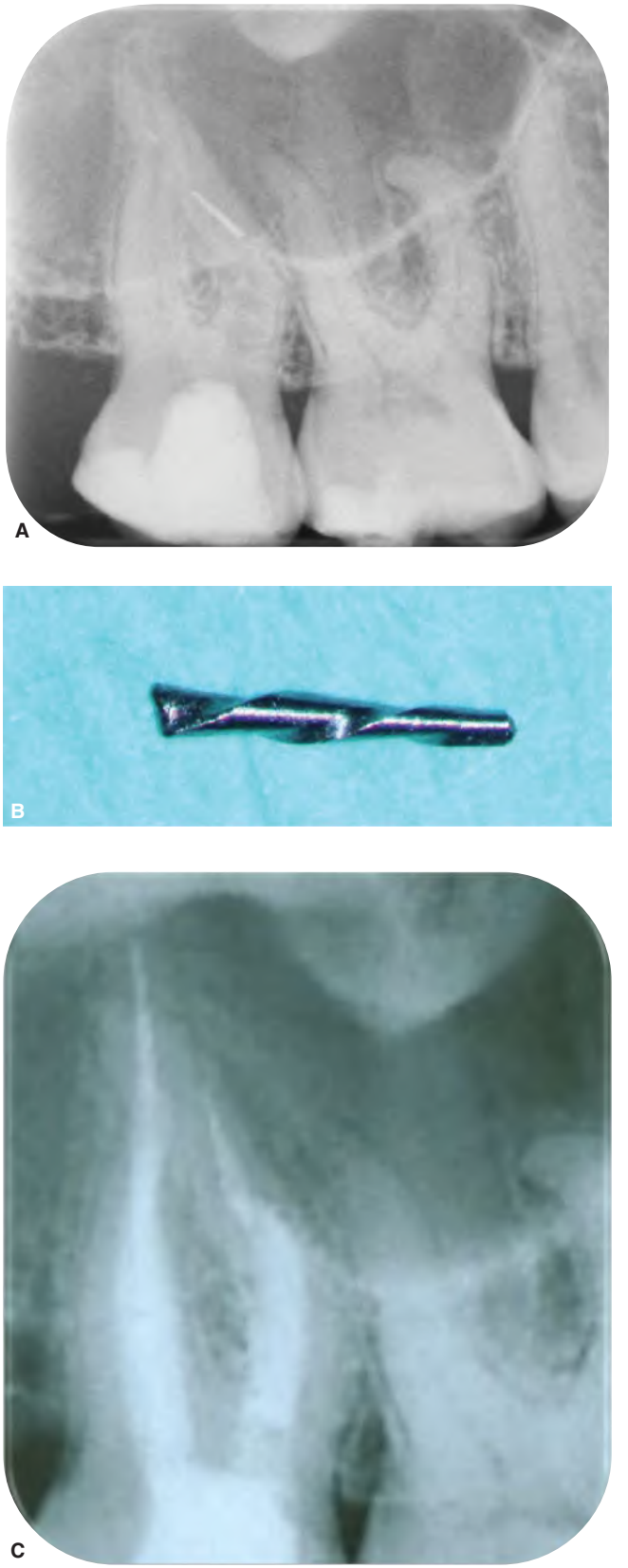


FIGURE 20-101 ***A.*** Fracture of a .08/25 NiTi instrument in a severely curved MB-2 canal. The use of greater tapered instruments in root canals with a small radius of curvature should be regarded as dangerous with a high risk of fracture. ***B.*** Removed fragment. ***C.*** Post-obturation follow up.

- Use of a motor with constant speed; no change of rotational speed during preparation.
- Use of a motor with defined torques, ideally individually adjusted for each file to be used for the preparation.
- Careful manual preparation of a glide path, before use of rotary instruments.
- Visual control of new and used instruments for any deformations (Figure 20-102).
- Passive use of instruments.
- Copious and constant irrigation during canal preparation.
- Use of instruments, only for few seconds, and early replacement of used instruments (single-use instruments are preferred)

Consequences of Instrument Fractures

A fractured instrument may be regarded as a stainless-steel or a NiTi root canal filling. However, this view overlooks two major problems. First, preparation and disinfection at the time of fracture usually have not been completed or performed to a sufficient degree. Second, the fragment has not been carefully and tightly adapted to the shape of the root canal, and in most cases, will not provide the necessary seal.

Regarding diagnosis and preparation technique, there are some basic differences:

- In a tooth with a necrotic pulp, 100% disinfection of the root canal space cannot be achieved. This may result in treatment failure.
- In a non-infected tooth with a vital, but irreversibly inflamed pulp, the most apical part of the pulp cannot be extirpated completely. Nevertheless this apical pulp stump may remain vital.
- When a small instrument fractures in an early stage of treatment, disinfection and removal of tissue are insufficient.
- If a large instrument fractures at a late stage of treatment, several cycles of preparation and irrigation already have been performed resulting in an advanced stage of disinfection at time of separation.

FIGURE 20-102 New instrument (left) and distorted instrument (right).

- When a large instrument fractures during a crown-down preparation, it may be screwed into the area the earlier instrument has created (taper lock), resulting in some congruence between fragment and root canal lumen. The result may be regarded as a NiTi root canal filling with questionable quality of seal.[93,315]
- When a small instrument fractures, there will be hardly any congruence between fragment and root canal.

These factors have to be thoroughly considered when discussing the necessity and technique of fragment removal. It has been shown that clinical success may be expected, even in cases with remaining fragments. However, several studies have shown that instrument fracture during treatment of teeth with apical periodontitis resulted in reduced success rate.[62,63,315,316] A recent meta-analysis study, on the impact of retained fractured instruments, on the outcome of root canal treatment has shown that teeth, either with or without periradicular pathosis, the prognosis of treatment was not significantly reduced.[317]

Additionally, several anatomical and technical factors may play a role when considering instrument removal, making this decision process more complex. Madarati et al.[318] offered a decision-making flowchart for that purpose. Also, several different techniques and instruments for removal of fractured instruments have been described. For more details, see Chapter 23.

IMPACT OF PREPARATION ON TREATMENT OUTCOME

Regarding the improved clinically relevant attributes of NiTi instruments, it is reasonable to assume, and therefore often claim that the use of NiTi hand or engine-driven instruments results in improved clinical outcomes.[319] However, very few clinical trials were conducted, with the main focus on the influence of specific instruments or preparation techniques on treatment outcome.[320-322]

When assessing the impact of root canal preparation on treatment outcome different outcome measures can be considered. The strictest criterion is "periapical health," whereby evaluating success is based on healing of periapical periodontitis, with periapical health (evaluated clinically and radiologically). Meanwhile "tooth survival" has been accepted by the American Association of Endodontists, as a further meaningful and relevant outcome measure. Finally, the criterion "flare-up" should be considered, when evaluating success as absence of interappointment emergency.[323]

Recently, a focused review attempted to systematically assess the impact of root canal preparation using NiTi hand or engine-driven instruments on treatment outcome.[324] This review concluded that evidence is limited regarding the impact of the type of root canal preparation, on treatment outcome of initial nonsurgical root canal treatment.

The available evidence suggests that:

- Preparation to larger apical sizes seems to result in increased success rates when treating teeth with necrotic pulps and periapical lesions,[325] and that preparation to a master apical file sizes of less than size 35, seems to be associated with increased healing time.[326]
- Better maintenance of the original canal curvature and shape seems associated with increased success rates.[327]
- Ledging of root canals seems to reduce success rates[327] and procedural errors seem to decrease treatment outcome.[326]
- The use of NiTi hand and engine-driven instruments does not seem to have a significant effect on long-term success rate of endodontic treatment.[328-331]

For a detailed discussion on treatment outcome, see Chapter 33, Outcome of Endodontic Therapy.

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Chapter 21

Irrigants and Intracanal Medicaments

Markus Haapasalo, Ya Shen, James S. Lin, Ellen Park, Wei Qian, Zhejun Wang

HOST DEFENSE AND INFECTIONS

The human body is well prepared to deal with various kinds of infectious challenges. Non-specific and specific immune responses maintain a healthy balance between the normal microbiota and the host. In cases where the first line of defense, the epithelium, is broken, the host's defense system will quickly limit the invasion of microbes into the tissue and eventually eliminate bacteria and other microorganisms. Occasionally, systemic antibiotic therapy or manipulative treatment, such as drainage of pus, is applied in order to resolve the infection. Elimination of endodontic infection, however, follows a different path. Host measures that are sufficient to eliminate the infective microorganisms at other sites of the body, are unable to completely eliminate endodontic infections because of the special anatomical challenges. Eradication of an endodontic infection is achieved by a combination of several host and treatment factors.[1–3] Success in all parts of the treatment will be needed for the elimination of the infection and healing of periapical pathosis. The sequence of events and procedures in control of endodontic infections are host defense system, systemic antibiotic therapy (rarely used, only with specific indications), instrumentation and irrigation ("cleaning and shaping"), intracanal medicaments (not always used) between appointments, permanent root canal filling, and coronal restoration.[1–3]

The host defense is responsible for the prevention of root canal infections spreading to the bone and to other parts of the body. Osteoclast cells resorb the bone surrounding the apical foramen and occasionally around the external openings of large, infected lateral canals. Bone is replaced by highly vascularized inflammatory tissue,[4–6] that is of significant importance in order to keep the bacteria from spreading into the bone and causing osteomyelitis. This defense is usually successful in stabilizing the lesion size and preventing its expansion after the initial growth period. However, because of the lack of circulation, the host defense mechanisms cannot effectively reach and attack the microbes residing inside the tooth, in the necrotic root canal system. While mechanical instrumentation removes a portion of the microbes from the main root canal space, its main purpose is to enhance irrigation and the placement of medication in the root filling. Irrigation supports mechanical instrumentation, by reducing friction and removing dead and living microbes from the root canal.[7] In addition, many irrigating solutions have tissue dissolution and/or antimicrobial activity and effectively clean the canal space and kill residual microbes in the canal and surrounding dentin. Intracanal antimicrobial medicaments are used in multi-appointment endodontic treatments to complete the work started by instrumentation and irrigation and, optimally, to render the root canal system bacteria-free.[1]

Several early studies that examined the presence of bacteria in the root canal indicated that a bacteria-free canal at the time of root filling is a prerequisite for high success rate, and the use of calcium hydroxide [$Ca(OH)_2$] as an intracanal medicament will predictably help reach this goal.[8–11] However, since then a number of other studies have challenged these results, and presently there is no clear consensus regarding the use of intracanal interappointment medicaments, and the microbiological and other advantages from their use.[12–14] Despite such unanswered questions, there is no disagreement of the microbiological goal of the treatment of apical periodontitis, eradication of the microbiota in the root canal system.[15]

This chapter focuses on the methods of irrigation, properties and function of different irrigants, and the role of irrigation and intracanal medication in killing and reducing the number of microorganisms in the root canal system. It is important, however, to always keep in mind, that irrigation and locally used antimicrobial intracanal dressings are part of a concerted effort to eliminate the microorganisms causing endodontic infections. Irrigation and disinfection alone cannot guarantee success if there are problems in the quality of other parts of the treatment.

IS COMPLETE ELIMINATION OF MICROBES FROM THE ROOT CANAL SYSTEM POSSIBLE?

Pulpitis is caused by microorganisms or their antigens, entering the pulp from a carious lesion or leaking filling through dentinal tubules. Although the pulp tissue is affected by inflammation, as long as the pulp remains vital, the number of bacteria in the pulp is considered to be minimal and of no clinical significance.[16] Established biofilms, that are the most resistant to endodontic treatment procedures, are not expected to be found in the root canal space during pulpitis. However, with proceeding infection, necrosis and apical periodontitis, the entire root canal system becomes invaded by microorganisms. It has been shown beyond doubt that microorganisms are the etiological factor

of apical periodontitis (Figure 21-1).[17–19] It has also been suggested that, in a small number of cases, non-microbial factors are responsible for the persistence of the lesion after treatment,[20,21] but opposing views have also been expressed.[15] For more details, see Chapter 3, "Microbial and non-microbial etiologies of endodontic disease."

There is a general consensus that, in an optimal situation, the goal of endodontic treatment is to remove and kill all microorganisms in the root canal and to neutralize any antigens that may be left in the canal. Reaching this goal is expected to guarantee healing of periapical lesions. In a high number of cases, high quality treatment is followed by complete healing.[22–24] However, complete killing and elimination of root canal microbes is a particularly difficult challenge. It has also been shown that a majority of cases harboring living bacteria at the time of filling have healed completely.[8] It is therefore clear that although complete elimination of the infective flora is the optimal goal, complete clinical and radiographic healing can also occur when the microbiological goal of the treatment has not been fully achieved. This situation may be compared to marginal periodontitis and gingivitis where, understandably, it is not possible to gain a totally microbial-free environment in the gingival crevice area. It is evident that good quality periodontal treatment will result in the healing of the periodontal disease.

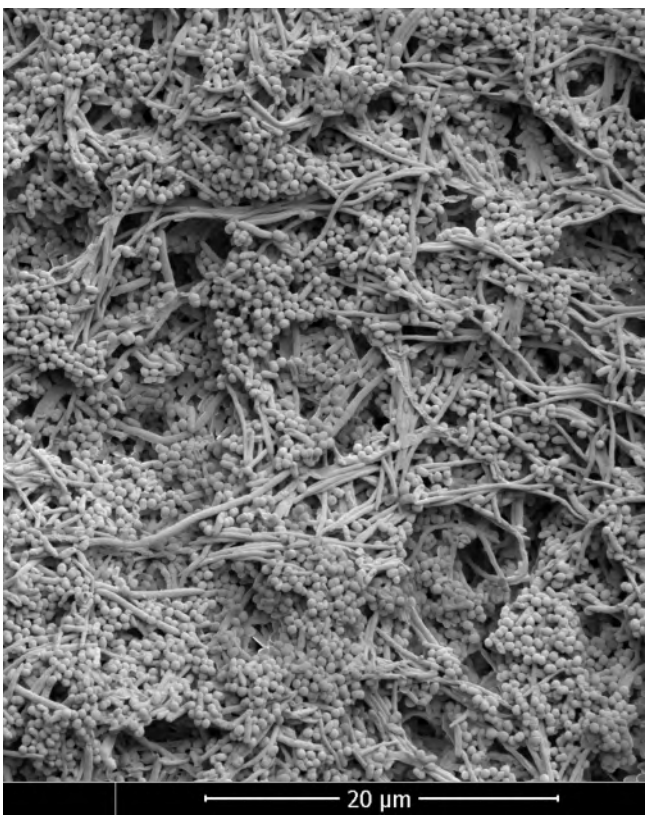

FIGURE 21-1 Microbial biofilm in the root canal is the cause of apical periodontitis and therefore the main target of irrigation and disinfection.

THE GOAL OF ENDODONTIC TREATMENT

The detailed goal of endodontic treatment depends on the diagnosis before treatment is started. In apical periodontitis, that is always caused by microbes, the main goal is the elimination of the microbiota from the pulp chamber and the root canal system. Maintaining or re-establishing the structural integrity of the tooth is another major goal of the treatment. In pulpitis, root canals are microbial free, therefore the goal is the prevention of microbial invasion into the root canal space and thereby deeper into the tissues.

In other words, the goal is prevention or elimination of a microbial infection in the root canal system.[25] In some special situations, such as resorptions and endodontic complications, there may be several intermediate goals of a more technical nature. Even then, the final success is dependent on successful infection control and/or preserving the structural strength of the tooth. There is a widely accepted view that instrumentation and irrigation ("cleaning and shaping") of the root canal system is the most important step toward a canal free from microbes.

THE ROLE OF INSTRUMENTATION IN THE ANTIMICROBIAL STRATEGY IN ENDODONTICS

The goal of instrumentation and irrigation is to remove all necrotic and vital organic tissue, as well as some hard tissue, from the root canal system. Instrumentation shapes the canal system to facilitate irrigation, placement of local medicaments, and placement of a permanent root filling. From a biological point of view, the key objective of instrumentation and irrigation is to remove and eradicate the microorganisms residing in the necrotic root canal system. Furthermore, the goal is to neutralize any residual antigenic material remaining in the canal after instrumentation and irrigation.

The classical Swedish studies from the 1970's have greatly influenced our understanding of the effects of instrumentation and irrigation on the intracanal microflora.[26] In a series of studies on teeth with apical periodontitis, the authors demonstrated that thorough mechanical instrumentation with stainless steel hand instruments, together with irrigation with either physiological saline, ethylenediaminetetra-acetic acid (EDTA), or EDTA and dilute sodium hypochlorite (NaOCl), they were unable to predictably produce bacteria-free root canals. Fifteen root canals were instrumented at five sequential appointments and sampled at the beginning and end of each appointment. The access cavity was sealed with a microbial-tight temporary filling, but the canals were left empty between the appointments. This procedure resulted in a 100 to 1000-fold reduction in bacterial numbers, but it was difficult, or in fact impossible, to obtain completely bacteria-free root canals.[26,27] Corresponding results were also reported by Ørstavik et al.[28]

and Cvek et al.[29] in teeth with closed and immature apices. The antibacterial effect of mechanical cleansing with sterile saline was reported to be very low and limited in teeth with fully developed roots. NaOCl increased the antibacterial effect compared to saline irrigation. Interestingly, no statistical difference was found in the antibacterial effect between 0.5% and 5.0% NaOCl solutions.[29]

The effects of instrumentation and irrigation were investigated in a series of studies by Dalton et al.[30] The authors measured the reduction in microbial counts in 48 patients, in teeth instrumented with 0.04 taper nickel–titanium (NiTi) rotary instruments or with stainless steel K-files using the step back technique with saline for irrigation. Bacteriological samples were obtained before, during, and after instrumentation. All teeth with apical periodontitis yielded positive growth at the beginning, whereas the control teeth with vital pulp and irreversible pulpitis were sterile. A reduction in bacterial counts was detected with progressive enlargement of the root canals with both techniques. However, only 28% of the teeth became bacteria-free following instrumentation. It is important to keep in mind that the results of root canal microbiological studies are only as accurate as the sensitivity of the methods to detect microbes. Cultural methods used in even earlier studies fail to obtain growth (and detection) of fastidious anaerobic bacteria. Moreover, sampling was performed, by paper points or files, in the same areas where the instrument action had been most effective. One can assume that the mechanical removal of microorganisms had been more effective in these areas than more remote, untouched parts of the root canal system. Therefore, use of ultrasound to facilitate the detachment of bacteria from such locations for sampling may give a more reliable picture of the residual microbiota. Even molecular technics depend on effective sampling and a minimum number of cells to allow detection by PCR and probes. However, there are no studies comparing the effect of ultrasound on microbial sampling from the root canal.

Observations similar to Dalton et al.[30] were reported by Siqueira et al.[31] when saline was used for irrigation. Interestingly, this former study showed that increasing the size of apical preparation from #30 to #40 resulted in a significant reduction in microbial counts. *Ex vivo* studies by Pataky et al.[32] using 40 human first maxillary premolars extracted for orthodontic reasons also verified the difficulty of obtained sterility of the infected canal space by instrumentation and saline irrigation. Although a considerable reduction in bacterial counts was detected after instrumentation, none of the teeth became bacteria-free. It should be mentioned, however, that the size of the master apical file in some of the studies was quite small, #25, and that may increase the possibility of positive cultures.[32] It can be concluded that instrumentation and irrigation with saline or directly antimicrobial agents such as sodium hypochlorite cannot predictably eliminate all microorganisms from infected root canals. Therefore, it is not surprising that the focus of activity in root canal disinfection is placed on the development and use of irrigating methods and solutions and other intracanal disinfecting agents with strong cleaning effectiveness and antibacterial activity. In recent years there has been considerable activity in the development of new methods of irrigation which would add effective mechanical or "energy" components to the hydrodynamic, chemical irrigation.[33,34]

RESEARCH ON ENDODONTIC IRRIGATION AND DISINFECTION: METHODS, MODELS, AND CHALLENGES

Our understanding and perceptions of the various root canal irrigants, irrigation methods, and their effects are based partly on clinical experience but also, and even more so, on *in vitro*, *ex vivo*, and *in vivo* studies of the effects, risks, and benefits of irrigation. Studying irrigation is, perhaps surprisingly, not easy.[35] One of the biggest challenges is the special microanatomy of teeth and the root canal system. Also, the difficulty to truly simulate the *in vivo* events in an *in vitro* setting adds to the challenge. In the following, different research strategies and models on irrigation are being critically discussed.

In Vitro Models to Test Antimicrobial Activity

The testing of the antimicrobial activity of various chemical compounds used in endodontics may appear to be straightforward and a simple procedure. In theory this may be true where the microbes of interest are exposed to the antimicrobial agent to be examined. At certain time intervals, microbiological samples are collected and cultured on suitable media. The results are then expressed as the length of time required to kill all microbes. The reality with endodontic disinfecting agents is, however, quite different.[35] Research focusing on the antimicrobial effectiveness of irrigating solutions and temporarily used intracanal medicaments has, in many occasions, adopted techniques originally designed for some other context; for example, testing bacterial susceptibility to systemic antibiotics using agar plates. Testing of systemically used antibiotics is based on tens of years of international standardization.[36–40] The chemical composition of both the antibiotic discs and the culture media used in testing are defined in detail in order to secure predictable diffusion of the active ingredient of the antibiotic and the absence of confounding chemical reactions between the agar medium ingredients and the antibiotic in question. Zones of growth inhibition around the antibiotic disc have been compared through clinical studies to the serum and tissue concentrations that can be reached with safe dosages of low toxicity, as well as assessing the clinical effectiveness of the antibiotics. In addition, reference strains from culture collections with known antibiotic susceptibilities are used as internal standards for quality testing of each new batch of plates. Most aerobic and facultative bacteria can be reliably tested using the disc diffusion method. However, despite years of extensive research, there is still no generally accepted

standard for susceptibility testing anaerobic bacteria using the disc diffusion method.[41] E-test strips impregnated with antibiotics are used to test growth of anaerobic bacteria on specific plates.[42,43] It offers advantages over conventional agar diffusion test with anaerobic bacteria, and has been used also in endodontic research.[44,45] There are no corresponding strip tests for endodontic disinfecting agents or solutions.

The use of agar diffusion method in endodontics, despite good intentions, is not based on standardization of the media or the tested materials. Chemical interactions between the media and the disinfecting agents are largely unknown. Furthermore, there are no true comparative studies enabling to draw conclusions from the size of zones of inhibition to the expected performance of the disinfectants *in vivo* against root canal bacteria. The antimicrobial effect of some endodontic medicaments is based on the pH effect. The buffering capacity of the agar plate is key to determine the diameter of the growth inhibition zone. Another example is EDTA, that causes a zone of inhibition on an agar plate, but fails to reduce the number of viable microbes even after 24-hour incubation in a test tube. Wang et al.[46] reported in a study of irrigant antibacterial effect in dentin that EDTA caused false positive reaction also when viability stain and confocal microscopy were used.

Another matter of importance in testing the antimicrobial effectiveness of different compounds, liquids, or solid materials is understanding the difference between bacteriostatic and bactericidal activities. Bacteriostatic action means prevention of growth of the microbial cells without killing them. Bactericidal activity, on the other hand, kills the affected microbes. In endodontic studies, this difference is not always clearly addressed, and the results are often reported as "antibacterial activity." The agar diffusion test, when properly used in systemic antibiotic testing, is an example of measuring bacteriostatic activity. In endodontics, a bactericidal effect of a disinfecting agent is more important than a bacteriostatic effect. In the necrotic root canal, a temporary prevention of bacterial growth (bacteriostatic) occurs as long as the disinfecting agent remains in the canal is of limited value, because the microbes may still grow back in numbers afterwards and cause a new challenge to the host.

Testing endodontic disinfecting agents *in vitro* can be done in test tubes using a mixture of the microbes (suspended in sterile water) and the medicament. The presence of a culture medium in the bacteria-medicament mixture is commonly reported in endodontic literature. This may pose a problem, because culture medium or other organic material is a confounding factor that may inactivate the disinfecting agent or protect the microbes by some mechanism.[47] Experiments done under such conditions may not give a reliable picture of the characteristics and activity of the medicament/disinfectant against the tested microbe.

"Carry-over effect" means that the medicament (chlorhexidine for example) is carried over along with the sample and the microbes into the dilution series and further to the culture (plate or liquid culture). On the culture medium, even lower concentration of the "carried" medicament may have either bactericidal or bacteriostatic effect and prevent microbial growth. A high enough concentration of the disinfectant, carried to the culture medium, can prevent microbial growth and thus cause a false-negative result.[47] Therefore, carry-over, if undetected, contributes to an excessively positive picture of the antibacterial effectiveness of the medicament. Endodontic irrigants and disinfecting agents, containing local antibiotics, are at particularly high risk of causing false-negative results. Effective inactivators are not available for many antibiotics.[47]

Specific inactivating agents are used to prevent the effects of carry-over. Citric acid has been used in the root canal to neutralize $Ca(OH)_2$, sodium thiosulfate neutralizes NaOCl,[48] and a mixture of Tween 80 and alpha-lecithin inactivates chlorhexidine (CHX).[49] However, inactivation is dependent on the concentration of the medicaments. When medicaments are used in high initial concentration, their inactivation may not be complete. An example of this is CHX that cannot be effectively inactivated with Tween and alpha-lecithin if CHX concentrations of 1% or more are used (Figure 21-2). The importance of the careful design of the experiments and proper controls cannot be overestimated, in order to avoid the possibility of false negative results (Figure 21-3).[47]

Ex Vivo and *In Vivo* Models

In vitro models may give valuable information about the antimicrobial potential of endodontic disinfecting agents. However, information from the *in vitro* experiments is not enough to predict their performance in a clinical situation in the root canal. Despite good *in vitro* results, the effectiveness of the medicament *in vivo* can be weakened by a variety of local factors in the root canal. These include problems in the delivery of the disinfecting agent to all parts of the canal, low overall volume, poor penetration into dentin, short contact time, or inactivation of the activity of the disinfecting

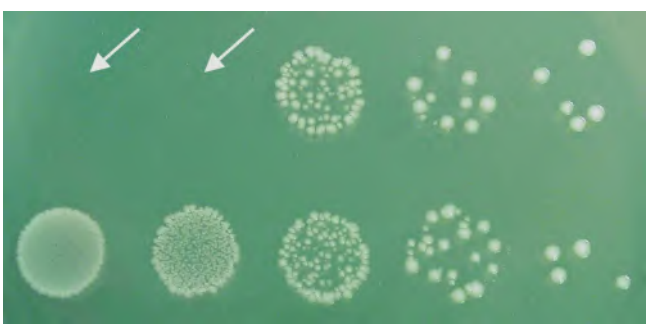

FIGURE 21-2 Samples of *Enterococcus faecalis* from 10-fold dilution series after a brief contact with 2% chlorhexidine (CHX). Because of the short contact time (two seconds), no killing has taken place. However, CHX "carry-over" has prevented growth of the first two samples (arrows) on the plate (upper row, left) despite the presence of Tween-lecithin inactivator in the dilution series. Lower row: control sample with no CHX. If only the first two dilutions had been made after the two seconds CHX exposure, the result would have indicated complete killing of the microbes in just two seconds.

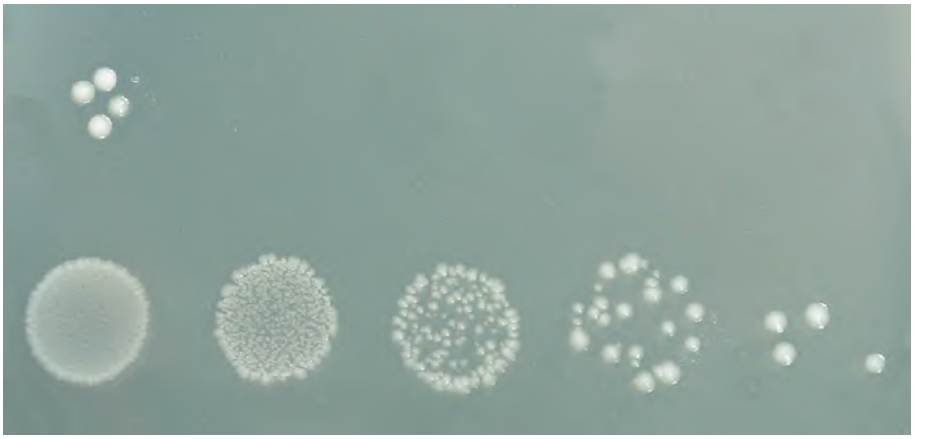

FIGURE 21-3 The size of the initial inoculum and the depth of the 10-fold dilution series determine the accuracy of measurement of the colony forming units. In this example, killing effectiveness can be calculated to a level of about 99.99%. Upper row: medicament has killed so many bacteria that only in the non-diluted specimen (leftmost) a few cells can be seen. Lower row: control specimen (water: no killing).

agent by the different chemical compounds present in the necrotic root canal. Therefore, a number of *ex vivo* and *in vivo* models have been developed in order to meet the challenge by the various confounding factors of the root canal and to improve the correlation between the test results and clinical performance. The *ex vivo* and *in vivo* models include the dentin block model,[50,51] dentin powder model,[52–54] and several modifications using roots/root canals from extracted teeth.[55,56] Recently, a dentin canal model with a massive presence of bacteria in dentinal tubules was developed and used for testing endodontic materials for their antimicrobial activity using confocal microscopy and viability staining.[46,57] Ultimately, clinical and *in vivo* studies are needed.[58,59]

The dentin powder model, with its modifications, makes it possible to obtain information about the inhibition of the medicament activity by dentin and other compounds (biomolecules, microbial biomass, etc.) in various concentrations.[52] It also allows standardization of certain experimental conditions for large series of tests. Prolonged incubation time to create dentin infection is not required because the dentin is powdered. The downsides of the powder model include partial loss of the microanatomical structure of the tooth and the difficulty to create and use biofilms.

The dentin block model has been widely used for testing endodontic medicaments.[50,51] The benefits of the model include simulation of the chemical and microanatomical environment of the tooth and the root canal system. The root canal can be standardized in size, making it easier to obtain comparable samples of dentin in different blocks. It also allows the use of microbial biofilms in the experiments. On the other hand, the dentin block model is quite laborious in use; it cannot be fully standardized as different blocks may vary in thickness and dentin microstructure. Handling of the block presents some challenges that may increase the risk for false-positive results (contaminations from outside the sampling area). A major limitation of the dentin block model with culturing is the limited number of bacteria invading the dentin of root canals as well as inaccuracies in sampling and culturing. Despite its limitations, the dentin block model has contributed to our understanding of dentin disinfection. While it is still used in endodontic research, more advanced methods have started to replace the "classical" block-culture model.

Newer *ex vivo* and *in vivo* models use "natural" root canals either in extracted teeth or directly *in vivo*. The obvious benefit of these experiments is a better simulation of the clinical situation; the results obtained with the various materials may best reflect their true activity clinically. However, in addition to the well-known difficulty to organize and run controlled clinical studies, and to have enough patients for such studies, there are also other potential pitfalls that may weaken the usefulness of the results. These include the difficulty to standardize the size of the apical preparation because of the great natural variation in canal sizes, different total volume of the canals, variations in the microanatomy of the root canals, and differences in the quality and quantity of the microbial infection. In addition to this kind of natural variation, there does not seem to be any standard way of dealing with the smear layer or taking the microbiological samples.[35]

New Biofilm Models

Open biofilm models are nowadays used to measure the effectiveness of antimicrobial solutions (such as NaOCl, CHX) and other endodontic materials (e.g., sealers) against bacteria polymicrobial biofilms.[60–62] In this model, standardized hydroxyapatite or dentin discs are used as the biofilm substrate on which monospecies or multispecies biofilms are grown in liquid media under microaerophilic or anaerobic atmosphere (Figure 21-4).[60–62] Biofilms are exposed to endodontic irrigating solutions, and killing of the bacteria in the biofilm can be measured using culturing or, preferably, by confocal laser scanning microscopy. One of the clear benefits of this model is that true *in vivo* like biofilms can be grown directly from oral/periodontal/root canal bacteria, "cloning" the same biofilm to numerous parallel discs. The model

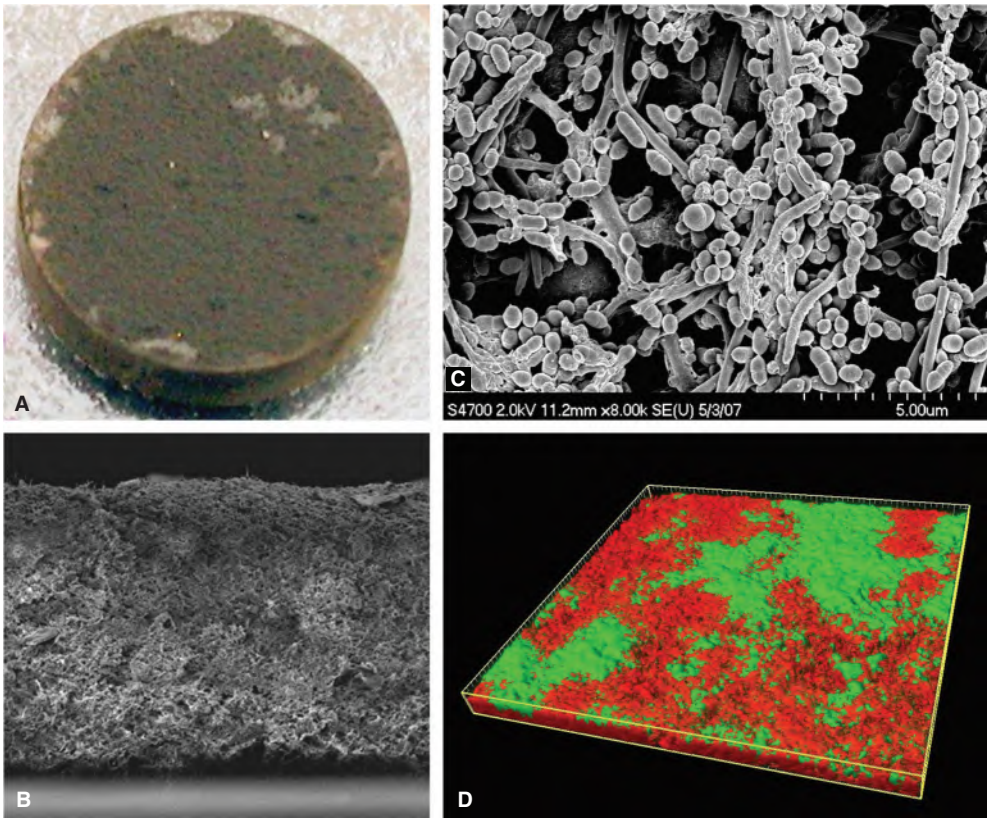

FIGURE 21-4 Research on the effectiveness of irrigation requires good biofilm models. ***A***. Multispecies biofilm grown on a collagen coated hydroxyapatite disc. ***B***. Lateral view of the biofilm, ***C***. SEM image of bacteria on the surface of the biofilm. ***D***. Confocal laser scanning microscopic image of biofilm stained with a "Viability stain" that shows killed bacteria red and live bacteria green.

allows following the disinfectant efficacy along time against biofilms of different age.

Stojicic et al.[62] examined the effect of iodine, NaOCl, and CHX on young and old biofilms and reported that multispecies biofilms from the oral cavity changed from sensitive to resistant to all three medicament groups tested after three weeks of biofilm growth. This is clinically important information as the true *in vivo* root canal biofilms are almost always older than three weeks when the treatment of apical periodontitis is started. After three weeks (and up to 2 months) there was no change in the resistance of the biofilm bacteria to the agents.

Dentin canal infection produced by centrifugation was introduced a few years ago in an effort to improve standardization of the experimental conditions. High numbers of bacteria are forced deep into dentin using the force of centrifugation. The two most important benefits of the model are the predictability of the presence of bacteria in most dentin canals in high numbers that makes it possible to quantitatively measure bacterial killing by confocal microscope, and the presence of the true microanatomy and chemistry of dentin.[46,57] In most of the published studies dentin has not been heat sterilized because possible contamination is not a quantitative problem, therefore dentin chemistry remains unaltered. Figure 21-5 shows a specimen that has been processed using these techniques after exposure to an antibacterial substance and viability staining.

Irrigation Hydrodynamics and Computational Irrigation Models

Irrigation is a balance between effectiveness and safety.[63] In optimal irrigation, the liquids should circulate effectively in all parts of the root canal system, including the main root canal, the area next to apical foramen (but not beyond), fins and isthmuses, and even lateral canals within the tooth structure. The prerequisite for effective positive pressure irrigation is high flow rate and concentration of the irrigant, and deep positioning of the irrigation needle. These may increase apical pressure by the irrigation that may, in some situations, contribute to extrusion of the irrigant into periapical tissues, tissue damage, and even severe pain.[64,65] Studies on irrigation hydrodynamics target this area of irrigation by analyzing patterns of irrigant flow in the canal system, shear stress forces at the canal wall, and pressure created towards the apical foramen.[66,67] Some of these approaches can only be done using computational fluid dynamics models based on complex mathematical calculations.[67–69] Models using small tubes or extracted teeth have been described where small pressure sensors have been used to measure the apical pressure created under a variety of irrigation parameters.[66,70] Recently, several studies have directly measured irrigant extrusion through the apical foramen under standardized conditions in order to better understand the risk of extrusion.[71–73] While these

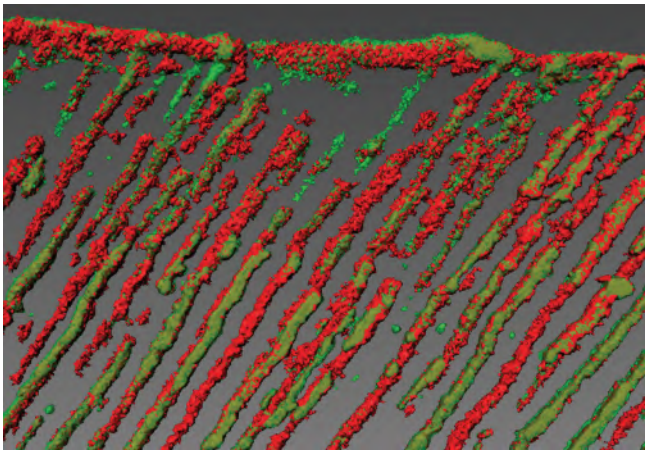

FIGURE 21-5 A root dentin specimen showing killed (red) and live (green) bacteria in dentin canal after hypochlorite irrigation and viability staining and Confocal microscopy. The dentin infection model allows studying the effect of irrigation on bacteria inside the dentin canals.

studies have already increased our understanding of irrigation, more studies are needed to better understand how well these new models reflect the reality of irrigation.

Qualitative or Quantitative Results; Detection Level: More Challenges?

Optimally, the goal of endodontic disinfection is the complete eradication of the microbes from the root canal system of the affected tooth. Although difficult to achieve, this noble goal may have affected the study design of several studies as the results are often expressed qualitatively, only as "growth" or "no growth." This approach may in some cases hide differences in the effectiveness of different treatment protocols and chemicals used for disinfection. For example, two different treatments that cause a 10% and 99.95% reduction in bacterial counts per canal, respectively, may be reported as equal ("growth"). It is possible that some of the poor results with $Ca(OH)_2$ as a disinfecting agent can be partly explained by the fact that qualitative rather than a quantitative approach has been used to measure the effectiveness of the disinfection. Quantitative measurement of the antimicrobial effect of endodontic disinfecting agents should therefore be preferred over qualitative approach. Accordingly, quantitative reporting has been used in some studies.[47,74]

There are a number of factors that create challenges for accurate and comparable counting of microbes in the samples. Residual medicament in dentin may result in a very low number of colony-forming units (CFU) per sample unless neutralizing agents are properly used. Dispersing the sample effectively may be required to detach the viable cells from the dentin chips, in order to increase the CFU numbers to correctly reflect the true number of microbes in the sample. Collection of small dentin samples is technically demanding and requires great care to secure a standardized yield from all samples. When the overall number of CFU in the sample is low, that is often the situation in infected and medicated dentin, it is important not to lose the microbes during a too vigorous dilution process.

Success in both qualitative and quantitative measurements of (surviving) microbes is dependent, among other factors mentioned above, on the design of the microbiological procedures that determine the detection level of the method. Detection level dictates whether the most effective killing can be announced, for example, on the level of 99%, 99.9%, or 99.99% (Figure 21-3). In other words, detection level tells how many cells in the test mixture must be alive so that growth can be detected by the culturing. Detection level is mainly dependent on the total number of cells in the initial reaction mixture/sample, proportion of the initial sample transferred into the dilution series, and the proportion of the mixture plated from each dilution. Theoretically, the detection limit can be easily calculated from the above numbers. In practice, detection limit is often reduced by the carry-over of strong medicaments such as CHX or other compounds (antibiotics for example) that may cause false-negative results. Detection limit provides important information about the design and quality of the methods in studies of endodontic disinfecting agents.[47] Unfortunately, detection limits are only rarely reported, making it more difficult to compare the results from different studies. Hopefully in the future, reporting the detection limit will be a routine requirement for studies on endodontic disinfection.

IRRIGATING SOLUTIONS

The use of irrigating solutions is an important part of endodontic treatment.[63] The irrigants facilitate the removal of necrotic tissue, microorganisms, and dentin chips from the root canal by a flushing action. Irrigants can also help prevent packing infected hard and soft tissue apically in the root canal and into the periapical area. Some irrigating solutions dissolve either organic or inorganic tissue. Finally, several irrigating solutions exhibit antimicrobial activity by actively killing bacteria and yeasts when in direct contact with the microorganisms. On the negative side, many irrigating solutions have cytotoxic activity or may cause severe pain reaction if accidentally pushed into the periapical tissues.[75]

An optimal irrigant should have all or most of the positive characteristics listed above, with none of the negative or harmful properties. Presently, none of the available irrigating solutions can be regarded as optimal. However, with a combined use of selected products, irrigation will greatly contribute to successful outcome of treatment.

SODIUM HYPOCHLORITE (NaOCl)

Antibacterial Effect

Sodium hypochlorite (NaOCl) is the most widely used irrigating solution in endodontics. In water, NaOCl ionizes to produce Na^+ and the hypochlorite ion, OCl^-, that establishes

an equilibrium with hypochlorous acid, HOCl. Between pH 4 and 7, chlorine exists predominantly as HClO, the active moiety, whereas above pH 9, OCl^- predominates.[76] It is the hypochlorous acid that is responsible for bacteria inactivation, the OCl^- ion being less effective than the undissolved HOCl. Hypochlorous acid disrupts oxidative phosphorylation and other membrane-associated activities as well as DNA synthesis.[77,78]

NaOCl is used in concentrations varying from 0.5% to 8%. It is a very potent antimicrobial agent and effectively dissolves pulpal remnants and organic components of dentin (Figure 21-6). It is used both as an unbuffered solution at pH 11 in the concentrations mentioned above and buffered with a bicarbonate buffer (pH 9.0) usually as a 0.5% (Dakin's solution) or 1% solution.[76] Contradicting earlier statements, Zehnder et al.[79] reported that buffering had little effect on tissue dissolution, and Dakin's solution was equally effective on decayed (necrotic) and fresh tissues. In addition, no differences were recorded for the antibacterial properties of Dakin's solution with an equivalent unbuffered hypochlorite solution.[80] However, other studies have indicated that the pH of NaOCl must be reduced more than the level discussed by Zehnder.[79] Kuroiwa et al.[81] reported that the acid used to lower the pH of NaOCl had a significant effect on the antibacterial activity of the solution.

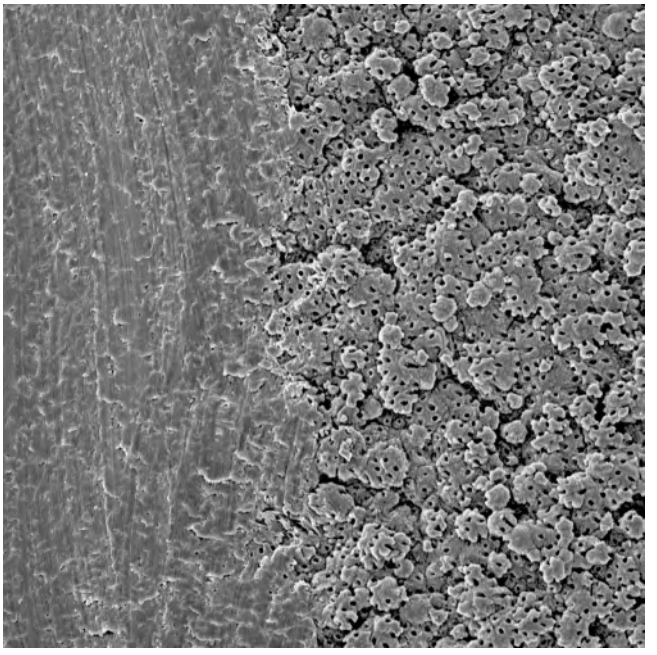

FIGURE 21-6 Instrumented (left half of the picture) and uninstrumented (right half) root canal wall after irrigation with 5% sodium hypochlorite (NaOCl) for several minutes. Pulpal remnants and predentin have been effectively removed with NaOCl while the instrumented part (smear layer) seems relatively unaffected. Notice the typical calcospherite structures at the left half of the image.

NaOCl is best known for its strong antimicrobial activity; it kills microorganisms very rapidly even at low concentrations. Waltimo et al.[82] showed that the resistant microorganism, *Candida albicans*, was killed in vitro in 30 seconds by both 5% and 0.5% NaOCl, whereas concentrations 0.05% and 0.005% were too weak to kill the yeast even after 24 hours of incubation. The high susceptibility of *C. albicans* to NaOCl was recently also verified by Radcliffe et al.[48] Later, Vianna et al.[83] contrasted these results partly, as 0.5% NaOCl required 30 minutes to kill *C. albicans*, whereas 5.25% solution killed all yeast cells in 15 seconds. In the latter study, however, organic material from the broth culture medium may have been present during the incubation with NaOCl that would explain delayed killing. Gomes et al.[84] tested *in vitro* the effect of various concentrations of NaOCl against enterococcus. *Enterococcus faecalis* was killed within 30 seconds by the 5.25% solution, whereas 10 and 30 minutes were required for killing all bacteria by 2.5% and 0.5% solutions, respectively. The higher resistance of *E. faecalis* to hypochlorite compared to the yeast *C. albicans* was suggested also by Radcliffe et al.[48] However, both of these studies are in contrast to the results reported by Haapasalo et al.[52] who demonstrated rapid killing of *E. faecalis* by 0.3% NaOCl, and by Portenier et al.[85] who showed rapid killing of *E. faecalis* strains in logarithmic and stationary growth phase by even 0.001% NaOCl. The reason for slow killing in some studies may be the presence of organic matter from the culture medium during testing which consumes the free chlorine.

Experiments with Gram-negative anaerobic rods *Porphyromonas gingivalis*, *Porphyromonas endodontalis*, and *Prevotella intermedia*, often isolated from apical periodontitis, demonstrated high susceptibility to NaOCl, and all three species were killed within 15 seconds with 0.5% to 5% concentrations of NaOCl.[83]

The differences between *in vitro* and *in vivo* studies include the volume of the medicament available for killing, access to all microbes, and absence of other materials in the *in vitro* experiments that potentially protect bacteria *in vivo*. Many of the *in vivo* studies have failed to show a better antimicrobial effect in the root canal by high concentrations of NaOCl as compared to low concentrations. Byström and Sundqvist[86,27] studied root canals naturally infected, mainly with a mixture of anaerobic bacteria. They showed that although 0.5% NaOCl, with or without EDTA, improved the antibacterial efficiency of preparations compared with saline irrigation, all canals were not bacteria-free even after several appointments. No significant difference in antibacterial efficiency *in vivo* between 0.5% and 5% NaOCl solutions was found in the study. Siqueira et al.,[87] using *E. faecalis*-infected root canals, demonstrated the superior antibacterial affect against root canal bacteria of hypochlorite in comparison with physiological saline. However, no difference was detected between 1%, 2.5%, and 5% NaOCl solutions.

The antimicrobial efficacy of NaOCl has traditionally been measured by culturing the microorganism on agar plates and counting the colonies (CFU).[47,50] Culturing works well in dynamic killing studies (along time) in planktonic suspensions, where the numbers of microbial cells can be easily standardized. However, in studies where sampling is done from contaminated root canals or infected dentin, the sensitivity of culturing faces many problems. Great variations in microbial loads at sampling sites next to each other often result in situations where the standard deviations are very high, that makes it difficult to detect significant differences. Therefore, while planktonic studies show great differences in killing *E. faecalis* cells at different metabolic phases, studies sampling from root canals usually fail to show differences.[87]

Recently, a new approach has been introduced to endodontic studies on the antimicrobial effect of various substances used in the treatment: confocal laser scanning microscopy (CLSM).[46,57] Bacteria are grown in biofilms, that are exposed to NaOCl or other irrigating solutions for different time periods and then analyzed for the proportions of dead and killed bacteria using a viability staining.[60–62] The BacLight viability stain is supposed to stain killed bacteria red, while the live bacteria stain green.[60] The method is based on the use of two fluorescent reagents that penetrate either all cells or just cells with a damaged cell wall. Shen et al.[61] using old, starved biofilms reported that while culturing was unable to detect any living bacteria in the biofilm, viability stain indicated 85% of the bacteria were still alive. The result may indicate that cultural studies from the root canal may contain an important element of uncertainty, as most biofilms in the root canal are likely to be old and in a state of starvation. Other new biofilm-CLSM studies have shown that there is a significant difference in the antibacterial potential of different NaOCl concentrations.[57] Biofilm-CLSM studies have also opened a new era for studies focusing in dentin disinfection.[46,57] These studies have shown the importance of NaOCl concentration in killing biofilm bacteria in dentin of root canals.[57]

In planktonic killing experiments, the death of 100% of the microbial cells usually is merely a question of time.[47] Contrary to this, new biofilm studies using CLSM clearly show that killing all bacteria in the biofilm is perhaps not even possible with the presently available substances and methods in endodontics. Shen et al.[61] and later Stojicic et al.[62] showed that after three weeks of growth, multispecies biofilms from oral bacteria changed from being sensitive to being much more resistant to NaOCl and other substances when used in root canal irrigation.

Sodium hypochlorite has low surface tension and can penetrate into dentin canals (Figure 21-7). Zou et al.[88] measured NaOCl penetration into dentin canals using stained dentin blocks and reported penetration between 77 and 300 μm, depending on the time of exposure. During 20 min of exposure, ca. half of the penetration depth was reached already after two minutes.[88] Using the dentin canal infection model and CLSM, Du et al.[89] reported that while 5% NaOCl killed 53% of *E. faecalis* cells in dentin during the first three minutes of exposure, the killing rate greatly reduced after that, and after 10 minutes almost no additional killing occurred despite refreshing the NaOCl (Figure 21-7). The explanation may be that unlike planktonic cells, biofilm contains sensitive areas and resistant areas. During the first

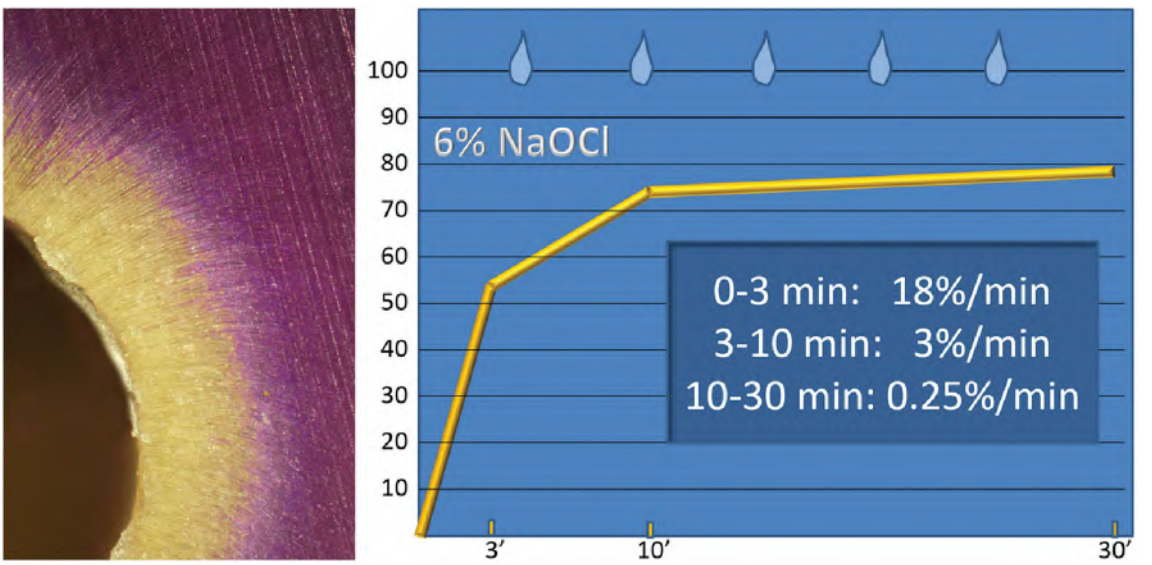

FIGURE 21-7 **Left:** NaOCl penetration into dentin canals as revealed by bleaching of stained dentin. **Right:** Dynamics of killing of *E. faecalis* in dentin canals during a 30-min exposure to 6% NaOCl. The hypochlorite solution was renewed every five minutes. After a rapid killing of bacterial cells during the first three minutes, the killing drastically slowed down, and after 10 minutes of exposure only little additional killing could be achieved. (Adapted from Du et al.[89])

few minutes bacteria in the sensitive areas are killed and after this time achieving more bacteria deaths becomes much more difficult.

Peciuliene et al.[90] studied the effect of instrumentation and NaOCl irrigation in failed, previously root-filled teeth with apical periodontitis. Old root fillings were removed with hand instruments, and the first sample was taken. No chloroform was used in order to avoid false-negative cultures. Bacteria were isolated in 33 of the 40 teeth before further instrumentation. *E. faecalis* was found in 21 teeth (in 11 as a pure culture), yeast *C. albicans* in six teeth, Gram-negative enteric rods in three teeth, and other microbes in 17 teeth. The canals were then hand instrumented to size #40 or larger and irrigated with 2.5% NaOCl (10 mL per canal) and 17% buffered EDTA (pH 7 and 5 mL per canal). After instrumentation and irrigation, *E. faecalis* was still detected in six canals. Other microbes persisted in five canals after preparation. Enteric Gram-negative rods were no longer present in the second sample.[90] The disappearance of yeasts but not *E. faecalis* in the root canals may reflect their different susceptibilities to the described chemomechanical instrumentation or it may be a result of different biofilm characteristics and dentin penetration by these species.

Tissue Dissolution

Dissolution of organic tissue is another main virtue of NaOCl.[63,79] In fact, NaOCl is the only commonly used root canal irrigating solution that dissolves organic matter. This is an important function, as it is crucial to dissolve and remove all necrotic tissue remnants from the canal. Otherwise, such tissue could serve as nutrients to residual microbes in the canal system. Tissue remnants would also have a negative impact on the quality of seal by the root filling. Several studies have shown that the tissue dissolving ability of NaOCl is dependent on its concentration.[91] The higher concentration, the faster soft tissue dissolution. Agitation, by sonic or ultrasonic means, and increased temperature also contribute to faster dissolution of tissue.[91] It has been reported that continuous agitation of 2% NaOCl dissolves soft tissue as fast as non-agitated 6% NaOCl solution. Similarly, heating the solution from 45°C to 60°C speeds up the dissolution.[91]

Sodium hypochlorite acts well on necrotic tissue whereas the effect on vital tissue is weaker.[92] This is good with regard to protection of vital tissue, for example, at perforation sites in the pulp chamber and in accidental or resorption perforations from the root canal to surrounding soft tissue or bone. The full concentration NaOCl can thus be used to disinfect pulp perforation sites before pulp capping. For more details, see Chapter 27. However, because of the limited effect on vital tissue, NaOCl may require more time in pulpitis treatments for the chemical cleaning of those areas of the root canal that are not mechanically cleaned.

In the main root canal where full concentration NaOCl has good access to the canal wall, necrotic tissue can be dissolved quickly; even in one to two minutes.[93] Lower concentrations can take considerably longer times. However, in fins and isthmus areas even prolonged irrigation with NaOCl does not guarantee complete cleaning of these locations.[94] Sodium hypochlorite does not dissolve mineralized tissue. This must be done either with EDTA, citric acid, or some other chelators/acidic compounds.[63]

Harmful Effects

The weaknesses of NaOCl include toxicity, unpleasant taste and its inability to remove the smear layer because of its lack of effect on inorganic material.[95,96] The poorer antimicrobial effectiveness of NaOCl *in vivo* than *in vitro* is also somewhat disappointing. There are several possible reasons for the reduced *in vivo* performance. Root canal anatomy, particularly the difficulty to effectively irrigate the most apical region of the canal, is generally acknowledged as one of the main challenges. In addition, the chemical milieu in the canal is different from a test tube. Haapasalo et al.[52] showed that the presence of dentin caused marked delays in the killing of *E. faecalis* by 1% NaOCl. The effect of other materials present in the necrotic or previously treated root canal, to the antimicrobial potential of NaOCl, has not been studied.

Studies measuring NaOCl cytotoxicity have indicated greater cytotoxicity and caustic effects on healthy tissue with 5.25% NaOCl compared to 1.0% and 0.5% solutions.[97,98] The fear of toxic and chemical complications is the main reason low concentrations 0.5 to 1% NaOCl solutions are used for canal irrigation, instead of the 5.25% solution that is used in many countries.[75] However, more *in vivo* studies on persistent endodontic infections and retreatment are necessary for a deeper understanding of the relationship between NaOCl concentration and its antimicrobial activity against specific microorganisms, before final conclusions can be drawn regarding the optimal NaOCl concentration.

Many dentists use NaOCl again after removing the smear layer with EDTA. However, when used after a chelating agent or an acid, NaOCl causes erosion of the root canal wall (Figure 21-8). While this aids the cleaning by removing the wall surface, it may also weaken the root dentin.[99–101] The reasons for vertical root fracture still remain partly unknown, it is therefore reasonable to avoid using NaOCl after smear layer removal, or at least limit its use to a short time of less than 30 seconds. For smear layer removal, a safer alternative would be to use a combination product that not only removes the layer but also kills microorganisms.

Ethylene Diamine Tetraacetic Acid (EDTA), Citric Acid, and Other Acids

Ethylene Diamine Tetraacetic Acid (EDTA) (17%, disodium salt, buffered to pH 7–7.5) has little if any antibacterial activity. On direct exposure for extended time, EDTA can extract some of the bacteria's cell surface proteins by combining with metal ions from the cell envelope. In theory, this

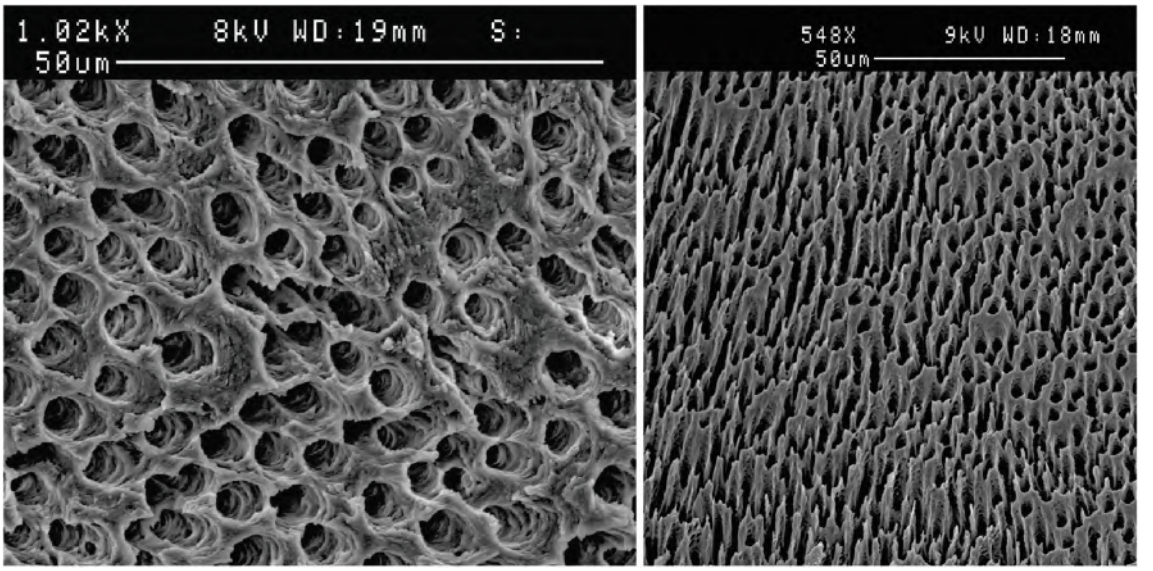

FIGURE 21-8 Erosion of root canal wall dentin after irrigation sequence NaOCl – EDTA – NaOCl. When EDTA or citric acid have been used to remove smear layer, dentin collagen on the surface and also deeper in dentin is exposed as EDTA removes hydroxyapatite. Using NaOCl again will attack the exposed collagen and cause dentin erosion, the magnitude of which depends on the length of the repeated exposure.

can cause even bacterial death. More importantly, EDTA is an effective chelating agent in the root canal.[102,103] It removes smear layer (Figures 21-9 and 21-10) when used together (but not simultaneously) with NaOCl by acting on the inorganic component of the dentin. Therefore, by facilitating cleaning and removal of infected tissue, EDTA contributes to the elimination of microorganisms in the root canal. It has also been shown that removal of the smear layer by EDTA (or citric acid) improves the antimicrobial effect of locally used disinfecting agents in deeper layers of dentin.[50,51] Niu et al.[101] studied the ultrastructure on canal walls after EDTA and EDTA plus NaOCl irrigation by scanning electron microscopy (SEM). More debris was removed by irrigation with EDTA and NaOCl than with EDTA alone. EGTA (ethylene glycol tetraacetic acid) also has the ability to chelate calcium from dentin, but it is weaker than EDTA.[99]

Citric acid can also be used for irrigation of the root canal and for removal of the smear layer.[102,104,105] Similar to EDTA, complete removal of the smear layer also requires irrigation with NaOCl before or after citric acid irrigation. Concentrations ranging from 1% to 50% have been used.[104] In comparison with ultrasound, 10% citric acid has been shown to remove the smear layer more effectively from apical root-end cavities than ultrasound.[106] In another study, powdered dentin-resin mixture was more soluble in a 0.5, 1, and 2 M citric acid than in 0.5 M EDTA.[107] Contrary to this, Liolios et al.[108] reported better removal of the smear layer by commercial EDTA preparations than with 50% citric acid, while other studies have found only small or no difference between citric acid and 15% EDTA in their capacity to remove the smear layer.[109,110] A comparative study showed that 10% citric acid was more effective than 1% citric acid, that was more effective than EDTA in demineralizing dentin.[111] Takeda et al.[112] reported that irrigation with 17% EDTA, 6% phosphoric acid, and 6% citric acid did not remove the entire smear layer from the root canal system. This is not unexpected, however, as it is known that NaOCl is also required for the complete removal of the smear layer. The acids demineralized the intertubular dentin making the tubular openings larger than when EDTA is used. The authors also showed that CO_2 laser was useful in removing the smear layer and that Er:YAG laser was even more effective than the CO_2 laser in smear layer removal. Qian et al.[99] reported stronger dentin erosion when citric acid was used after EDTA than when NaOCl was used after EDTA.

Smear layer removal facilitates penetration of sealers into the dentinal tubules. It also enhances the disinfection of the root canal wall and deeper layers of dentin. Both EDTA and citric acid can effectively remove the smear layer when used after NaOCl. Citric acid and EDTA may have weak antimicrobial activity as standalone products. However, their antimicrobial effectiveness has not been extensively documented and appears to be of minor importance.

Hydrogen Peroxide

Hydrogen peroxide (H_2O_2) is a biocide that has been widely used for disinfection and sterilization.[76] However, in endodontics, H_2O_2 has not been very popular, and presently it is rarely used. H_2O_2 is a clear and colorless liquid. Concentrations from 1% to 30% have been used. H_2O_2 produces hydroxyl-free radicals (HO.) that attack microbial components such as proteins and DNA.[76] H_2O_2 is non problematic from an environmental point of view because it degrades into water and oxygen. H_2O_2 is relatively stable in solution, but many products contain stabilizers to prevent decomposition. H_2O_2 has antimicrobial activity against

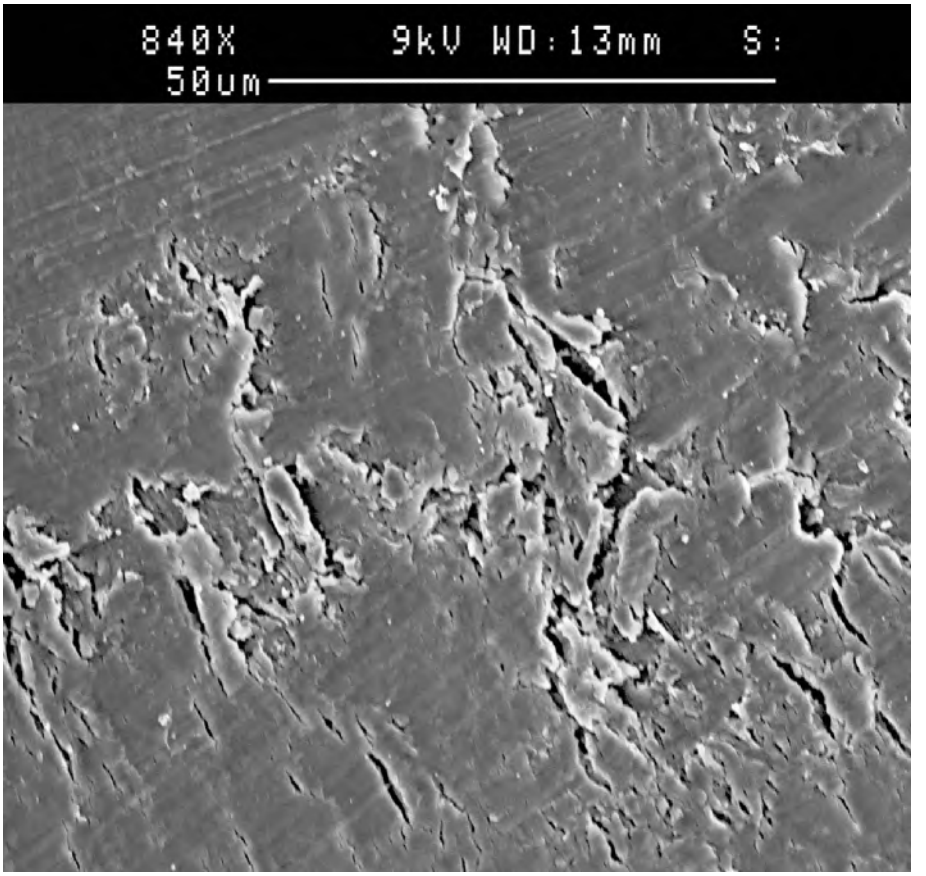

FIGURE 21-9 Smear layer on the canal wall after instrumentation and irrigation with NaOCl.

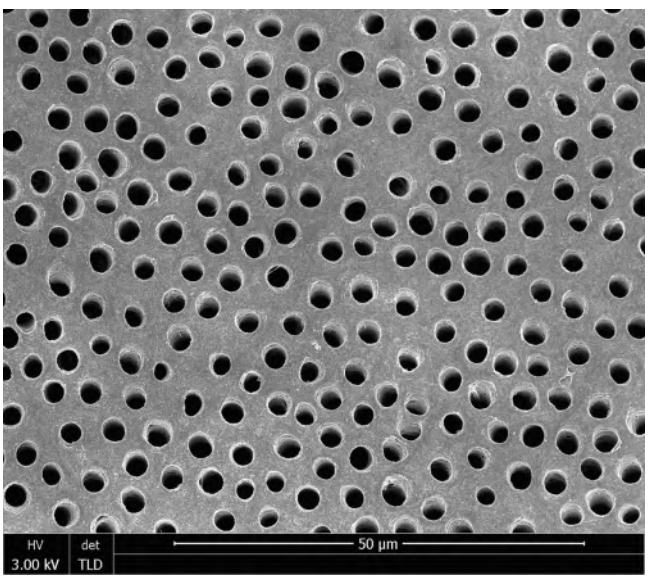

FIGURE 21-10 Root canal wall after removal of the smear layer. NaOCl removes at least partly the organic matter in smear layer, and EDTA (or citric acid) removes the inorganic matter, exposing the clean root canal wall and open dentin canals.

various microorganisms including viruses, bacteria, yeasts, and even bacterial spores.[113] It is more effective against Gram-positive than Gram-negative bacteria. Catalase or superoxide dismutase produced by several bacteria can provide them partial protection against H_2O_2.

Use of H_2O_2 in endodontics has been based on its antimicrobial and cleansing properties. Thirty percent H_2O_2 (Superoxol) has been recommended as the first step in tooth surface disinfection after mechanical cleaning in microbiological studies to prevent contamination from tooth surface.[114] H_2O_2 acts on the organic matter on the tooth making other disinfectants, such as iodine, more effective. It has been widely used earlier for cleaning the pulp chamber from blood and tissue remnants. It has also been used in canal irrigation, but evidence supporting the effectiveness of H_2O_2 as a root canal irrigant is scarce. On the contrary, Siqueira et al.[115] reported that a combination of NaOCl and H_2O_2 gave no advantage over NaOCl alone against *E. faecalis* in contaminated root canals *ex vivo*. Heling and Chandler[116] found strong synergism between H_2O_2 and CHX in disinfection of infected dentin: a combination of the two medicaments at low concentrations was far more effective in sterilizing dentin than these or any other medicament alone.[116] The synergistic mode of action between CHX and H_2O_2 was later documented by Steinberg et al.[117] In a recent study, 10% H_2O_2 was used as part of the irrigating protocol in monkey teeth.[118] Although the bacterial counts were greatly reduced, the study indicated the difficulty to completely eradicate bacteria from infected root canals in monkey teeth.[118] Al-Ali et al.[119] in a

recent study reported that alternating H_2O_2 with NaOCl was effective in removing soft tissues from root canal complexities.

Despite the long history of H_2O_2 use in endodontics, evidence supporting its use inside root canals is rather weak. It still has a role as part of the tooth surface disinfection protocol. Furthermore, the potential benefit of the suggested synergistic effect with CHX in deep dentin disinfection remains to be evaluated clinically.

Chlorhexidine Digluconate (CHX)

Chlorhexidine digluconate (CHX) is widely used in disinfection because of its excellent antimicrobial activity.[120–122] It has gained increased popularity in endodontics as an irrigating solution and as an intracanal medicament. Unlike NaOCl, CHX does not have a bad smell, it is not irritating to periapical tissues, and it does not cause spot bleaching of patients' clothes. Its antimicrobial effectiveness is well documented in endodontics. However, it completely lacks tissue-dissolving capability that is an important reason for the popularity of NaOCl (Figure 21-11).

CHX is perhaps the most widely used antimicrobial agent in antiseptic products. It permeates the cell wall or outer membrane (Gram-negative cells) and attacks the bacterial cytoplasmic and inner plasma membrane of the yeast. In high concentrations, CHX causes coagulation of intracellular components.[76] CHX gluconate has been in use for some time in dentistry because of its antimicrobial properties, its substantivity (long-term continued effect), and its relatively low toxicity compared to some other agents. However, the activity of CHX is dependent on pH and is also greatly reduced in the presence of organic matter.[122]

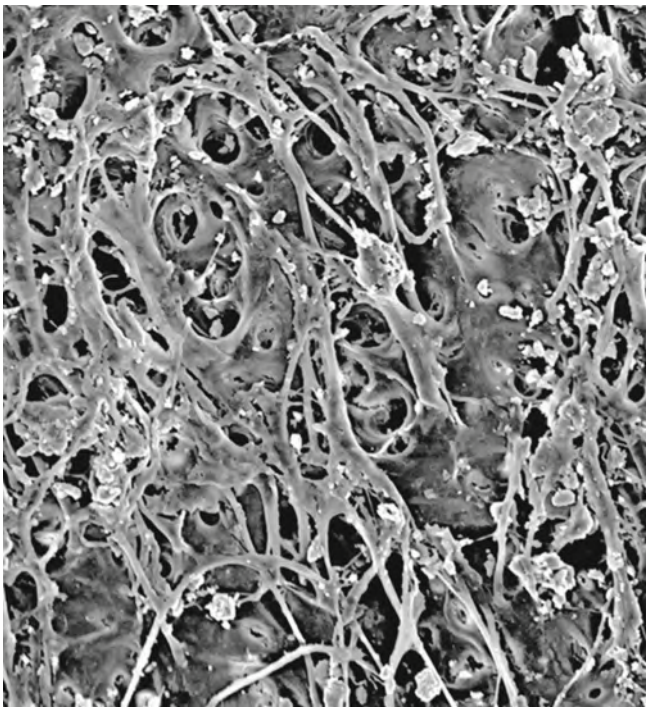

FIGURE 21-11 Chlorhexidine does not have any tissue dissolution activity. Both tissue debris and predentin are left behind in the root canal after irrigating with CHX only.

CHX is effective against both Gram-positive and Gram-negative bacteria as well as yeasts, although the activity against Gram-negative bacteria is not as good as against Gram-positive bacteria.[123–125] Mycobacteria and bacterial spores are resistant to CHX.[120,121] Therefore, CHX is not as suited to chairside sterilization of gutta-percha cones as NaOCl.[126,127] Studies have indicated similar, good performances by CHX and NaOCl in gutta-percha disinfection, but have not used bacterial spores in testing.[128] CHX is not very effective against viruses, its activity is limited to viruses with a lipid envelope.[129] CHX is cytotoxic in direct contact with human cells. A study using fluorescence assay on human periodontal ligament cells showed no difference in cytotoxicity by 0.4% NaOCl and 0.1% CHX.[98] The potential benefits of using CHX in endodontics have been actively researched over the last several years. Several studies have compared the antibacterial effect of NaOCl and CHX against intracanal infection.

While many studies show little or no difference between their antimicrobial effectiveness,[116,130–132] their mode of action may indicate important differences. The effects of 15 minutes of irrigation of experimental biofilms, by mixtures of endodontic bacteria on dentin blocks, have been evaluated by SEM and by culturing.[133] Six percent NaOCl was the only irrigant that completely removed the biofilm, as verified by SEM observations, and killed all bacteria. Two percent CHX was equally effective in bacterial killing, and no growth was detected in any of the samples after CHX treatment. However, an important difference was observed in how the biofilm was structurally affected by irrigation with these two solutions; 6% hypochlorite completely removed the biofilm while the CHX solution had no effect on the biofilm structure.[133] Although the bacteria were killed, the result indicates that as the biofilm remains in the canal after CHX irrigation, it may continue to express its antigenic potential if allowed to communicate with living periapical tissue. Moreover, such residual organic tissue may have a negative impact on the quality of the seal of the permanent root filling.

Some studies have indicated differences in the killing of certain endodontic microbes by hypochlorite and CHX. An *in vitro* study demonstrated differences in the killing of enterococci by CHX and NaOCl. While 5.25% NaOCl killed *E. faecalis* within 30 seconds, lower concentration (4.0% to 0.5%) of hypochlorite required 5 to 30 minutes for complete killing of the bacteria.[84] In the same study, 0.2% to 2% CHX killed the *E. faecalis* cells in 30 seconds or less in all concentrations tested. The result was later supported by two other studies using *E. faecalis* and *Staphylococcus aureus* as test organisms.[83,134] However, the results of these studies have been contradicted by other studies with regard to the effectiveness of NaOCl.[52,85]

Several studies have measured the activity of CHX in gel form against root canal bacteria. Vianna et al.[83] found that CHX in a gel form required a longer time to kill *E. faecalis* than the corresponding concentration in a liquid. Oliveira

et al.[135] reported that 2% CHX gel and 5.25% NaOCl showed excellent activity against *E. faecalis*. When diluted to 1.5% solution, NaOCl reduced the *E. faecalis* counts initially, but the bacterial counts increased during the 7-day follow-up period to the level comparable to the control group.

New studies using biofilms and CLSM with viability staining have to some extent contradicted the results of many of these earlier studies. Stojicic et al.[62] reported that CHX was significantly less effective than 1% and 2 % NaOCl against 3 week old multispecies oral anaerobic biofilm grown on collagen coated hydroxyapatite discs. Studies on dentin canal infection have shown CHX to be equally effective in killing *E. faecalis* inside dentin as one and two per cent NaOCl, but significantly weaker than 6% NaOCl.[57] It is possible that in the "open" biofilm model with hydroxyapatite discs the dynamic of penetration into the biofilm and interaction with local chemistry is different from dentin canal biofilm, where bacteria have been forced into dentin canals by centrifugation.

The antifungal effectiveness of CHX has been shown in several studies.[82,136–138] In a study of the effectiveness of various endodontic disinfecting agents, it was found that combinations of disinfectants were equally or less effective against fungi than the more effective component alone.[82] An interesting, but not yet fully understood synergism has been reported between CHX and H_2O_2.[116] *In vitro* studies using the dentin block model indicated strong synergism between these two agents against *E. faecalis* infection in dentinal tubules. Complementary *in vitro* experiments by Steinberg et al.[117] demonstrated that the combination of CHX and H_2O_2 completely eradicated *E. faecalis* in concentrations clearly lower than required when the compounds were used alone. It is possible that CHX, as a membrane active agent, makes the bacterial membranes more permeable to H_2O_2, allowing easier penetration into the cells and consequently causing damage to the intracellular organelles.[117] Although NaOCl and H_2O_2 are rarely used together in root canal irrigation, such synergistic effect has not been detected between them in the dentin block model.[116] The CHX-H_2O_2 synergism has also been demonstrated in a study of antiplaque mouth rinse.[139] So far there are no published studies of the clinical performance when using combinations of CHX and H_2O_2. Combinations of CHX and carbamide peroxide have been shown to be additive in their cytotoxicity,[140] but corresponding experiments with CHX and H_2O_2 are lacking. Contrary to results of the additive or synergistic action of CHX and H_2O_2, Stojicic et al.[141] reported poor effect of a low concentration of CHX-H_2O_2 combination against several *E. faecalis* strains and mixed plaques, both in planktonic culture and in biofilm. The effect of CHX-H_2O_2 was weaker than that of EDTA-H_2O_2 and CHX–EDTA combinations.

Inhibition of the antimicrobial activity of endodontic irrigants, by substances present in the root canal, has recently been discussed in a number of studies.[52–54] Haapasalo et al.[52] showed in an *in vitro* study that the effect of CHX is reduced or delayed, by the presence of dentin. In following studies by the same group, Portenier et al.[53] detected loss of CHX antimicrobial activity by high concentration (18%, v/w) of bovine serum albumin. In a clinical situation, inflammatory exudate rich in proteins may thus have a negative impact on the effectiveness of CHX. A subsequent study showed that organic dentin matrix and heat-killed microbial cells were effective inhibitors of CHX activity.[54] In a study by Sassone et al.,[142] CHX was incubated together with low concentration (0.5%, v/w) albumin, and no inhibition of the CHX activity could be detected. Albumin concentration in human serum is approximately 2% to 3%, and the total protein concentration is approximately 7%.[143]

Despite some shortcomings, there is evidence that CHX gluconate, as a 2% solution (liquid or gel), may offer an alternative for root canal irrigation. Interestingly, Ng et al.[144] reported in a prospective clinical study that not using CHX in addition to NaOCl had a positive effect on the prognosis of the endodontic treatment. In addition, one should bear in mind that the majority of research on the use of CHX in endodontics is done using *in vitro* and *ex vivo* models and Gram-positive test organisms, mostly *E. faecalis*. The possibility cannot be excluded that the experimental designs give a too positive and biased picture of the usefulness of CHX as an antimicrobial agent in endodontics. More research is needed to identify the optimal irrigation regimen for various types of endodontic treatments. CHX is presently marketed as a water-based solution, as a gel (with Natrosol), and as a liquid mixture with surface-active agents. Future studies of the various CHX preparations will establish the comparative effectiveness of the various CHX combinations *in vivo*. Since CHX lacks the tissue dissolving activity of NaOCl, there have been efforts to simplify the clinical work by combining the two solutions to obtain combined benefits from both. However, CHX and NaOCl are not soluble in each other and a brownish-orange precipitate is formed (Figure 21-12). Although the antimicrobial and other characteristics of the precipitate and the liquid phase have not been thoroughly examined, the precipitate prevents clinical use of the mixture. Marchesan et al.[145] showed that the precipitate was soluble to 0.1 mol/L acetic acid, but the brown/orange color of the solution remained. Atomic absorption spectrophotometry indicated that the precipitate contained iron that may be the reason for the color.

Iodine Potassium Iodide (IPI)

Iodine compounds are among the oldest disinfectants still actively used. They are best known for their use on surfaces, skin, and operation fields. Iodine is less reactive than the chlorine in hypochlorite. However, it kills rapidly and has bactericidal, fungicidal, tuberculocidal, virucidal, and even sporicidal activity.[146] Molecular form, I_2, is the active antimicrobial component.[146] Poor stability of iodine in aqueous solutions motivated the development of iodophors ("iodine carriers"): povidone–iodine and poloxamer–iodine. Iodophors are complexes of iodine and a solubilizing agent that gradually releases the iodine.[146] Iodophors are less active against some yeasts and bacterial spores compared to the

FIGURE 21-12 Orange-brown precipitate forms immediately when CHX and NaOCl are mixed together. The precipitate may contain toxic substances such as para-chloroaniline.

alcoholic iodine solutions (tinctures). Iodine penetrates rapidly into the microorganisms and causes cell death by attacking the proteins, nucleotides, and other key molecules of the cell.[146,147]

Iodine potassium iodide (IPI) has been successfully used in tooth surface disinfection.[114] Potassium iodide is used to dissolve iodine in water, but the antimicrobial activity is carried by the iodine, while potassium iodide has no activity against the microbes.

The effectiveness of 2.5% NaOCl and 10% iodine for disinfection of the operation field (tooth, dental dam, and the clamp) has been compared by bacterial culturing and polymerase chain reaction.[148] The operation field was treated with 30% H_2O_2 and either by 10% iodine or 2.5% NaOCl. No significant difference in the recovery of cultivable bacteria from various sites in either group was detected. However, bacterial DNA was detected significantly more frequently from the tooth surfaces after iodine treatment (45%) than after NaOCl (13%) treatment.[148]

Molander et al.[149] suggested that irrigation with 5% IPI before $Ca(OH)_2$ medication did not have an effect on the overall antimicrobial power. However, it is possible that IPI reduces the frequency of persisting strains of *E. faecalis*. Peciuliene et al.[90] studied the effect of iodine irrigation in 20 teeth with previously root-filled canals and apical periodontitis. The results showed that when used after normal chemomechanical preparation, IPI increased the number of culture negative canals.

In the root canal, iodine compounds come in contact with a variety of substances such as dentin and various proteins. Studies of the interaction of IPI with the chemical environment of the necrotic root canal have shown that dentin can reduce or even abolish the effect of 0.2/0.4% IPI against *E.* faecalis.[52,53] However, pure hydroxyl apatite or bovine serum albumin had little or no effect on the antibacterial activity of IPI. Portenier et al.[54] have shown that dentin matrix (mostly dentin collagen) and heat-killed cells of *E. faecalis* and *C. albicans* inhibit the antibacterial activity of IPI. These studies indicate that inactivation of iodine compounds is one factor explaining the difficulty in obtaining sterile root canals. A study of three minute exposure of *in vitro* multispecies biofilms to different disinfectants demonstrated that 0.6% IPI had equally weak effect on the biofilm as 2% CHX, and weaker than 1% NaOCl.[62]

COMBINATION PRODUCTS FOR CANAL IRRIGATION

In recent years, several new products for root canal irrigation have been introduced that contain more than one active ingredient.[63] The purpose for such development is obviously a desire to make irrigation more effective and also save time. The challenge for combination products is that many substances have interactions that may weaken or even abolish the activity of one or both of the substances. Some interactions may result in formation of toxic by-products. The two most commonly used endodontic irrigants, NaOCl and EDTA cannot be combined without the reduction of activity.[150]

Antibiotic Containing Solutions: MTAD and Tetraclean

MTAD [a mixture of tetracycline isomer, acid, and detergent (Biopure, Dentsply, Tulsa, OK, U.S.A.)] is a combination product for root canal irrigation.[151,152] Tetraclean (Ogna Laboratori Farmaceutici, Muggiò, Italy) is another combination product similar or close to MTAD.[153] MTAD has a low pH (2.15) because it contains citric acid, it removes the smear layer after NaOCl irrigation, and it has antimicrobial activity. The authors who introduced MTAD have recommended the use of 1.3% NaOCl during instrumentation, followed by MTAD to remove the smear layer.[152] However, 1.3% NaOCl may not be strong enough to completely clean the uninstrumented parts of the root canal. Beltz et al.[154] found that MTAD solubilizes dentin, whereas organic pulp tissue is unaffected by it. Zhang et al.[155] showed that MTAD is less cytotoxic than eugenol, 3% H_2O_2, $Ca(OH)_2$ paste, 5.25% NaOCl and EDTA, but more cytotoxic than 2.63% NaOCl.

MTAD contains doxycycline (tetracycline) in high concentration. Shabahang et al.[156] and Shabahang and Torabinejad[157] investigated the effect of MTAD on root canals contaminated with either whole saliva or *E. faecalis* of

extracted human teeth and reported good antibacterial activity. Portenier et al.[47] showed that MTAD killed planktonic *E. faecalis* cells *in vitro* in less than 5 minutes. In an *ex vivo* study, Kho and Baumgartner[158] compared the antimicrobial effectiveness of NaOCl/EDTA and NaOCl/MTAD in extracted roots infected for 4 weeks with *E. faecalis*. After chemomechanical preparation and irrigation, the roots were pulverized in liquid nitrogen and viable bacteria were counted. No difference was measured between the two irrigation regimens. Stojicic et al.[159] reported that QMiX and 1% NaOCl killed all planktonic *E. faecalis* and plaque bacteria in five seconds, while 2% chlorhexidine and MTAD were unable to kill all plaque bacteria in 30s, and some *E. faecalis* cells survived even 3 min of exposure. Using multispecies anaerobically grown biofilms of oral bacteria, the same authors showed that QMiX and 2% NaOCl killed up to 12 times more biofilm bacteria than 1% NaOCl, 2% CHX and MTAD. There are only a few reports so far on the antibacterial effectiveness of Tetraclean, indicating good effect against biofilm.[160,161]

SmearClear, SmearOff, QMiX

SmearClear, SmearOff, QMiX are all EDTA based products for smear layer removal. They also contain other active substances to either alter the surface tension of the product (to facilitate wetting of dentin surface) or to add an antimicrobial effect.[63] The exact composition and concentration of the added substances are not revealed in detail by the manufacturers. One reason to add antimicrobial activity to an EDTA product is to avoid the need to go back to NaOCl after smear layer removal. Studies showed that if the canal is irrigated again by NaOCl, after EDTA or citric acid, this will result in considerable erosion of the canal wall dentin (Figure 21-8).[99] So far no peer review published reports exist on SmearOff.

A few studies have compared the smear layer removal efficacy of SmearClear to 17% EDTA, but there are no studies of its antimicrobial activity.[162,163] QMiX is the most extensively studied of the three. Its efficacy in smear layer removal has been shown to be equal to or better than 17% EDTA,[159,164,165] while dentin microhardness studies have shown that QMiX reduced dentin microhardness less compared to 17% EDTA or 2.25% paracetic acid.[166] Chandrasekhar et al.[167] showed that QMiX was less toxic to the rat subcutaneous tissue than 3% NaOCl, 2% CHX, and 17% EDTA. Alkahtani et al.[168] reported that while both NaOCl and QMiX were toxic to human bone marrow mesenchymal stem cells, QMiX was less cytotoxic than NaOCl. QMiX contains small amount of chlorhexidine that may raise concerns about formation of para-chloroaniline (PCA) if QMiX and NaOCl are intentionally or accidentally mixed in the root canal. However, Kolosowski et al.[169] reported that a precipitate containing PCA was formed in the tubules of dentin irrigated with NaOCl followed by CHX. No precipitates or PCA were detected in the tubules of dentin irrigated with NaOCl followed by saline and QMiX. Arslan et al.[170] mixed NaOCl either with chlorhexidine or QMiX and found that according to nuclear magnetic resonance spectroscopy, para-chloroaniline was present in the mixture of chlorhexidine and NaOCl. However, the mixture of QMiX and NaOCl did not result in para-chloroaniline formation. Other studies have shown better wettability on dentin by resin based sealers such AH+ (Thermaseal) after QMiX than EDTA final rinse, and no effect on dentin binding by Biodentine or MTA.[171,172] In another study, the same author showed that QMiX did not weaken the bonding strength of glass fiber posts cemented with a self-adhesive resin cement to root dentin.[173]

Electrochemically Activated Water

Several new technologies have been introduced during the last few years to improve the effectiveness of root canal disinfection. Increasing attention has been focused on the use of ozone, photoactivated disinfection with low-energy laser, electrochemically activated water, and electric current.[174–178] In a comparative study, 3% NaOCl was more effective than electrochemically activated water in eradicating *E. faecalis* in an *ex vivo* model.[175] Nagayoshi et al.[177] reported relatively equal effectiveness in killing *E. faecalis* with ozonized water and 2.5% NaOCl, when the specimen was irrigated with sonication. However, NaOCl was superior to ozonized water in killing *E. faecalis* in broth culture and in biofilms.[179] Estrela et al.[180] studied root blocks infected for 60 days with a strain of *E. faecalis* and were unable to eradicate all bacteria with any of the methods used, including ozonized water, gaseous ozone, 2.5% NaOCl, and 2% CHX. Many of the new methods offer a biological approach to canal disinfection. However, available evidence so far has failed to show that these methods would be superior or sometimes even equal to existing ones with regard to their antimicrobial effectiveness in the infected root canal.

IRRIGATION OF THE APICAL ROOT CANAL

Despite the great technological advances that have been made in instrumentation and irrigation formulations, decades of research has repeatedly demonstrated the variable success with which we are able to deliver irrigant to the apical root canal (Figure 21-13). Studies have also demonstrated the great extent to which root dentin remains uninstrumented despite the use of endodontic files, especially in the apical 4 mm of a root canal.[181] Uninstrumented recesses in oval canals of mandibular incisors can be present in up to 65% of teeth after balanced force instrumentation of canals,[182] while 65.2–74.4% of the apical 4 mm of the distal roots of mandibular molars can remain untouched by instruments after mechanical debridement has been completed by rotary files.[181] The shortcoming of current rotary and hand instrumentation lies in the fact that they cannot adapt to the anatomical complexity of a root canal system. Studies

FIGURE 21-13 Scanning electron microscope image of apical root canal after finished instrumentation and copious irrigation with NaOCl and EDTA. Dentin debris that was created during instrumentation and packed into the most apical canals was not dissolved or removed by irrigation.

on canal morphology have shown that root canals are rarely conical or straight, and have lateral canals, apical deltas, fins, webs, and transverse anastomoses,[183] that harbor intraradicular biofilm in the apical third in those teeth diagnosed with apical periodontitis.[184] Computational fluid dynamic (CFD) modelling has demonstrated at least the partial irrigation of a simulated lateral canal under ideal and varied placement of a conventional irrigation needle in a root canal model.[185] Thus, it becomes even more important for intracanal irrigants to be delivered to where rotary instruments are unable to perform their function, and the role of root canal instrumentation shifts out of necessity from that of debridement, demonstrated to be variably successful, to one of creating apical radicular access for irrigants.[186]

Fluid Dynamics and Factors Affecting Irrigation Efficacy in Apical Root Canal

Contemporary studies of irrigation have closely examined variables associated with irrigation efficiency, but unlike the previous decades, these studies are utilizing novel experimental models. Hsieh et al. used thermal image analysis to record fluid distribution during irrigation in extracted teeth.[187] They found that a size 27 gauge needle should be placed 3 mm from the apical stop in order to achieve successful irrigation when the canal was prepared to ISO size 30 (0.3 mm). When the needle tip was placed 6 mm from the apical stop, successful irrigation was seen only when the root canal was prepared to at least ISO size 50 (0.5 mm), demonstrating that irrigation was improved by apical placement of the needle tip and larger size of canal preparation. Another study, using real-time imaging of bioluminescent bacteria inoculated into a root canal, confirmed that irrigation is affected by the depth of placement of the needle tip. Emission of bioluminescence was imaged before and after irrigation, showing significant reduction of bacteria when the needle was placed at 1 mm from the working length compared to 5 mm from the working length.[188] The same technique was used to show that increased canal curvature negatively affected irrigation effectiveness.[189] A study by Huang et al.[190] ranked the importance of different factors influencing the removal of a stained collagen bio-molecular film in extracted teeth. The collagen bio-molecular film thickness approximated the thickness of bacterial biofilm. The percentage of canal surface covered with stained collagen was quantified before and after irrigation. The authors found that, in order of decreasing priority, the depth of placement of the needle tip, the apical size and taper of the canal preparation, and the use of dynamic or static irrigation techniques were factors influencing the removal of a collagen film in a root canal.[190] Real-time fluid movement analysis using microparticle image velocimetry in soft lithography-based models have also verified that dynamic irrigation techniques, such as continuous ultrasonic-assisted irrigation, are correlated with significant biofilm reduction and effective fluid flow effects in the apical third in comparison to conventional syringe irrigation.[191]

Irrigant clearance beyond the needle tip (Figure 21-14) was examined in a study by Park et al.[66] that prepared a plastic root canal model and filled the canal with dye. The dye was irrigated from the canal using various needle designs, with the needle held at 3 mm from the working length. The irrigant was delivered at precise flow rates from 1 to 15 mL/min, and the length of dye cleared from the end of the needle tip measured. There was a flow rate dependent increase in the length of dye cleared from the end of the needle tip starting at 1 mL/min, but this length of dye clearance plateaued at irrigation flow rates above 4 mL/min (Figure 21-15), regardless of the needle tip design and needle size.[66] The length of dye clearance beyond the needle tip at irrigation flow rates above 4 mL/min was between 2 to 3 mm, resulting in a similar range of dye clearance regardless of needle design. CFD and analytical modelling have confirmed the existence of a stagnation plane beyond which irrigant will not penetrate from the end of the needle tip (Figure 21-16).[192] One CFD study identified the limit of irrigant replacement beyond the needle tip as 1 to 1.5 mm, regardless of the flow rate used, that included very high flow rates of 15 mL/min.[67] Increased root canal taper and larger apical preparation sizes were shown to improve irrigant replacement through CFD analysis.[193,194] When the depth of needle placement was investigated by CFD analysis, Boutsioukis et al.[195] found

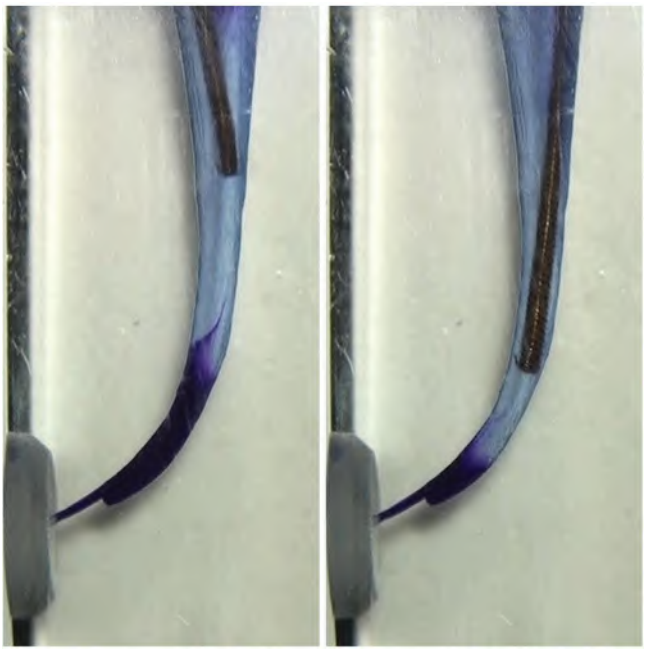

FIGURE 21-14 Irrigant clearance is the length of the zone beyond the needle tip where irrigant penetrates during irrigation. Despite occasional claims to the contrary, irrigants do penetrate longer than the position of the needle tip when side vented ("safety tip") or open-ended needles are used. However, clearance zone is typically limited to 1 mm to 3 mm, and it does not grow anymore longer above flow rates of ca. 5 mL/min.

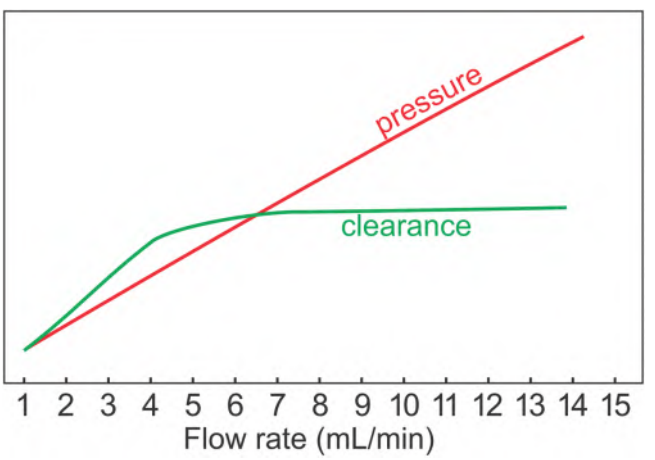

FIGURE 21-15 A study on irrigant flow rate, apical clearance zone, and irrigation pressure towards the apex showed that clearance beyond the needle tip increased when flow rate increased to 5 mL/min. The depth of the clearance zone did not continue to grow at higher flow rates. However, the pressure caused by the irrigation continued to grow in a linear fashion with increasing flow rate.[66]

that a 30 gauge closed-end side-vented needle achieved 1 to 1.5 mm of irrigant replacement apical to the end of the needle regardless of its position in the canal. Hence, it is highly likely that maximum exchange of irrigant without the need for very high irrigation flow rates could be achieved.

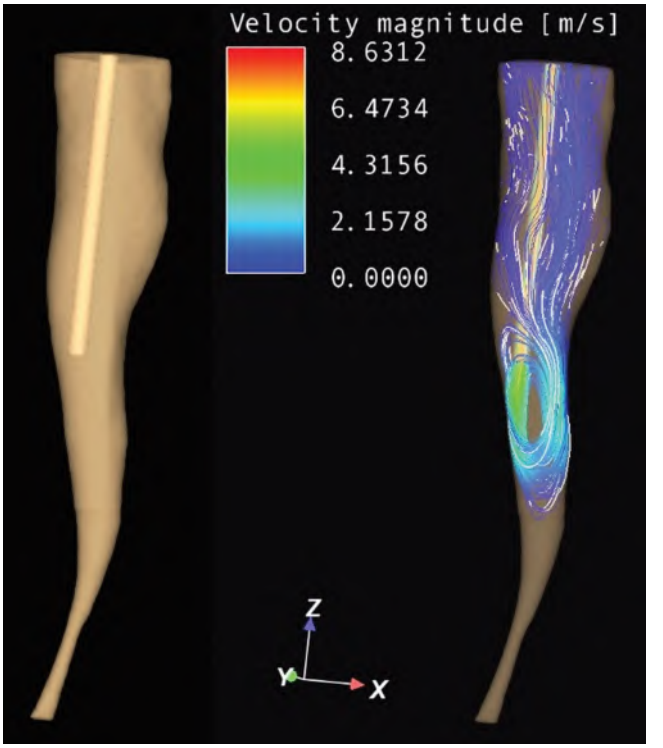

FIGURE 21-16 A computational fluid dynamics model of irrigant flow in a canine root canal virtual model, based on micro-CT scanning of a real canine tooth. The complex mathematical model predicts the extent of the flow beyond the needle tip, flow speed in various parts of the canal and canal wall shear stress caused by the irrigant.

Apical Pressure Measurement; Positive Vs. Negative Pressure

With the recommendations that irrigation needles should be placed approximately 1 mm from the working length or at least in the apical third of the root canal in order to achieve effective irrigation, whether positive pressure or negative pressure methods are used, it is of interest to know what kind of apical pressures are generated when specific irrigation parameters such as different flow rates are used. The available CFD studies are difficult to compare as different simulated irrigation flow rates, a range of simulated needle designs, and different canal preparation sizes were used. Furthermore, the software algorithms used are potentially different. When only similar needles are compared, the apical pressure generated by a beveled needle tip at 3 mm from the working length is reported to be approximately 18 kPa (135 mmHg) by one group using a flow rate of 15.6 mL/min,[196] but only 1.707 kPa (13 mmHg) by another group using an irrigation flow rate of 6 mL/min.[68] If one compares a side-vented closed-end needle design, the apical pressure generated at 3 mm from the working length is reported to be approximately 10 kPa (75 mmHg) by one group using a flow rate of 15.6 mL/min,[196] and 0.256 kPa (2 mmHg) by another group using a flow rate of 6 mL/min.[68] These pressures differ by several orders of

magnitude. The rotational orientation of a side-vented needle has also been shown to influence irrigant flow pattern and apical pressure in a root canal.[185]

It is not possible to compare the reported apical pressures to a reference point at the current time, and it is also unknown what pressures might result in a sodium hypochlorite extrusion accident, but it is interesting to consider that the average capillary pressure in the human body is 25 mmHg, or 3.3 kPa.[197] Khan et al.[70] suggested that irrigation pressure of as low as 5.88 mmHg (human central venous pressure) may be an upper limit for safe irrigation. Park et al.[66] measured the pressure generated during positive and negative pressure irrigation at the periapex of an *in vitro* tooth model using a novel method of measurement, investigating the effect of flow rate and needle design. A mesiobuccal canal of a mandibular molar was instrumented and placed into a chamber coupled to a pressure transducer. Irrigation was performed via a digital peristaltic pump using flow rates from 1 to 15 mL/min with irrigation needles of different size and design. Positive pressure irrigation revealed a flow rate dependent increase in apical pressure (Figure 21-15). The apical pressure at high irrigation flow rates was several times higher than at low flow rates. Needle designs with closed ends and/or side vents yielded statistically significant lower apical pressures than blunt open-ended needles. In contrast, measurement of apical pressure during apical negative pressure irrigation revealed that no positive pressures were generated, even when any of the needles used were placed at 1 mm from the working length, thus confirming the safety benefit of this irrigation method for patients.[66] Coupling this finding with the evaluation of dye clearance beyond the needle tip during negative pressure irrigation, using a blunt open-ended needle tip, should prove to be effective and safe.

Negative pressure irrigation is reported to address the limitations associated with positive pressure irrigation, such as the difficulty in delivering and replenishing irrigant to the apical third, and also the safety issues that might occur when a needle is placed close to the root apex and apical foramen in attempting effective delivery and replenishment. The ability of the EndoVac irrigation system to remove significantly more debris than conventional syringe irrigation has been reported in several studies. Some studies report no difference in debris removal at the 3-mm level but a significant difference at the 1-mm level.[198,199] A different study reported significantly less debris after use of the EndoVac compared to conventional syringe irrigation at 1.5 mm and 3.5 mm from the apex.[200] The antimicrobial efficacy of irrigation with the EndoVac system in comparison to syringe irrigation and supplementary sonic agitation techniques has shown that these techniques are comparable and highly effective in bacterial reduction.[201,202] Apical negative pressure is at least comparable to that of conventional irrigation in its ability to obtain culture-negative root canals in necrotic teeth.[203] One study also reports the superior antimicrobial efficacy of the EndoVac system,[204] but these differences can be attributed to differences in bacterial sampling after irrigation.

The major benefit of apical negative pressure for irrigation is the prevention of irrigant extrusion apically, even when placed very close to the root apex. This has been verified in several studies and tested in several different ways. One such study collected the volume of irrigant extruded during irrigation from the root end of teeth and reported that the EndoVac microcannula and macrocannula did not extrude any irrigant at all even when placed at full working length, in comparison to other techniques such as manual or ultrasonic activation.[205] As an extension of the ability of apical negative pressure irrigation to prevent extrusion of irrigant yet deliver irrigant to the full working length, one group has reported that the incidence of post-operative pain in patients where the EndoVac was used was significantly less than in those patients where positive pressure syringe irrigation was used.[206]

PHYSICAL MEANS OF FACILITATING CANAL CLEANING AND DISINFECTION

Syringe-needle Irrigation

Conventional positive pressure irrigation using a syringe and needle is the most common way of irrigating root canals.[63] Small syringes of 1 to 5 mL offer easier handling and better tactile control of irrigant flow than large syringes of 10 mL and higher. Luer lock attachment of the needle to the syringe should be regarded as mandatory in order to avoid unintended accidents. While there is no clear consensus of the optimal needle design, it seems that the preferred needle type and size is a 30-gauge side vented needle with closed end (Figure 21-17). Needles of this type, by various manufacturers, usually have a rounded tip that facilitates smooth gliding of the needle in the canal despite mild curves and canal wall irregularities. In severely curved canals needles with a non-metallic tip allow better access to the apical canal (Figure 21-18). The Endovac system (Kerr Co., Orange, CA, U.S.A.) constantly irrigates the canal system and evacuates debris (Figure 21-19)

It is important to understand that syringe-needle irrigation does not guarantee flow of the irrigant to all areas of the root canal system. In fact, careless irrigation may leave large areas of the canal(s) un-irrigated.

Sonic Devices

Sonic devices transmit mechanical energy through vibrations. The best known sonic device in endodontics probably is the EndoActivator, that has thin, plastic tips of different sizes attached to a special hand piece.[205,207–210] The pulp chamber and the canal are filled with the irrigant and the tip is placed close to working length to "activate" the solution. "Activation" in fact means that the solution is mechanically agitated with

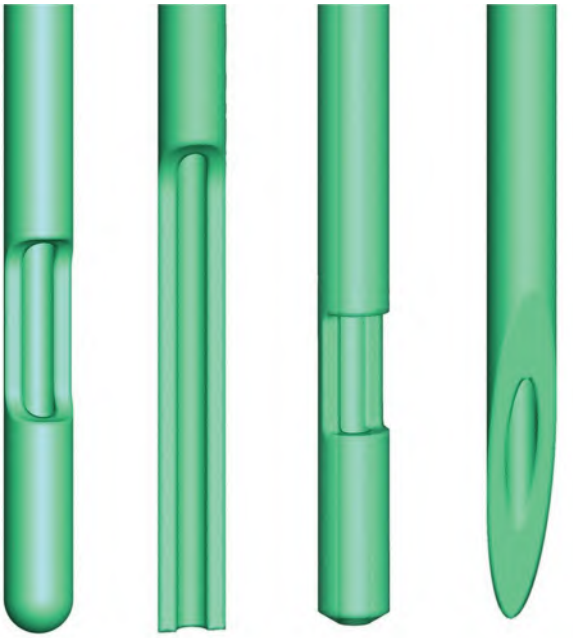

FIGURE 21-17 Four different needle designs, produced by computerized mesh models based on true and virtual needles. From left to right; side vented ("safety tip"), notched, side vented open ended, beveled.

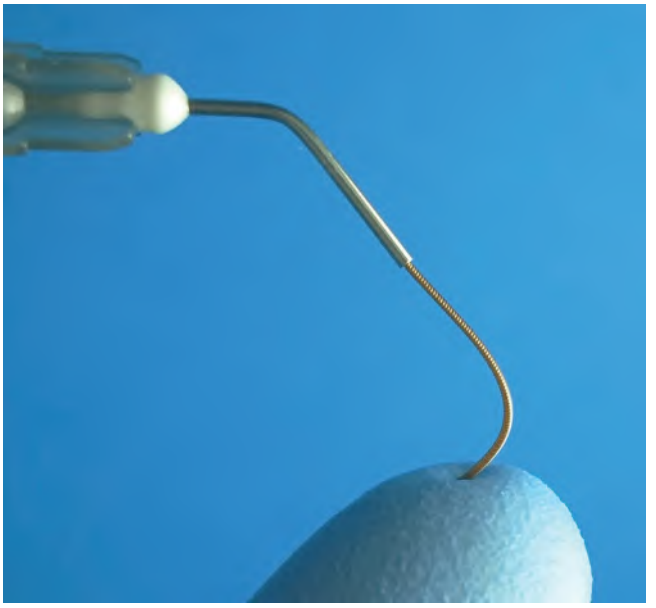

FIGURE 21-18 A Flexi-Glide needle has non-metallic flexible tip that can reach the apex in severely curved canals.

the vibrating tip. The 3-speed sonic motor provides options of two, 6000 and 10,000 vibrations per minute. As the plastic tip is softer than dentin, it does not create a smear layer. A number of studies comparing different irrigation methods have demonstrated some improvement in cleaning of the canal space by EndoActivator, while other studies have not reported such differences.[205,207–210] In short, EndoActivator is an easy-to-use, safe facilitator of canal irrigation.

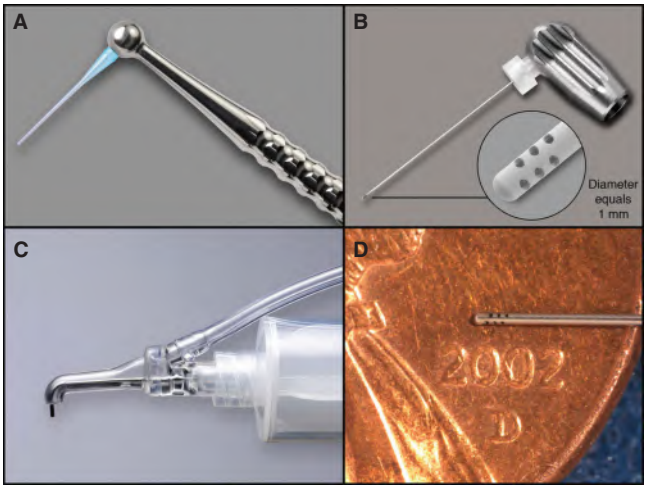

FIGURE 21-19 EndoVac system. ***A***. Macrocanula constantly evacuates debris and irrigant during canal preparation to size #35/.04. ***B***. Microcanula .32 mm diameter provides constant irrigation to full working length. ***C***. Master Delivery Tip constantly supplies irrigant to pulp chamber reservoir. ***D***. Micro relative size vs. U.S.A penny. (Courtesy of Dr. John Schoeffel, Dana Point, California.)

Ultrasonic Cleaning

Ultrasound is used in a variety of tasks including finishing access cavity preparation, removing pulp stones, locating canal orifices, opening calcified canals, fractured instrument removal, placing cements and root filling materials, and retro canal preparation during surgical procedures.[211] The use of ultrasonic energy for cleaning of the root canal and to facilitate disinfection has a long history in endodontics. The comparative effectiveness of ultrasonics and hand instrumentation techniques has been evaluated in a number of earlier studies.[211–215] The majority of these studies concluded that the ultrasonics, together with an irrigant, contributed to a better cleaning of the root canal system than irrigation and hand instrumentation alone. Cavitation and acoustic streaming of the irrigant contribute to the biological chemical activity for maximum effectiveness.[216] Analysis of the physical mechanisms of the hydrodynamic response of an oscillating ultrasonic file suggested that stable and transient cavitation of a file, steady streaming, and cavitation microstreaming all contribute to the cleaning of the root canal.[217] However, it has also been suggested that cavitation using traditional ultrasonic equipment with files may not extend further than a few micrometers from the file.

Several different methods and criteria have been used to study the effect of ultrasound on the cleanliness of the canal. These include bacteriological, histological, and microscopic techniques.[218–222] Studies focusing on the ability of ultrasound to remove the smear layer have shown contradictory results. This is not, in fact, surprising because it is generally known that the smear layer can be removed primarily by chemical means only, or by appropriate laser treatment.[223,224] It has also been shown that to work effectively, ultrasonic files must be free in the canal without contact with the canal walls.[225]

Among the areas that are particularly difficult to clean are anastomoses between double canals, isthmuses, and fins (Figure 21-20). Several studies have indicated the importance of ultrasonic preparation for optimal debridement of the root canals and isthmuses.[224,226–228] Ultrasonics also eliminated bacteria from canals more effectively than hand instrumentation.[228–230] However, not all studies have supported this finding.[231] It has been proposed that canal anatomy is more important than ultrasound for effectiveness of the cleaning procedures.[232]

The direct bactericidal effect by ultrasonic energy seems to be very limited at best.[233,234] Ultrasonics seems to exert its antimicrobial effect when used together with irrigants, perhaps via the physical mechanisms of cavitation and acoustic streaming. It is also possible that ultrasonics helps irrigants penetrate into areas, in complex canal systems, not easily reached by normal irrigation.

In a series of studies on the effect of canal shape (taper) and instrument design, Lee et al.[235] demonstrated in simulated plastic root canals that the diameter and taper of root canal influenced the effectiveness of ultrasonic irrigation to remove artificially placed dentin debris. After ultrasonic irrigation, the amount of debris in the size #20/.04 taper group was significantly higher than that for the size #20/.06 group and the size #20/.08 group. Van der Sluis et al.,[236] using roots from human teeth *ex vivo*, reported that there was a tendency for ultrasonic irrigation to be more effective in removing artificially placed dentin debris from simulated canal extensions of canals with greater tapers.

A study using a split tooth technique found that ultrasonic irrigation *ex vivo* was more effective than syringe irrigation in removing artificially created dentin debris placed in simulated uninstrumented extensions and irregularities in straight, wide root canals.[235] In another study, passive ultrasonic irrigation with 2% NaOCl was more effective in removing $Ca(OH)_2$ paste from artificial root canal grooves than syringe delivery of 2% NaOCl or water as an irrigant.[237]

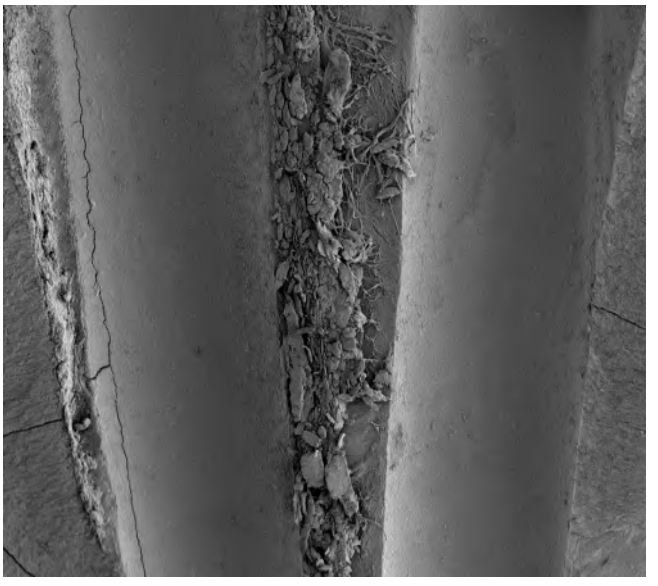

FIGURE 21-20 Scanning electron microscope image of two joining canals with a narrow isthmus between the canals. Instrumentation has packed dentin debris and organic matter (fibers) into the isthmus. Prolonged syringe-needle irrigation of over 10 minutes was not able to remove the debris.

Ultrasonic instrumentation can also have an impact on canal dimensions and in some cases can cause unwanted complications such as straightening of the canal (transportation), perforations, and extrusion of infectious material beyond the apex.[225,238–243] A histobacteriological study of teeth with non-vital pulps showed compacted debris and bacteria in the apical region and in the dentinal tubules after ultrasonic instrumentation.[242] In one study, the step preparation technique was shown to cause more extrusion than the standard technique, whereas the least extrusion was detected with the crown down and ultrasound techniques.[241] Interestingly, Van der Sluis et al.[236] suggested that a smooth wire during ultrasonic irrigation is as effective as a size 15 K-file in the removal of artificially placed dentin debris from grooves in simulated root canals in resin blocks. It is possible that using an ultrasonic tip with a smooth, inactive surface, means preparation complications are less likely to occur. Studies using micro-CT have examined the effectiveness of ultrasonic activation to remove dentin debris from fins and isthmuses where it was packed with rotary files.[244] The results showed that only some but not all of the debris could be removed. In line of this result, other studies about removal of interappointment calcium hydroxide paste from molar root canals have shown that sonic or ultrasonic energy together with copious irrigation failed to remove all residual calcium hydroxide from the canals.[245–248]

Ultrasonic irrigation has traditionally been done as passive ultrasonic irrigation, "PUI." The pulp chamber and the root canals have been filled with the irrigant, followed by the introduction of the ultrasonic tip into the canal and setting on the energy. One of the new models for ultrasonic irrigation, EndoUltra (Figure 21-21) (Vista Dental Products, Racine, WI, U.S.A.) has a high frequency of 40 kHz, instead of the common 20 to 30.000 kHz. Some of the modern ultrasonic devices combine continuous flow of the irrigant into canal with continuous use of ultrasound. These devices have hollow needle-like tips that allow flow of the irrigant into the pulp chamber or into coronal/middle root canal while vibrating at the same time by ultrasonic energy.[249–252] ProUltra PiezoFlow (Dentsply Tulsa Dental Specialties, OK, U.S.A) (Figure 21-22) irrigating system is one of the

FIGURE 21-21 EndoUltra high frequency (40-kHz) unit for passive ultrasonic irrigation.

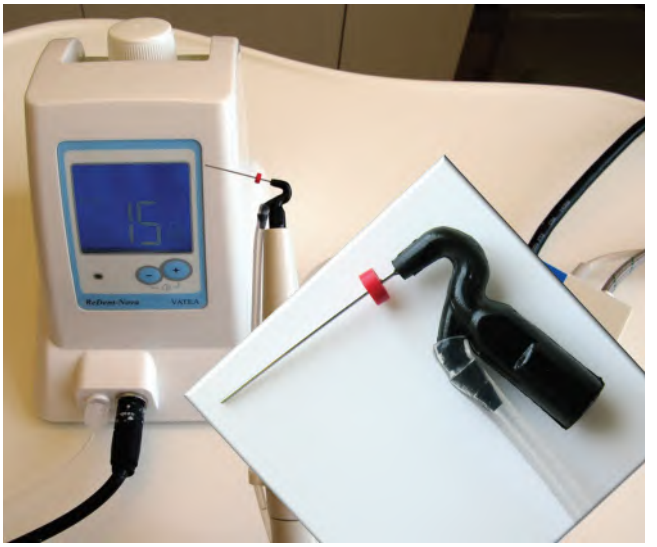

FIGURE 21-22 A ProUltra PiezoFlow active ultrasonic system for irrigation of root canals. The irrigant flows through the hollow needle into the coronal/mid root canal while the needle is activated by ultrasonic energy.

commercial devices based on studies of its prototype at the University of Ohio. These studies showed good/excellent results in cleaning the usually hard-to-reach areas such as fins and isthmuses.[249–252]

Lasers

The potential of different endodontic lasers in eradicating root canal microbes has been a focus of interest for many years. Early comparative studies indicated, however, that the antibacterial effectiveness of lasers in the root canal was inferior to NaOCl irrigation.[253–255] Excellent antibacterial efficiency against *E. faecalis* was reported by Gutknecht et al.[256] using a holmium:yttrium–aluminum–garnet (Ho:YAG) laser on root canals infected with this species *in vitro*, 99.98% of the bacteria were eliminated. However, Le Goff et al.[257] obtained only an 85% decrease in the bacterial counts by a CO_2 laser, clearly less compared to irrigation with 3% NaOCl. Contrary to the main stream of results with laser treatment, Kesler et al.[258] indicated that complete sterility of the root canal can be obtained with a CO_2 laser microprobe coupled onto a special hand piece attached to the delivery fiber. Schoop et al.[259] in subsequent studies indicated that the effect of laser is dependent on the applied output power and specific for different bacteria. Complete sterility still seems to remain a challenge with laser treatment.[260,261] Another important aspect of laser radiation in endodontics is the effect of lasers on the smear layer, that may also facilitate effective disinfection of the canal. Recently, a new technology using laser-activated irrigation was introduced. Photon Induced Photoacoustic Streaming (PIPS™) uses an Erbium 2,940 laser to pulse extremely low energy levels of light to generate a photoacoustic shockwave throughout the entire root canal system without the need to enlarge the canals (Figure 21-23).[262,263] Using extremely short bursts of laser energy with high peak powers, a specially designed tapered and stripped tip creates a pressure wave that streams irrigants throughout the root canal system. Unlike other conventional hand and ultrasonic systems, the PIPS tip is placed in the pulp chamber only and not in the canals. The resultant effect is a passive reflux of tissue debris that exits coronally up and out of the roots while cleaning the canal system.

Mathew et al.[33] compared antimicrobial effect by irrigation using three different devices. They reported that the diode laser group showed better antibacterial efficacy and least viable bacteria when compared to conventional needle irrigation, PIPS and EndoActivator groups in minimally instrumented, experimentally instrumented

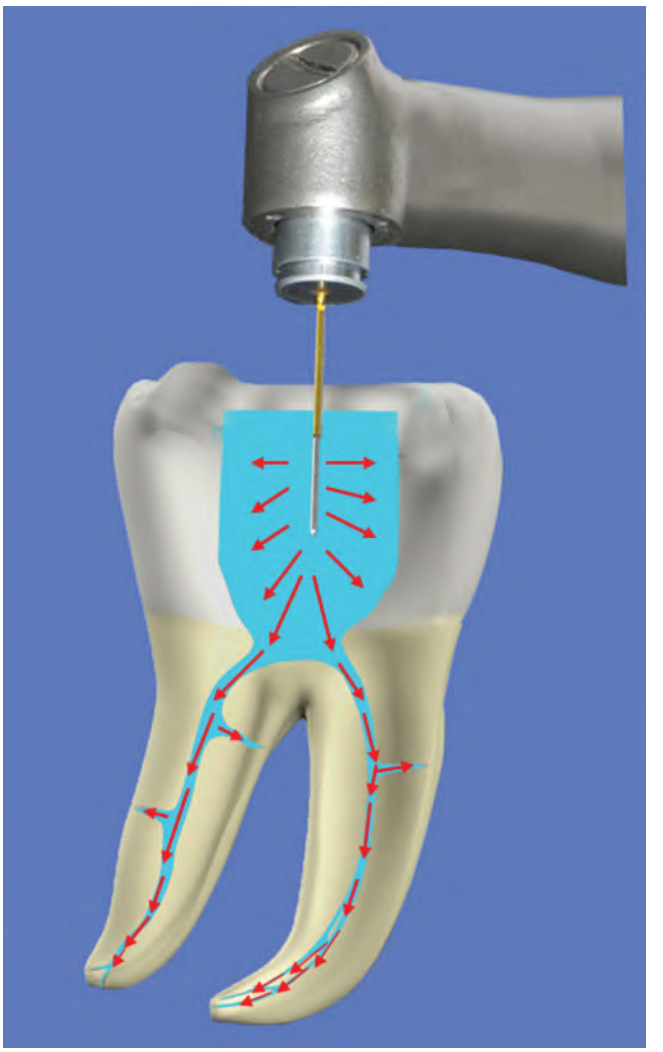

FIGURE 21-23 Photon Induced Photoacoustic Streaming (PIPS™) uses an Erbium 2,940 laser to pulse extremely low energy levels of light to generate a photoacoustic shockwave throughout the entire root canal system. (Courtesy of Dr. Enrico DiVito, Scottsdale, AZ.)

root canals. PIPS and EndoActivator were more effective than conventional needle irrigation.[33] Pedullà et al.[262] and Zhu et al.[264] reported no difference in the antibacterial effect on root canal microbes in an *ex vivo* study that compared PIPS to NaOCl only. However, Al Shahrani et al.[265] showed better effect by PIPS + 6% NaOCl against *E. faecalis* biofilm in root canal compared to 6% NaOCl alone.

Arslan et al.[209] found that PIPS was superior in removing $Ca(OH)_2$ compared to needle irrigation, sonic irrigation, and ultrasonic irrigation from artificial grooves in straight root canals. Olivi et al.[266] reported that PIPS appears to be effective in enhancing the effect of the irrigants commonly used in endodontics. Lloyd et al.[267] examined the removal of dentin debris by irrigation using either standard needle irrigation or PIPS. The authors found that irrigation using PIPS increased the canal volume and eliminated debris from the canal system 2.6 times more than needle irrigation. Tissue dissolution effectiveness were compared by Guneser et al.,[268] who reported that 5.25% NaOCl with Er:YAG laser dissolved ca. 50% more pulp tissue than same concentration NaOCl alone, while NaOCl with PIPS dissolved 20% more pulp tissue than NaOCl alone.

Wide Spectrum Sound Energy for Cleaning of Root Canals

A new device using sound energy far beyond the ultrasound range, GentleWave (Sonendo, Orange, CA, U.S.A.), was recently tested for its ability to dissolve soft tissue, and compared it to other forms of irrigation, including ultrasound.[34] The device consists of a central unit from which high pressure pumps send high speed degassed irrigant flow to a special hand piece (Figure 21-24), with a tip of the handpiece placed into the pulp chamber of the tooth. The irrigant stream hits the tip of the nozzle and spreads as a cloud to the root canal system (Figure 21-25). According to the manufacturer the effect is based on wide spectrum sound energy, cavitation, and chemical reactions. The irrigant is sucked back through several small holes in the hand piece covering the access cavity, and creating a stable negative pressure throughout the root canal system. The study of tissue dissolution showed that even using water, organic tissue was dissolved as well as using 2% NaOCl and ultrasonic irrigation.[34] This may indicate the presence of cavitation, as water is not expected to dissolve soft tissue. Overall, the study showed that soft tissue dissolution was up to 8 times faster than with conventional irrigation with 6% NaOCl and ultrasound. Another study showed that GentleWave was the only system that completely removed calcium hydroxide from molar root canals, whereas syringe-needle irrigation with and without ultrasound always left some calcium hydroxide in the canal space, particularly in the apical third of the root canals.[269]

FIGURE 21-24 GentleWave handpiece delivers the "high speed" irrigant into the pulp chamber, from where it spreads into the root canal system.

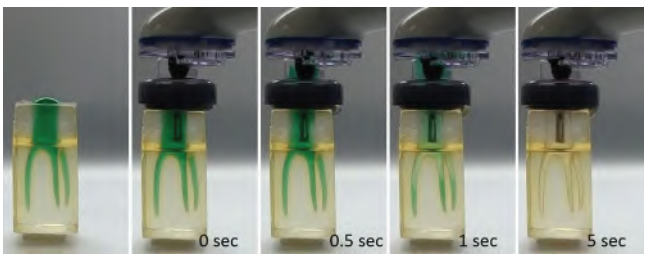

FIGURE 21-25 A demonstration of the exchange of colored liquid by the GentleWave system in root canals of a molar. Irrigant in all canals has been refreshed in a few seconds simultaneously. The tooth model is based on micro-CT imaging of a real tooth.

INTRACANAL INTERAPPOINTMENT MEDICAMENTS

In the treatment of teeth with a vital pulp that is inflamed but not necrotic, there is no need for intracanal medication for microbiological reasons. However, if time does not allow completion of the treatment in one appointment, it is generally recommended that the root canal should be filled between appointments with an antimicrobial dressing, for example, $Ca(OH)_2$, to maintain sterility in the canal space until a permanent root filling is placed. However, there are no studies comparing the bacteriological status of root canals following pulpectomy, when the canals have been left empty or filled with an antibacterial dressing.

The question of the role of intracanal medicaments becomes more relevant, and complex, in the treatment of pulpal necrosis and apical periodontitis. There is overwhelming evidence in the literature that many, if not most, root canals contain viable microorganisms after the completion of the chemomechanical preparation at the end of the first appointment.[12–14,27,86,90,270–273] Therefore, a variety of intracanal medicaments have been used between appointments to complete disinfection of the root canal. In addition to killing microorganisms, intracanal medicaments may have other beneficial functions. $Ca(OH)_2$ neutralizes the biological activity of bacterial lipopolysaccharide and makes necrotic tissue more susceptible to the solubilizing action of

NaOCl at the next appointment.[274,275] Another aspect in using intracanal medicaments may be that a more thorough cleanliness is achieved because of the longer overall time used for the treatment. On the other hand, several appointments can also increase the risk for aseptic complications, for instance, through a leaking temporary filling and poor patient compliance.[276]

Several studies have indicated a poorer prognosis of the treatment of apical periodontitis if viable bacteria are residing in the root canal system at the time of filling.[9–11] Other studies, however, have contradicted these results and reported no significant differences in healing between teeth filled after positive or negative cultures from the root canal,[12] or between treatments performed in one or two appointments.[12,13] It has also been suggested that "intracanal sampling techniques suffer from deficiencies that limit their predictive value."[277] A permanent root filling of high quality, using endodontic cements with antimicrobial activity, can effectively seal and entomb residual microorganisms in the canal, and prevent communication with periradicular tissues. Continued killing of the microorganisms could take place due to the antimicrobial activity of the root-filling materials[74,278] and unavailability of nutrients. However, some antimicrobial root canal filling materials may also be cytotoxic. Systematic reviews of single-visit vs. multiple visit root canal treatment have not found much difference in healing between the two strategies.[279,280]

Calcium Hydroxide [$Ca(OH)_2$]

Calcium hydroxide [$Ca(OH)_2$] has a special historical position in endodontics. Indications for the use of $Ca(OH)_2$ in the prevention and treatment of various pulpal and periapical conditions have been numerous. In addition to endodontic infections, use of $Ca(OH)_2$ has been widely advocated in dental traumatology and in the treatment of resorptions. The classical studies in the 1970s and 1980s at the University of Umeå, Sweden, were a strong stimulus for the wide spread use of $Ca(OH)_2$ as a local disinfecting medicament in the root canal for the treatment of apical periodontitis. Byström et al.[10] reported that $Ca(OH)_2$ was an effective intracanal medicament, rendering 34 out of 35 canals bacteria free after a 4-week period. The effectiveness of interappointment $Ca(OH)_2$ was also reported by Sjögren et al.,[281] who demonstrated that a 7-day dressing with $Ca(OH)_2$ eliminated all bacteria in the root canal. However, these pioneering studies have been challenged by others who reported a residual flora from 7% to 35% of teeth after one or more weeks with $Ca(OH)_2$ in the canal.[28,282–284] Kvist et al.[285] reported the antimicrobial efficacy of endodontic procedures performed in a single visit, with 10-minute iodine irrigation, compared to a two-visit procedure, with an interappointment dressing with a $Ca(OH)_2$ paste. Residual microorganisms were detected in 29% of the one-visit teeth and in 36% of the two-visit-treated teeth, with no statistically significant differences between the groups. A systematic review by Sathorn et al.[14] concluded that calcium hydroxide has minimal effect in reducing root canal microbes.

Zerella et al.[58] compared the antibacterial activity of $Ca(OH)_2$ mixed either with water or with 2% CHX in vivo. Pure $Ca(OH)_2$ completely disinfected 12 out of 20 teeth, while the $Ca(OH)_2$-CHX paste disinfected 16 of 20 teeth. The difference, however, was not statistically significant because of small sample size. Siqueira et al.[286] examined bacterial reduction in teeth with apical periodontitis, after instrumentation and irrigation with 0.12% CHX solution and after 7 days of intracanal medicament with a $Ca(OH)_2$-CHX (0.12%) mixture. After finishing the chemomechanical preparation, 7 of the 13 cases still showed growth, while after the $Ca(OH)_2$-CHX treatment only one of 13 teeth was culture positive.

Vivacqua-Gomes et al.[55] examined the benefit of interappointment $Ca(OH)_2$ medicament in root canals in an *ex vivo* model. The premolar teeth were infected with *E. faecalis* for 60 days, and the canals were instrumented using rotary instruments. Irrigation, interappointment medication, and root filling were performed following five different protocols, either in a single visit or in multiple visits. A second bacteriological sample was obtained 60 days after the root filling, where the root fillings were removed and samples taken. Bacteria were found in 3 of 15 teeth (20%) irrigated with 2% CHX gel and filled in single visit, and in 4 of 15 teeth (25%) irrigated with CHX and filled with $Ca(OH)_2$ for 14 days before the gutta-percha/sealer root filling was placed. Teeth that were left empty for one week after irrigation and before root filling, or irrigated with saline only instead of CHX, or filled without sealer showed bacteria in 40 to 100% of the teeth 60 days after the root filling was placed. Because of the small size of the experimental groups, far-reaching conclusions cannot be made. However, the results support the finding of other studies indicating that interappointment $Ca(OH)_2$ may not add to the antibacterial effectiveness of the treatment. Moreover, the results emphasize the importance of not leaving the root canal empty (no medicament, no root filling) as well the role of sealer in the joint effort to combat infection.

$Ca(OH)_2$ was earlier the material of choice in treating young teeth with open apex and large canal space. Apexogenesis and apexification were the long term goals, with up to one to two years of $Ca(OH)_2$ treatment.[287,288] However, reports of the weakening of root dentin because of longstanding $Ca(OH)_2$ in the canal[289,290] and the introduction of MTA and other bioceramic material, such as Biodentine and Root Repair materials, have led to a clear change in the treatment strategy of young teeth with open apex.[290,291] However, a recent study of the long-term effects of three calcium hydroxide preparations did not detect any weakening of the root dentin as compared to control group after six months of calcium hydroxide treatment.[292] For more details, see Chapter 28.

Several studies in recent years have focused on a familiar clinical challenge; the difficulty in removing $Ca(OH)_2$ from the root canal before root filling.[247,293,294] Syringe-needle irrigation, sonic activation, and even ultrasound have all been shown to be ineffective in removing all $Ca(OH)_2$

from the canals. However, a laser based system, PIPS, has shown to remove more $Ca(OH)_2$, than irrigation with ultrasound from artificial grooves in straight canals,[209] and the wide spectrum sound energy based GentleWave was reported to completely remove $Ca(OH)_2$ from molar root canals (Figure 21-26).[269]

Chlorhexidine Digluconate

CHX has been used by many as an irrigating solution during or at the end of instrumentation. However, CHX has also been used as an intracanal medicament between appointments. As a disinfecting agent, interest has been focused on the effectiveness of CHX in gel form or as a mixture with $Ca(OH)_2$ as an intracanal interappointment dressing.[294,295] The information available is based mostly on *in vitro* and *ex vivo* experiments in which several intracanal medicaments have been compared for their activity against induced dentin infection. Siren et al.,[295] using a bovine dentin block model, reported that $Ca(OH)_2$ mixed with CHX was much more effective in disinfecting dentin infected with *E. faecalis* than pure $Ca(OH)_2$. Ercan et al.[294] reported 2% CHX gel was significantly more effective than $Ca(OH)_2$ combined with 2% CHX, or $Ca(OH)_2$ alone, against root dentin infected with *E. faecalis* and the yeast *C. albicans* after 7, 15, and 30 days of incubation. Similarly, it has been reported that 2% CHX gel alone completely inhibited the growth of *E. faecalis* after 1, 2, 7, and 15 days in the root canal, whereas $Ca(OH)_2$ allowed some microbial growth at all experimental times.[56] Interestingly, in this study, the combination of the CHX gel and $Ca(OH)_2$ had killed all bacteria in the one- and two-day samples, but failed to secure sterility in the 7- and 15-day samples. The antibacterial efficacy of intracanal medication with $Ca(OH)_2$, 2% CHX gel, and a combination of both was assessed in a clinical study in teeth with chronic apical periodontitis.[59] Bacterial samples were taken before and seven days after filling the canals temporarily with the medicaments. CHX and $Ca(OH)_2$, alone as well as their mixture, all performed equally well, and no statistically significant differences could be detected in their antibacterial effectiveness. Saatchi et al.[296] published a systematic review of the combined use of chlorhexidine and $Ca(OH)_2$ and concluded that mixing chlorhexidine with CHX did not improve its *ex vivo* antibacterial property as an intracanal medicament against *E. faecalis*.

Other Alternatives for Interappointment Medicaments

Intracanal Medicaments Containing Antibiotics

Throughout the history of endodontics, there have been time periods with increased interest in the use of local antibiotics as temporary canal dressings for root canal disinfection.[297–304] Locally used antibiotics have not, however, become an established part of root canal disinfection and eradication of the infection. There are several reasons for the failure of antibiotics to overtake endodontic infection control. Many of the antibiotics tested are bacteriostatic, that may not be a good strategy for treating endodontic infections. Generally, bacteriostatic antibiotics prevent the growth of the microorganisms without killing them, giving the host defense a possibility to deal with the infection. However, in the necrotic root canal there is no host defense because of the lack of blood circulation. Therefore, the antibacterial effect of such antibiotics in the root canal may be

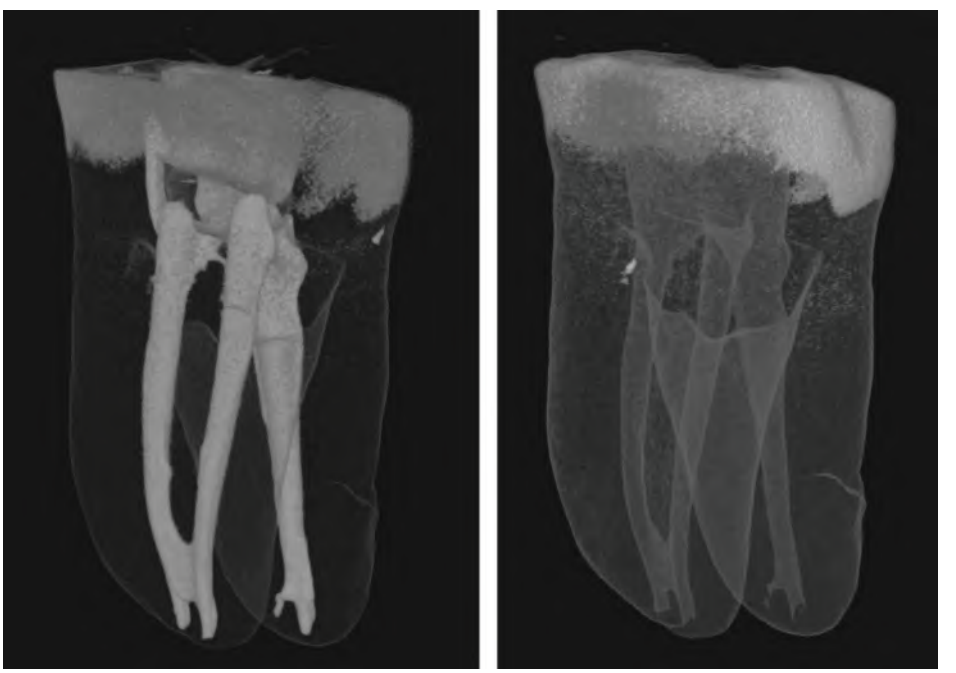

FIGURE 21-26 Micro-CT scans of a mandibular molar. **Left:** canals filled with calcium hydroxide paste; **Right:** same tooth scanned after removal of the calcium hydroxide using the GentleWave multisonic irrigation.

only temporary. On the other hand, it is possible that some bacteriostatic antibiotics can have a bactericidal effect when used in high concentrations; usually this is the case with locally used antibiotics. However, information about this is scarce and presently there is no direct evidence of bacteriostatic antibiotics used in the root canal, although one study indicated that mixing erythromycin with $Ca(OH)_2$ improved the effectiveness against *E. faecalis* as compared to $Ca(OH)_2$ alone.[305]

With bactericidal antibiotics, the potential problem in the root canal may be the metabolic and physiological state of the microorganisms. Many bactericidal antibiotics are most effective when the microbial cells are in active growth phase, that may not be the case in the necrotic root canal where only limited nutrients are available. In general, specific information about the effectiveness of intracanal antibiotics in infection control in endodontics is limited. In regenerative endodontics, antibiotic cocktails have been used in the treatment of teeth with immature apices and apical periodontitis.[306,307] It is possible that better circulation and survival of sufficient pulpal cells in the apical root canal are among the key factors for the promising results been reported so far. Future research will show whether this approach can be extended to the treatment of teeth with closed apices and apical periodontitis.

Phenol Compounds

Chemicals of the phenol group such as phenol, formocresol, cresatin, parachlorophenol (monoparachlorophenol), camphorated phenol, and camphorated parachlorophenol (CMCP) have a long history in endodontics as locally used root canal disinfecting agents. They have been applied into the pulp chamber via a moist cotton pellet (vapor effect), or the whole canal has been filled with liquid, using various concentrations of the phenol compound.[10,308–315] The rationale of using phenol compounds for root canal disinfection is their past role as general disinfecting agents. However, emphasis on safety in addition to effectiveness has resulted in a dramatic decline in their general use. Also in endodontics, concerns have been raised regarding the toxicity and possible mutagenicity of the disinfecting agents of the phenol group.[311,316–319] There are several demonstrations of their cytotoxicity,[316,320,321] however, other studies indicate that the risk of genotoxicity by the various phenol compounds used in endodontics is small.[317–319] Comparative studies of the antimicrobial effectiveness of the phenol compounds have not been able to show superiority of one over the other.[308,312,316] In addition, Byström et al.[10] reported that $Ca(OH)_2$ was superior to camphorated parachlorophenol (CMCP) in its antibacterial potential when used for four weeks as the local intracanal medicament. Several studies have indicated relatively rapid loss of activity of CMCP in the canal, although the results show variation.[313,322] Considering the benefits and the demonstrated and potential weaknesses of phenol compounds, it can be predicted that they will soon be replaced by other, more biological disinfecting agents.

Current Irrigation Recommendations

Based on growing amount of research evidence, current recommendations for irrigation protocol in conventional positive pressure needle suggests the use of a small size needle, such as a 30 gauge needle, with a side-vented or closed-end design, and with the needle placed in a non-binding manner 1 to 3 mm from the working length or at least in the apical third.[68,154,196] In order to avoid high apical pressure, and yet to gain maximum exchange of irrigant beyond the needle tip, one should use an irrigant flow rate of 4 to 6 mL/min, without sacrificing irrigation effectiveness. If open-ended needles are used, the safe distance from the apical foramen/working length is approximately 3 mm.[66] Negative pressure irrigation offers excellent safety and good cleaning in apical canals. Use of various means of physical energy to facilitate irrigation is recommended. It should be emphasized that without proper apical instrumentation of sufficient size effective irrigation of the apical root canal is not possible.

CONCLUDING REMARKS

Irrigation remains an important part of root canal treatment. Cleaning of the canal of organic matter and debris, together with the eradication of the microbial biofilms, are crucial components for a successful treatment. While numerous studies have shown the difficulty to completely clean and disinfect the root canal, the introduction of new strategies and various types of energy sources to enhance the action of the existing materials promises an exciting future in this area.

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Chapter 22

Obturation of the Radicular Spaces

Arnaldo Castellucci

The ultimate clinical objective of root canal treatment is the three-dimensional obturation of the endodontic spaces after being completely cleaned, shaped, and disinfected. The purpose of obturation is to seal the main root canals and all "portals of exit" to impede any future communication, or exchange, between the endodontium and periodontium. The filling must, therefore, completely and durably fill the root canal space so that no empty spaces should remain. It has been demonstrated that many endodontic failures are related to incomplete root canal obturation.[1–3]

BIOLOGICAL CONSIDERATIONS FOR ROOT CANAL OBTURATION

In 1931, Rickert and Dixon[4] proposed the "hollow tube" theory: an empty space within a living organism tends to fill with tissue fluids within a short period of time. This theory was based on the observation of an inflammatory reaction around the ends of hollow steel and platinum anesthetic needle fragments implanted in experimental animals. This reaction did not occur if the implant was made of a solid, non-porous material.[4] Two years later, Coolidge[5] arrived at the conclusion that, just as within unfilled or underfilled root canals, fluids that accumulate within empty spaces are rapidly colonized by microorganisms reaching these spaces by means of "anachoresis" and causing inflammtory reaction. The term anachoresis refers to bacteria transported by the blood circulation (bacteremia) colonizing a tissue, where they remain sheltered from phagocytosis by the organism's defenses.

For years, this theory has influenced the concept that the root canals must be filled to the apex. If not, empty spaces would quickly be colonized by bacteria, through anachoresis, and would prevent or delay healing of the periapical tissues.

Later studies have shown evidence refuting the "hollow tube" theory.[6–10] It has been demonstrated, in experimental animals, that empty spaces made inside plastic teeth, implanted in fresh sockets, did not produce any inflammation around the open ends.[9] Furthermore, in many cases these spaces were subsequently filled with fibrous tissue or bone.[9] The latter occurred more frequently with larger size apical openings.[9] Delivanis et al.,[11] using an animal model, could not prove the existence of anachoresis within an empty tube filled only with tissue fluids or in a root canal following pulpectomy. These studies indicated that empty spaces within a living tissue are not necessarily accompanied by inflammation or tissue destruction; on the contrary, they can be associated with physiological repair (Figure 22-1).[7,8]

The selective localization of blood-borne bacteria in areas of chronic inflammation is a well known phenomenon.[12–17] It may explain the egress of microorganisms into a pulp that has not been exposed to the oral environment but has been compromised by a trauma. In order for anachoresis to occur, the presence of blood vessels is necessary. Microorganisms can easily invade a space where tissue is present, inflamed, or partially necrotic, traveling via the blood circulation. However, this is not the case where active blood circulation doesn't exist.

It is prudent that clean and shaped root canals must be completely obturated to prevent re-infection. However, it

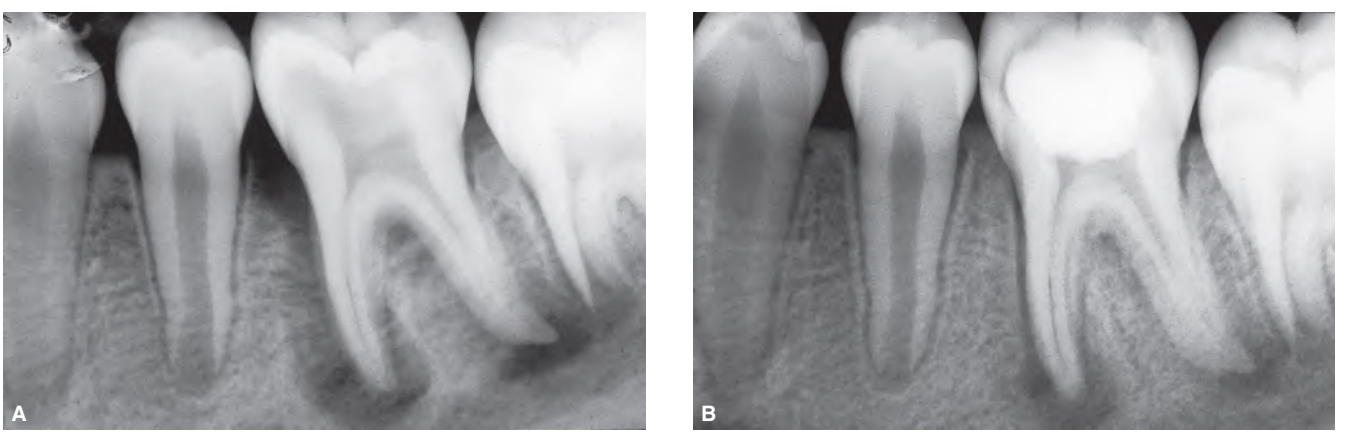

FIGURE 22-1 ***A***. Preoperative radiograph of a mandibular left first molar. ***B***. Radiograph taken two months later. The gap in treatment was due to negligence on part of young patient. Four canals had been cleaned, shaped, and medicated with Cresatin. Note the reduction in the periapical radiolucency in spite of the canals not been obturated.

is recognized that complete *sterilization* and removal of *all* pulpal debris from an infected root canal is very difficult, if not impossible[18–23] (Figure 22-2). What is achieved, in most cases, is the *disinfection* of the root canal system. For more details, see Chapters 3 and 21.

Residual microorganisms may remain inside the root canal system, mainly within dentinal tubules.[24] Presence of necrotic pulp remnants, together with the accumulating exudate, can contribute to their viability. If, however, the root canal system is completely obturated in all three dimensions, any remaining microorganism may be "entrapped" without nutritional sources and with reduced possibility of proliferation[25–29] (Figure 22-3).

Sjogren et al.[30] studied the influence of the presence of infection at the time of root canal filling, on the outcome of endodontic treatment, in teeth with apical periodontitis. They theorized that success obtained, despite positive bacterial cultures, was due to the bacteria "entombed" in the canal. Confirming Morse's findings,[31] Moawad[32] has demonstrated that bacteria entrapped within a completely filled root canal become nonviable within five days after root canal filling. Peters et al.[29] demonstrated that there is no evidence that special measures (calcium hydroxide or iodoformic paste) should be taken to kill the bacteria in the dentinal tubules. Those bacteria either do not survive the treatment, become inactive, or remain in insufficient numbers to cause or sustain infection.[33–35] Other in vivo studies demonstrated that most bacteria in the dentinal tubules died within 24 hours after removal of the nutrient medium.[36]

In conclusion, the reason for a complete root canal obturation is not only to prevent *apical leakage* but also to prevent *coronal leakage*. Figure 22-1 shows healing in progress without any obturating material in the root canals. However, if the coronal seal was compromised, the entire root canal system would be reinfected and a periapical lesion would reappear.

Obturation with gutta-percha and sealer after chemomechanical cleaning and *disinfection* with sodium hypochlorite also deprives the remaining microorganisms from their nutrient supply, thereby reducing their ability to cause or maintain disease. It has been demonstrated that most microorganisms in the dentinal tubules died within 24 hours after removal of the nutrient medium.[36] Gutta-percha has a certain bacteriostatic activity, perhaps due of its zinc oxide content.[37]

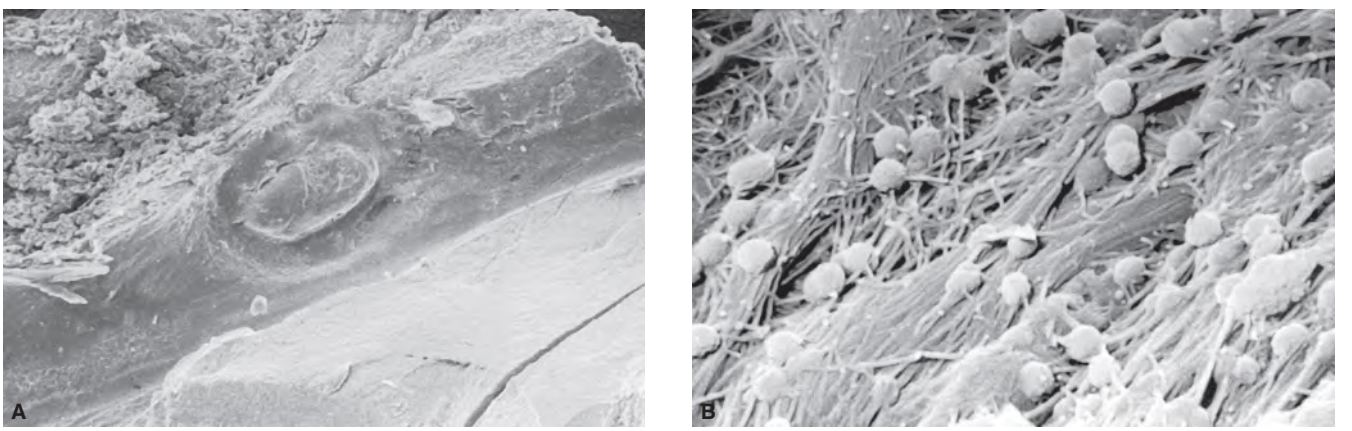

FIGURE 22-2 ***A***. Scanning electron microscopy (SEM) view of the apical third of a cleaned and shaped mandibular incisor. Note the presence of a large calcification adhering to the canal wall a few millimeters from the foramen (original magnification ×60). ***B***. Higher magnification view of A showing the presence of organic material in the area immediately apical to the calcification, where the instruments could not reach (×4,000).

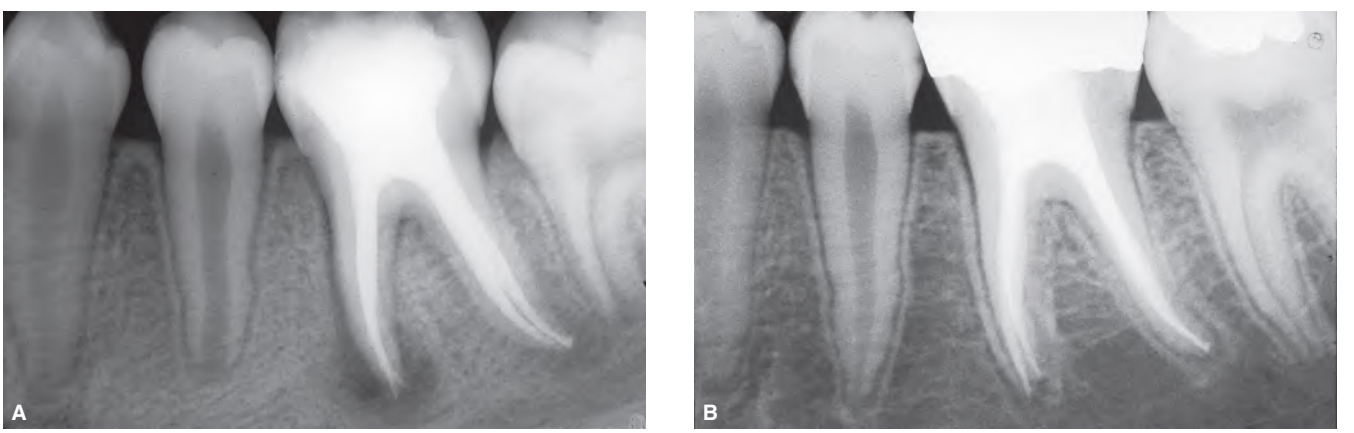

FIGURE 22-3 ***A***. Postoperative radiograph following canals obturation of the case shown in Figure 22-1. ***B***. Two-year recall radiograph showing active healing.

However, this antimicrobial activity is very limited and gutta-percha and sealer alone should not be used without prior disinfection and shaping of the root canal system.

SELECTING THE CORRECT TIME FOR OBTURATION

Once cleaning and shaping of the root canal system has been completed, the clinician may proceed to the obturation procedure when the following requirements are met:

- The tooth is asymptomatic and the patient must feel perfectly comfortable.
- There is no exudate from the canal.
- The canal can be proporly dried.
- There is no foul odor indicating the presence of microorganisms.
- The temporary filling is intact if the root canal has been shaped in a previous visit.

In the past, obtaining a negative bacterial culture was advocated prior to obturation. Today, bacterial cultures are obtained less frequently[27]. Bacterial growth occurs only in the presence of substrates in the canal, but if the canal is completely free of such protein degradation products, bacteria cannot reproduce. Bacterial cultures may not necessarily determine the success of therapy, nor does not performing them lead to failure. While some consider culture obsolete, others have found them to be important.[27] Clearly, a reliable chair-side culturing method that produces immediate results would be beneficial.

APICAL EXTENT OF OBTURATION

Cemento-dentinal Junction

Grove,[38] in 1929, suggested that canal obturation must stop at the cemento-dentinal junction, that corresponds to the maximal apical constriction. The pulp tissue terminates at this point, and the tissue of the periodontal ligament begins. The walls are no longer of dentin, but of cementum. In theory, this view is correct in that the apical constriction should ensure a good barrier to the filling material respecting the periodontium. By obturating to this point, the root canal is obturated without invading the periapical tissues.

On the other hand, Coolidge,[39] also in 1929, claimed that the site of the cemento-dentinal junction is so variable that attempting to use it as a landmark is of little help to the endodontist. This junction often has unclear limits and can be found at different levels within the root canal (Figure 22-4). The cemento-dentinal junction can sometimes be found on the external surface of the root.[40] Skillen[41] emphasized that it is histologically impossible to define a clear line of demarcation between the pulp on one side and the "periodontal membrane." It is also histologically impossible to find a point within the root canal where the pulpal tissue ends and the periodontal tissue begins (Figure 22-5). In agreement with Coolidge, Orban[42] stated that from a practical point of view

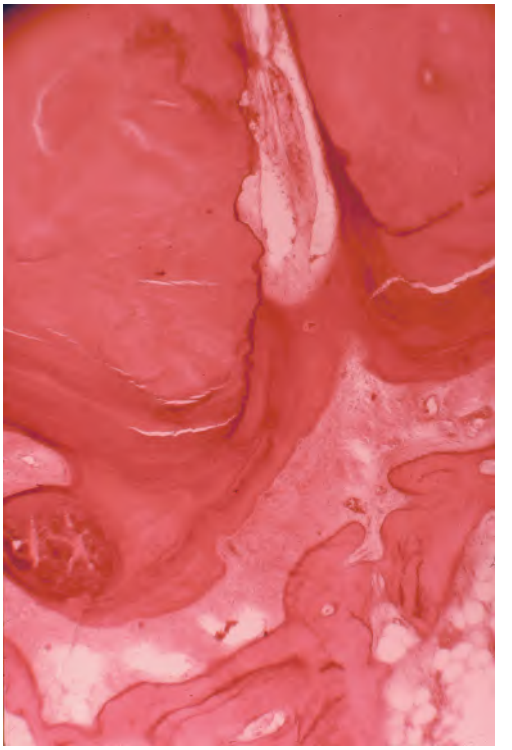

FIGURE 22-4 The exact position of the cemento-dentinal junction is difficult to locate, even histologically.

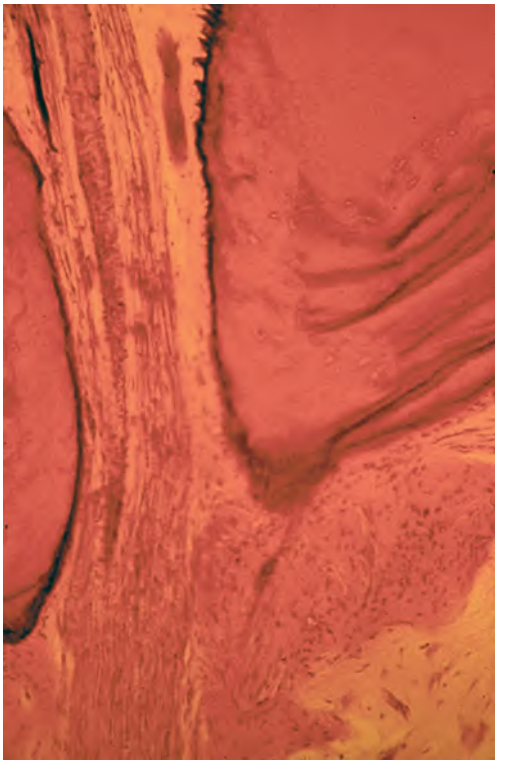

FIGURE 22-5 The neurovascular bundle of the pulp has the same characteristics both before and after crossingthe apical foramen.

it is not possible to use the cemento-dentinal junction as a boundary of endodontic obturation. When identified, more often than not, it is by chance.

Additionally, relying on tactile sensation to locate the cemento-dentinal junction can also be misleading. The maximal constriction of the canal lumen may be due to the narrowing of the canal (Figure 22-6) or to a calcification that may vary in distance from the true end of the endodontium (Figure 22-7).

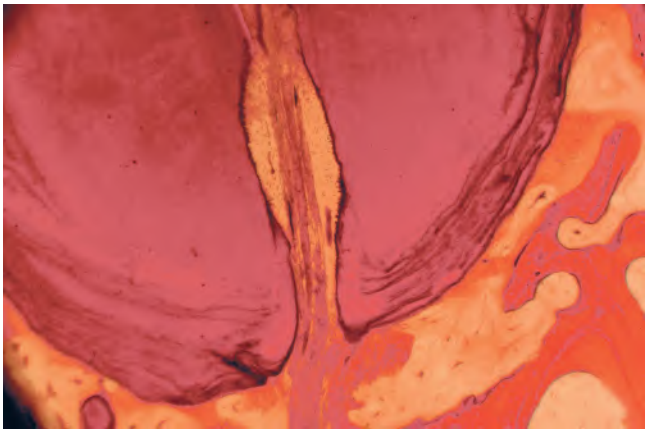

FIGURE 22-6 The apical constriction in this case is due to a narrowing of the canal lumen, not due to the cemento-dentinal junction.

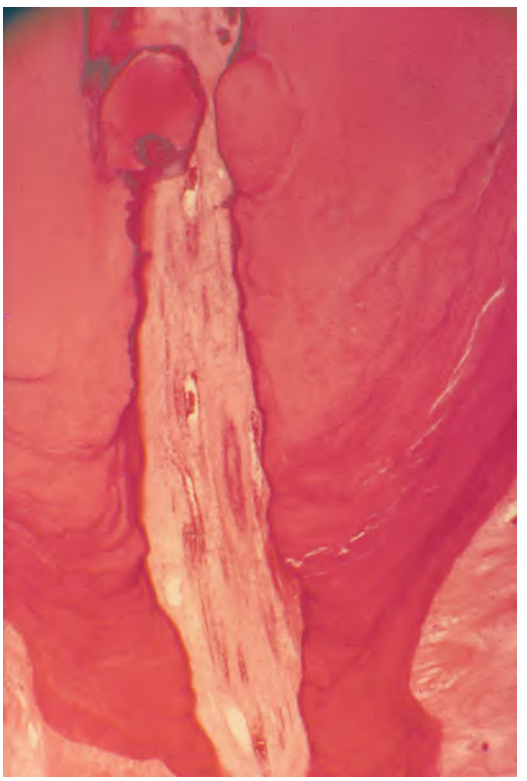

FIGURE 22-7 Apical constriction occurred due to a calcification located further away from the cemento-dentinal junction.

Ricucci and Langeland[43] suggested that the apical limit of canal instrumentation and obturation should not be the radiographic terminus of the canal, nor should it be the cemento-dentinal junction or the distance of 1 mm from the radiographic apex, but rather the "apical constriction." This anatomical location, however, cannot be determined clinically with accuracy since it is "ever-changing"; it has been demonstrated as far as 3.8 mm from the anatomic apex.[44] Therefore, "the instrumentation and the obturation should terminate where the instruments stop" (Figure 22-8).[43] Others[45,46] suggest that the arbitrary rule that canal preparation should terminate 1 mm short of the radiographic apex should not be accepted in modern endodontic therapy. The "one millimeter" technique could result in instrumentation short of the true canal terminus, possibly leaving necrotic and infected debris behind, leading to treatment failure. According to Schilder,[47] having *three-dimensionally* obturated root canal as far as 0.5–1 mm from the radiographic terminus of the canal is in practice equivalent to having filled it completely, leading to the success of the therapy.

To address the conflicting views in the literature, one must note that the *radiographic terminus* of the canal refers to the point at which the endodontic instrument, within the root canal, radiographically encounters the external profile of the root. It is not to be confused with the *anatomical apex*, that represents the geometric vertex of the root, or with the *radiographic apex*, that represents the anatomic apex as seen on the radiograph. The terminal point of the root canal is not always at the radiographic apex (Figure 22-9) and therefore cannot be used as a reference point to the actual working length. The *endodontic* or *physiologic apex* indicates the cemento-dentinal junction, which usually (but not necessarily) corresponds to the narrowest area of the canal lumen. The term *apical foramen* refers to the opening of the root canal on the external surface of the root (Figure 22-10).

Radiographic Terminus of the Canal

Some authors claim that it is preferable to extend the obturation to the radiographic terminus of the canal, since this gives the greatest assurance of having obturated the entire root canal system, even if this sometimes leads to overfilling of few fractions of a millimeter beyond the apex. On the other hand, it is well known that some very curved canals emerge in the roots at points that are not radiographically visible, several millimeters away from the anatomic apex. In these cases, obturation to the radiographic terminus of the canal is to be avoided, because this would entail considerable excess, but at the same time filling the root canal 0.5-1 mm short is also to be avoided, because the filling material would be equally in the periapical tissues (Figure 22-11). Therefore the author of this chapter suggests to use the "electronic apex" as a landmanrk, in addition to the radiograph.

The successful outcome of cases obturated to the radiographic terminus of the canal (Figure 22-12) is therefore due to the completeness and three-dimensional cleaning,

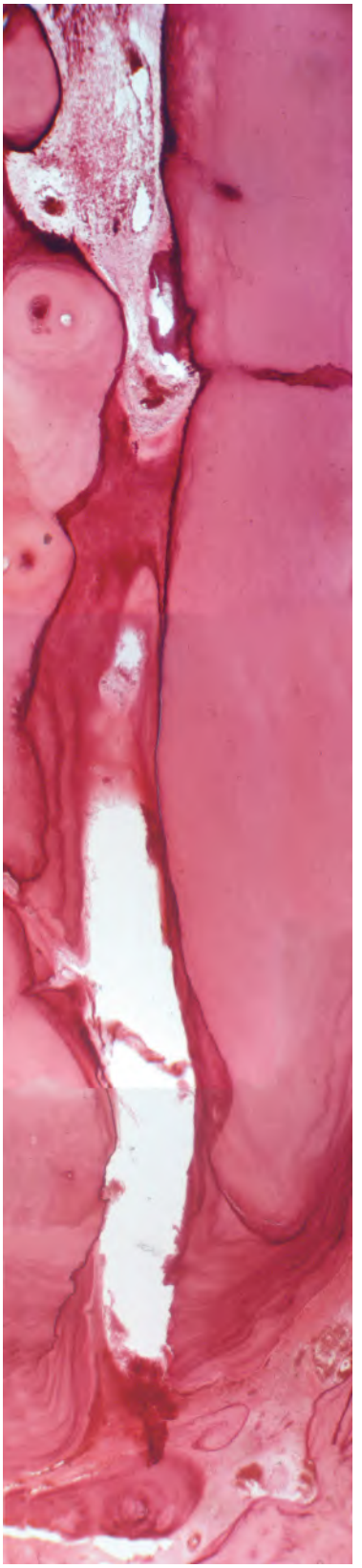

FIGURE 22-8 Completion of instrumentation and obturation at the "constriction" means clinically to remain 7 mm short (or even more), since the calcification causing a constriction is located more coronally. It will impede the introduction instruments more apically, thus not reaching the "correct" working length.

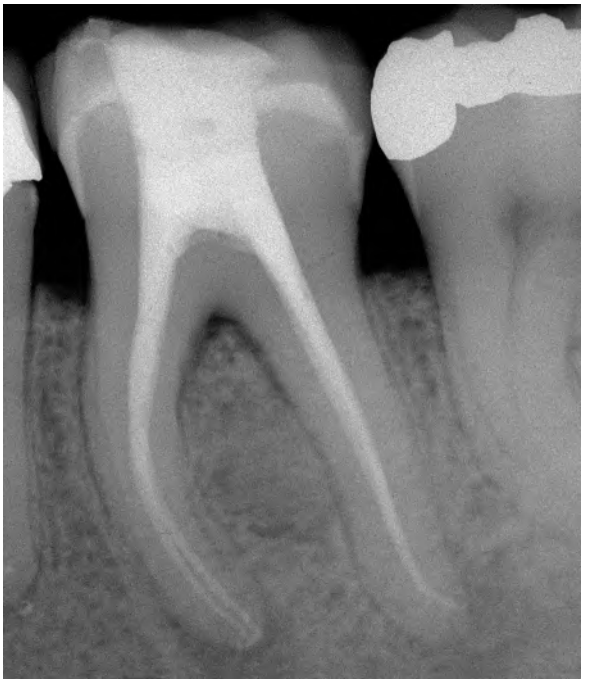

FIGURE 22-9 Postoperative radiograph of mandibular molar treated endodontically. In the distal canal, the canal apical foramen does not coincide with the radiographic apex.

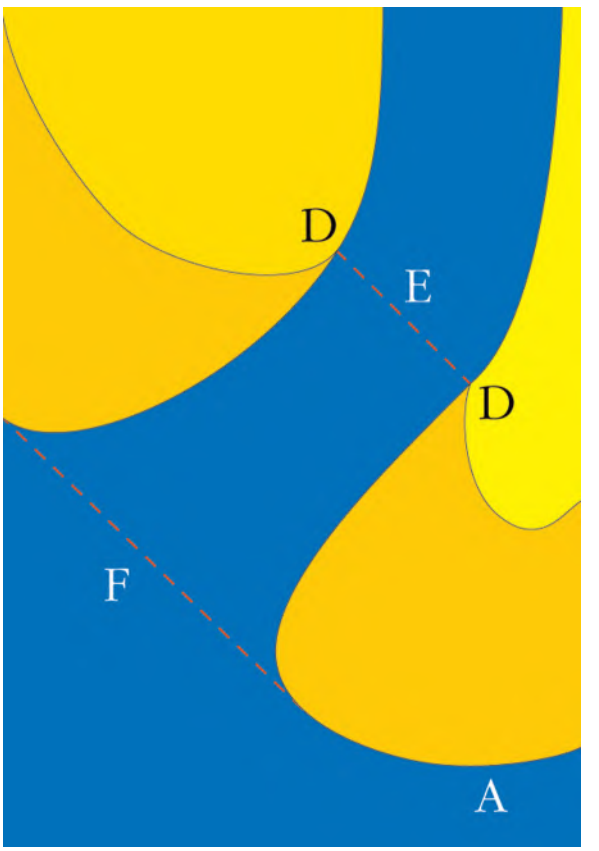

FIGURE 22-10 Schematic illustration representing the apical area of the root showing anatomic apex (A), radiographic apex (F), cemento-dentinal junction (D), and endodontic apex (apical foramen) (E).

shaping and of the obturation. The minimal overfilling is irrelevant, just as minimal underfilling is.

Sjogren et al.30 looked at root canals filled short and long, with positive and negative culturing results in each group. They showed that high success rates were achieved regardless of long or short filling when the culture came back negative, but when the culture came back positive only the fully filled cases worked predictably. The authors theorized that success was achieved because the remaining bacteria were **entombed** in the canal. Infected or not (among the bacteria present at the time of root filling in cases that healed successfully, the authors of the study found Peptostreptococcus anaerobius, Actinomyces naeslundii, Fusobacterium nucleatum and Enterococcus faecalis), all of the cases worked when the canals were filled to or beyond the terminus. From this interesting study, we can conclude the following: because no one can insure sterility in any given root canal space, the surest chance of clinical success is gained when root canal systems, in all of their complexities, are filled to their full apical and lateral extents, even though that means that there may be surplus material beyond the confines of the root canal space. This is in agreement with the findings of Peters et al.[49] who demonstrated that the presence of a positive bacterial culture at the time of filling did not influence the outcome of treatment.

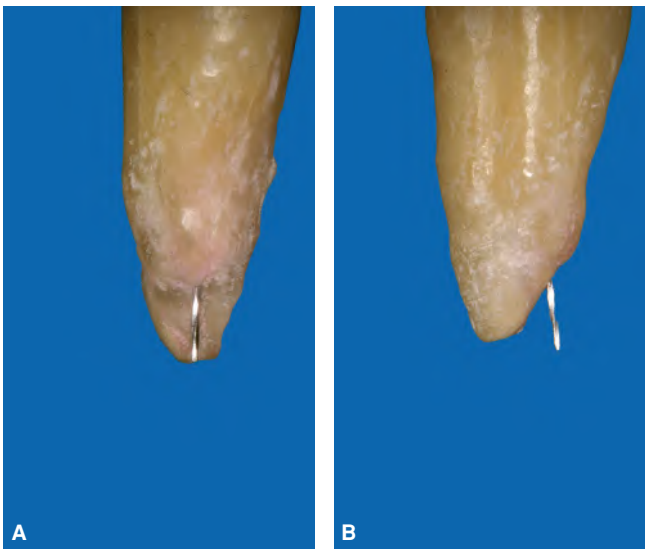

FIGURE 22-11 The apical foramen of this mandibular premolar opens on the buccal aspect of the root and is therefore not visible in a 2-D radiograph. ***A***. A buccal view photograph showing the distance between the opening of the apical foramen and the tip of the root. ***B***. Mesial view. This typical example illustrates the importance of terminating the canal preparation and obturation at the "electronic apex." Obturation of 0.5 to 1 mm short of the the radiographic will cause extrusion of material beyond the apical foramen.

In conclusion, the difference between the "radiographic apex" and the "radiographic terminus of the canal" must be well underdstood. If the root canal is not straight and the foramen is on the distal, or on the mesial aspect of the root, it is obvious that it has nothing to do with the "radiographic apex", therefore it makes no sense to advocate an instrumentation and then an obturation .05 mm or more from the radiographic apex. On the other hand, the mesial or distal opening of the foramen can be radiographically appreciated as the "radiographic terminus of the canal". On the other hand, when the foramen in on the buccal or on the lingual aspect of the root it is impossible to radiographically see the teminus of the canal. In such a case the instrumentation and obturation are performed at the "electronic apex" and the final result will appear to be "radiographically short".

Many reliable electronic apex locators are available today, and we know that they indicate the end of the canal (which is the foramen) and for sure they don't indicate the location of the cemento-dentinal junction or the apical constriction. Therefore we can assert that all of our endodontic treatments are performed at the "electronic apex", which about

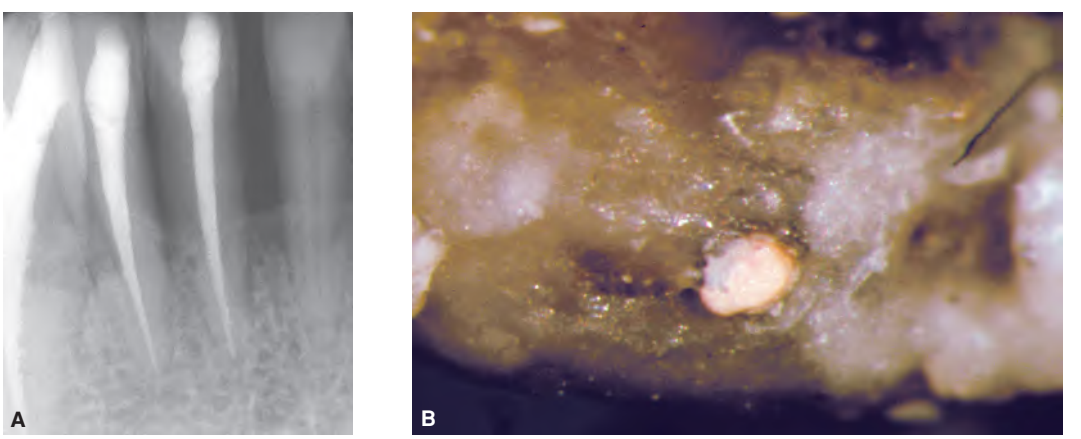

FIGURE 22-12 ***A***. Postoperative radiograph of two mandibular incisors treated endodontically. The obturation was done to the radiographic apex. ***B***. The central incisor was extracted for periodontal reasons eight years later and was photographed under a stereomicroscope. Note the gutta-percha that seals the apical foramen without extrusion (original magnification x64).

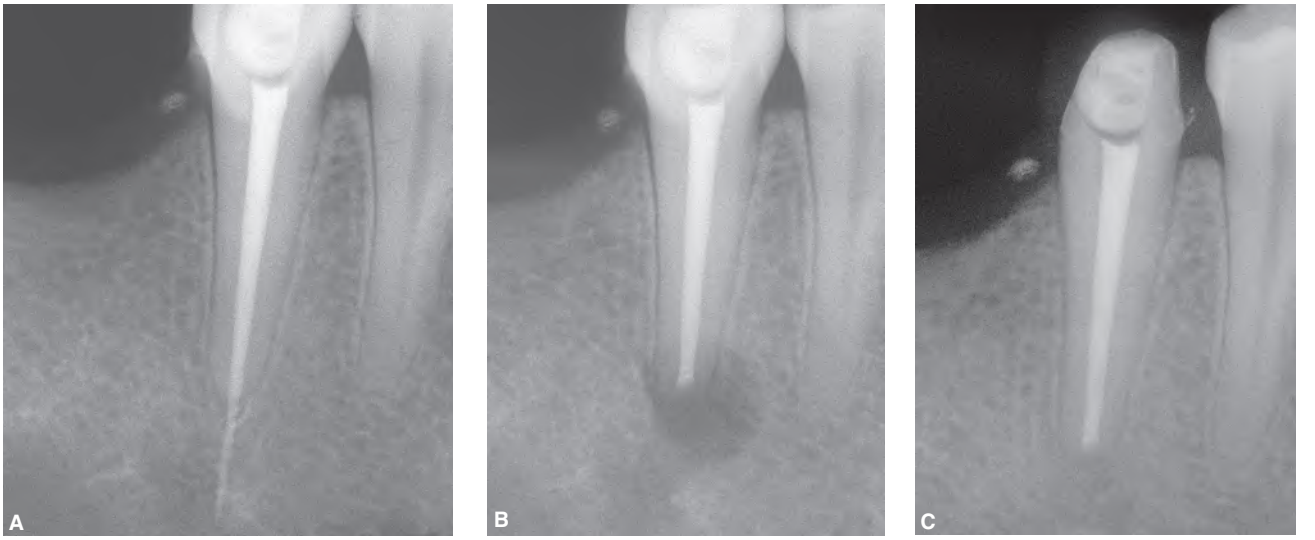

FIGURE 22-13 Excess of obturation material is an indication for surgical treatment if it does not seal the root canal system, or if it has been pushed against anatomical structures and the patient is symptomatic. In this case, the gutta-percha cone was pushed against the mental nerve and the patient developed paresthesia. ***A***. Preoperative radiograph. ***B***. Radiograph taken immediately after the apical surgery. Ultrasonic tips (piezoelectric surgery) were used since high speed burs could have further damaged the nerve bundle. Two weeks after the surgery the paresthesia disappeared completely. **C**. 2-year follow-up radiograph. The patient was asymptomatic and paresthesia did not recur.

50% of the times coincides with the radiographic apex because the root canal is straight. The other 50% of the times the root canal is not straight but is curved and when it makes a curvature, about 40% of the times the curvature is to the mesial or to the distal (so that the "radiographic terminus of the canal" can still be seen on the radiograph), and only about 10 % of the times the curvature is to the buccal or to the lingual. These are the cases where we must rely on the electronic apex locators only and these are the cases that appear to be "radiographically short".[48]

Overextension and Overfilling

Over- and under-extension refer to the vertical component of the obturation; either beyond or short of the apical foramen. Underfilling refers to obturation that has been performed inadequately in all dimensions (e.g., short filling in a longer and wider root canal, or only a cement filling with voids). Overfilling refers to obturation, that has been performed in three dimensions, in which a portion of material extrudes beyond the foramen. The situation of a canal with a slight overfilling is quite different from that of a canal with a vertical overextension and an underfilling. In the former, the obturation fills the entire endodontium in its three dimensions, and an excess of material extrudes from the apical foramen. In the latter case, the obturation material protrudes beyond the apex without sealing the apical foramen and thus without three-dimensionally obturating the root canal system (Figure 22-13).

Ingle[49] stated that high success rates can be achieved in spite of overfillings. Weine[50] states that fortunately, since

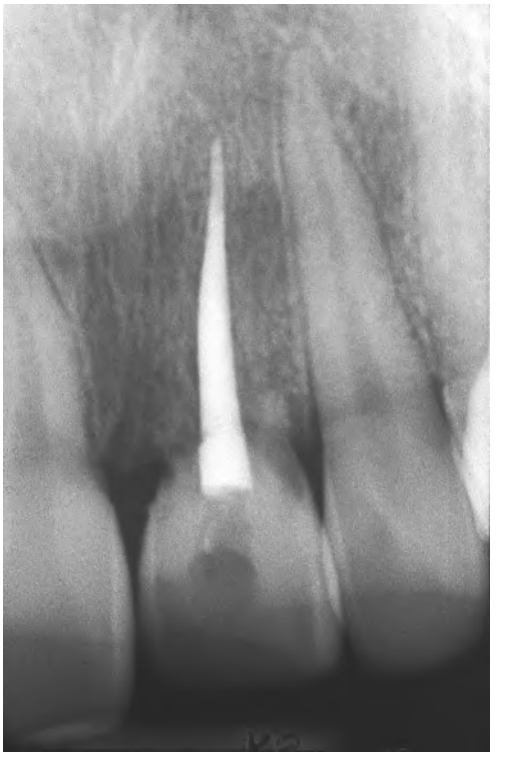

FIGURE 22-14 Radiograph of the maxillary left central incisor reimplanted after a traumatic avulsion that occurred 3 years previously. The root underwent replacement resorption. Radiographically, the gutta-percha appears to be in direct contact with bone and there is no radiographic evidence of inflammation.

gutta-percha is so well tolerated by periapical tissue, only rarely is a post-treatment failure noted in conjunction with an overfilling. Most instances show no abnormal radiographic evidence (Figure 22-14), and in some cases there is an actual amputation of the overfilling with phagocytosis of the extra mass (Figure 22-15). It appears that the major factors associated with endodontic failures are inadequate root canal debridement or incomplete root canal seal,[51–54] while the apical extent of root canal fillings is not a determining factor.[30,55,56,57]

Histologic studies on experimental animals by Deemer and Tsaknis[57] and by Tavares et al.[58] have demonstrated

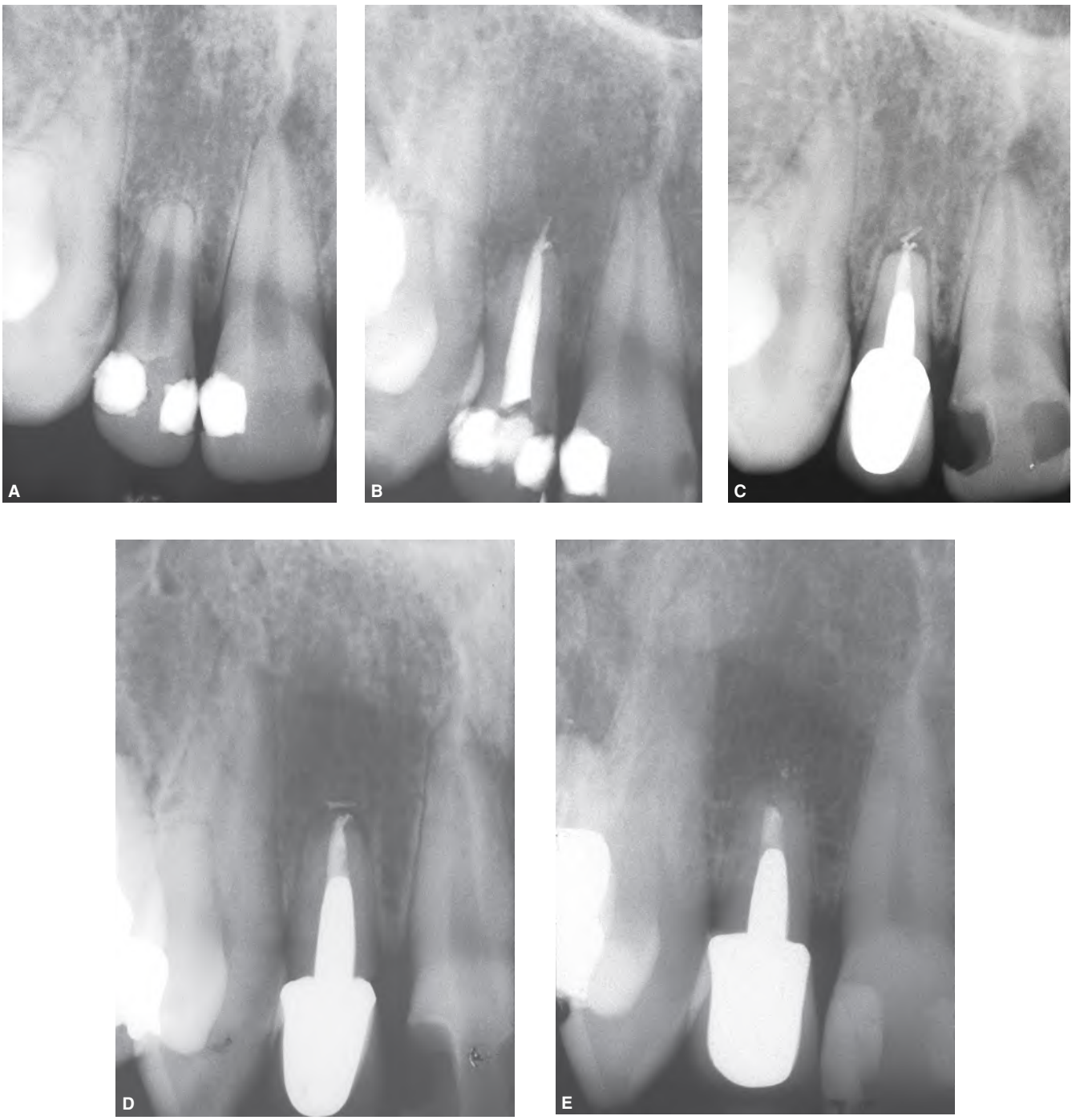

FIGURE 22-15 Endodontic treatment in a maxillary right lateral incisor with a resorbed root. ***A***. Preoperative radiograph. ***B***. Postoperative radiograph. The tip of the cone slid 2–3 mm beyond the apex, probably bacause the "tug-back" of the cone was not at the apical area. ***C***. One year later, the excess material appears to be sectioned at the apical foramen. ***D***. Two-year postoperative radiograph. The tip of the cone lies horizontally over the root apex. ***E***. 34-year recall confirmed that an overfilling in a three-dimensionally obturated root canal was NOT necessarily an indication for surgery or cause for endodontic failure.

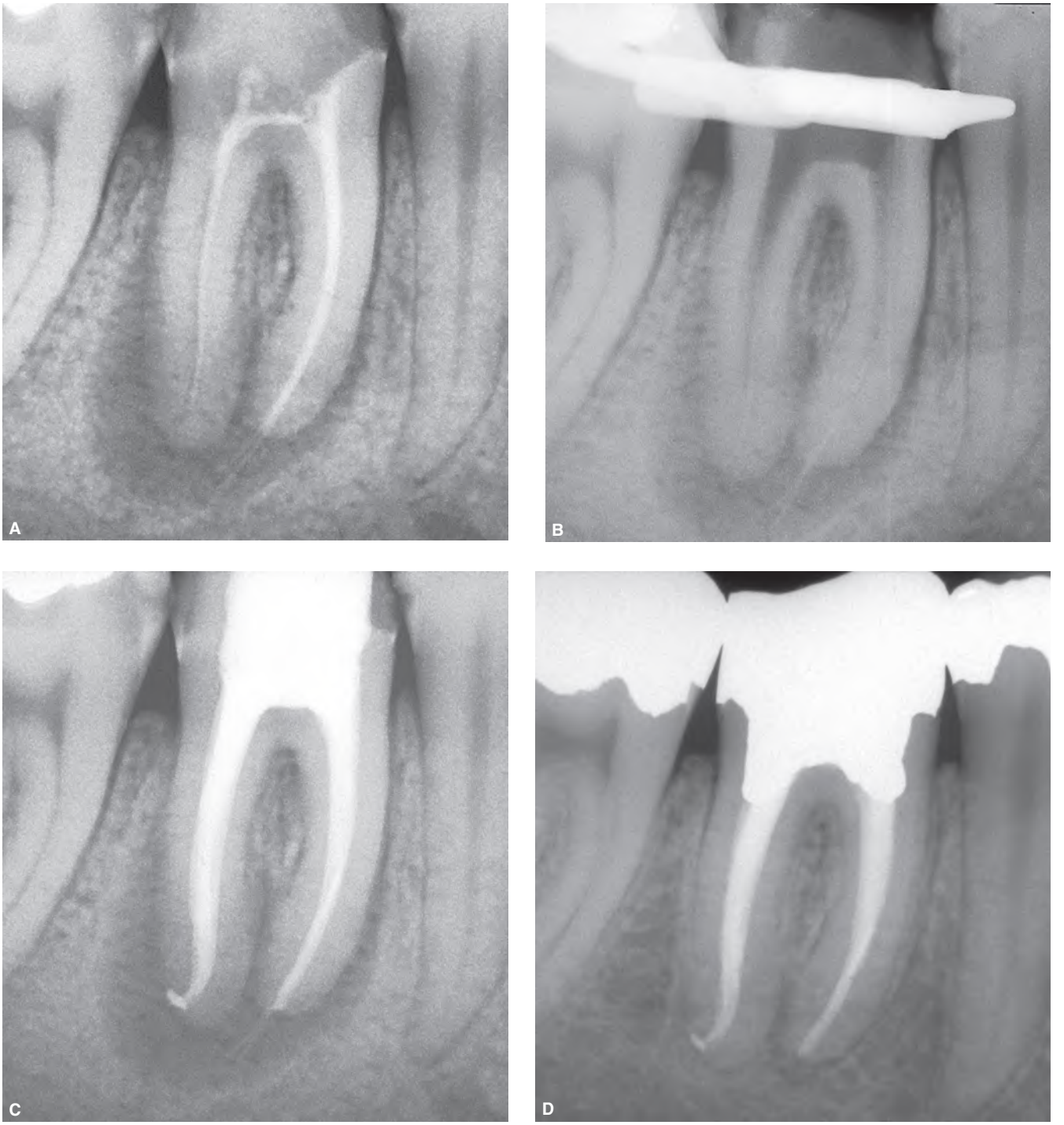

FIGURE 22-16 ***A***. Preoperative radiograph showing an improperly treated mandibular right first molar. The distal canal is underfilled and the mesial canals have gutta-percha cones overextending into the periapical tissues. ***B***. Intraoperative radiograph showing a failed attempt to remove the gutta-percha protruding beyond the apical foramen. ***C***. Immediate postoperative radiograph showing the two gutta-percha cones still present in the lesion. ***D***. 3-year recall radiograph. The apical lesion has resolved and the two gutta-percha cones completely disappeared. The apical lesion was most probaly caused by an infected root canal system and NOT by a foreign body reaction to the overextended gutta-percha.

that gutta-percha is well tolerated by the surrounding tissues. Their results agree with those of Schilder [59] on human specimens. Gutierrez et al.[60] demonstrated in experimental animals that gutta-percha in contact with the tissues and tissue fluids disintegrates and is subsequently removed by macrophages. This corresponds to the clinical phenomenon noted in the periapical region of human teeth. Bergenholtz et al.[61] stated that in the case of overfilling, the filling

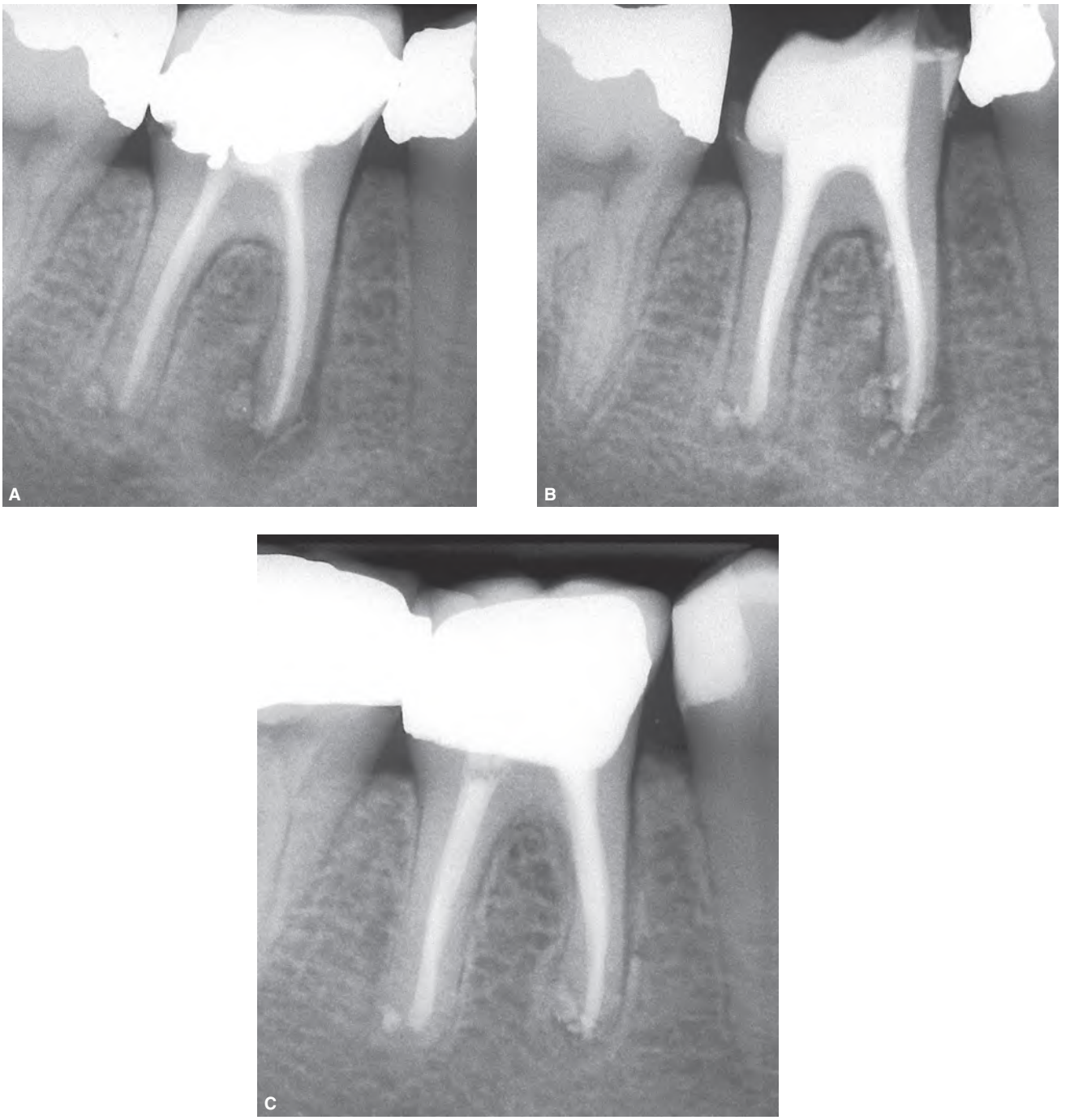

FIGURE 22-17 ***A***. Preoperative radiograph of the mandibular right first molar improperly treated. A radiolucency is associated with the apex of the mesial root. Filling material (sealer and/or gutta-percha) is present in the lesion. ***B***. Postoperative radiograph. During retreatment two missed root canals from the prior treatment were found. The distolingual and the middle mesial canals had independent foramina. ***C***. Seven-year recall radiograph showing that the apical lesion has resolved and the excess material disappeared. Again, the apical lesion was most probably caused by an infected root canal system and NOT by a foreign body reaction to the overextended gutta-percha.

material *per sé* is not necessarily the immediate cause of failure. Gutta-percha, both *in vitro* in cell cultures,[62] and in *in vivo* implants in experimental animals,[63,64] has been shown to be biocompatible. Recent radiographic studies have shown that, given time, the extruded canal filling materials would not cause chronic inflammation and eventually be removed from the periradicular tissues.[55,65–70] The factors that impede healing in cases of overfilling need further investigation.

Yusuf[66] has demonstrated inflammation around the small pieces of dentin and cementum found beyond the apex in the granulation tissue, where they act like a foreign body.

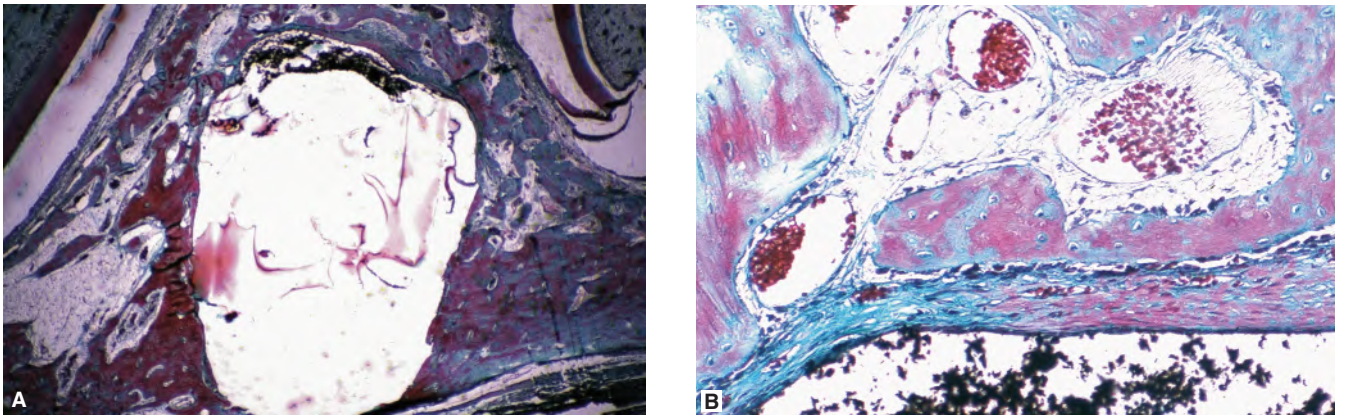

FIGURE 22-18 Osseous reaction to implanted Pulp Canal Sealer at four weeks. ***A***. Normal bone marrow (Original magnification: ×5; Masson's Trichrome). ***B***. Layer of fibrous connective tissue between the Pulp Canal Sealer and bone, without evidence of inflammatory cells. Normal appearance of the bone with viable osteocytes bordered by a layer of osteoblasts. (Original magnification ×25; Masson's Trichrome). (Courtesy of Dr. Wilhelm-Joseph Pertot, Paris, France.)

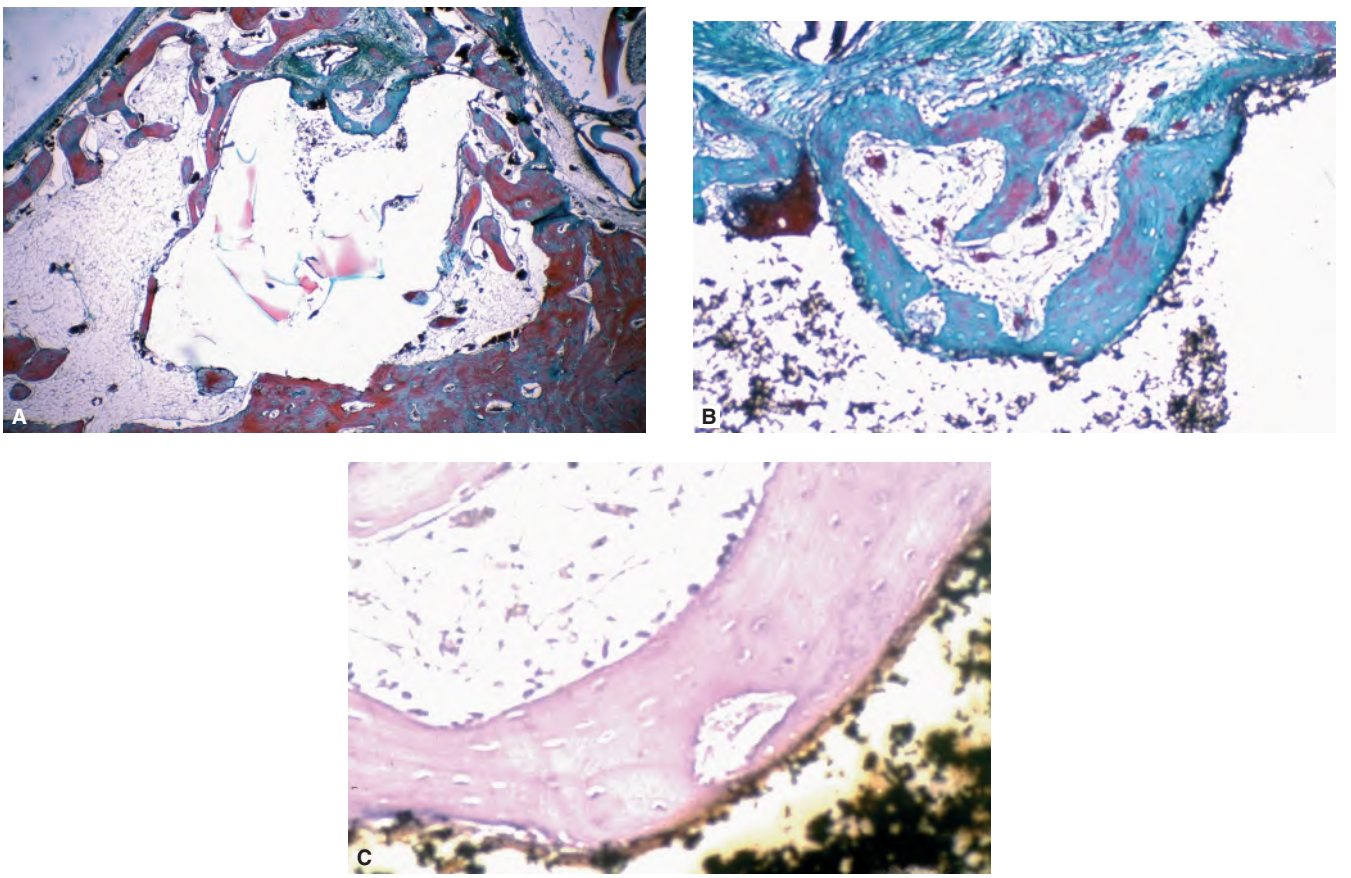

FIGURE 22-19 Osseous reaction to implanted Pulp Canal Sealer at 12 weeks. ***A***. Normal bone marrow with new bone ingrowth into the implant (Original magnification ×5; Masson's Trichrome). ***B***. New-formed bone is in direct contact with the Pulp Canal Sealer without fibrous interposition or inflammatory cells (Original magnification ×10; Masson's Trichrome). ***C***. Normal appearance of newly formed bone with viable osteocytes and normal bone marrow (Original magnification ×50; Hematoxylin-eosin). (Courtesy of Dr. Wilhelm-Joseph Pertot, Paris France.)

In contrast, small pieces of amalgam or other canal obturation materials were usually associated with a fibrous reaction and encapsulation, without active inflammation. Thus, accidental minor overfilling of a properly cleaned, shaped, and three-dimensionally filled canal is *not* desired but is *not* an indication for surgical removal of the excess material unless there are clear signs of treatment failure (Figures 22-16 to 22-23).

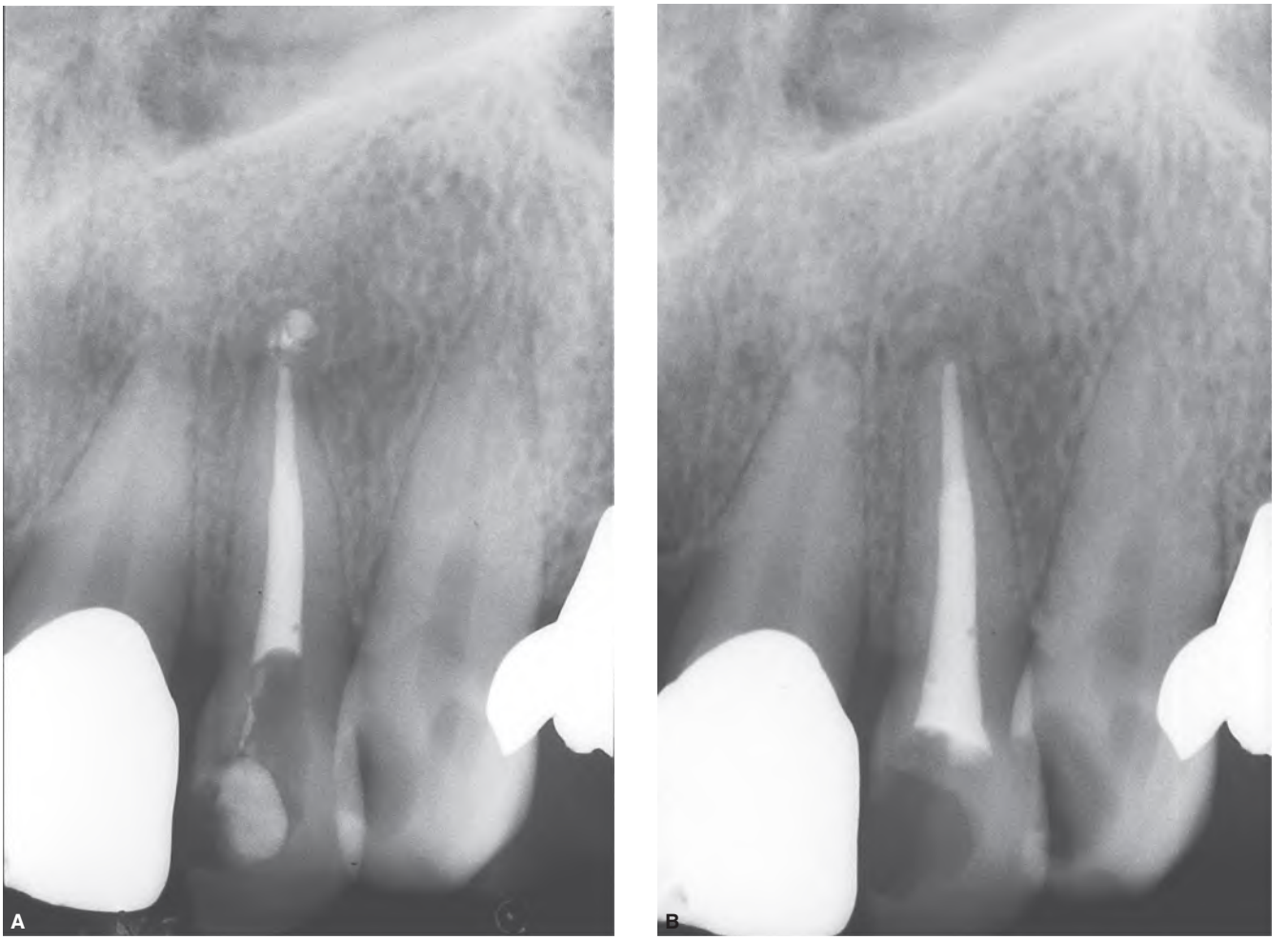

FIGURE 22-20 ***A***. Postoperative radiograph of a maxillary left lateral incisor treated endodontically. Note extruded material (probably sealer) beyond the apical foramen. ***B***. Radiograph taken 7 months later showing the excess material has disappeared.

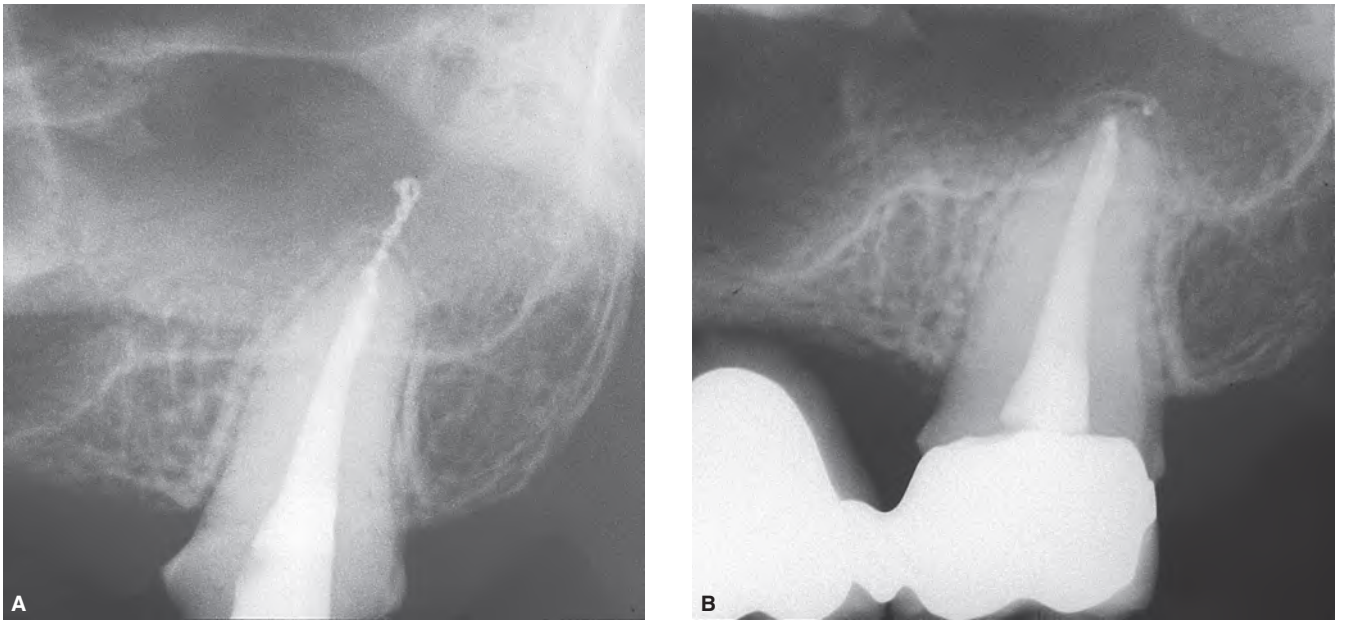

FIGURE 22-21 ***A***. Postoperative radiograph of a maxillary left second molar treated endodontically. Note extruded material beyond the apical foramen. ***B***. Radiograph taken eight months later showing the excess material is no longer present.

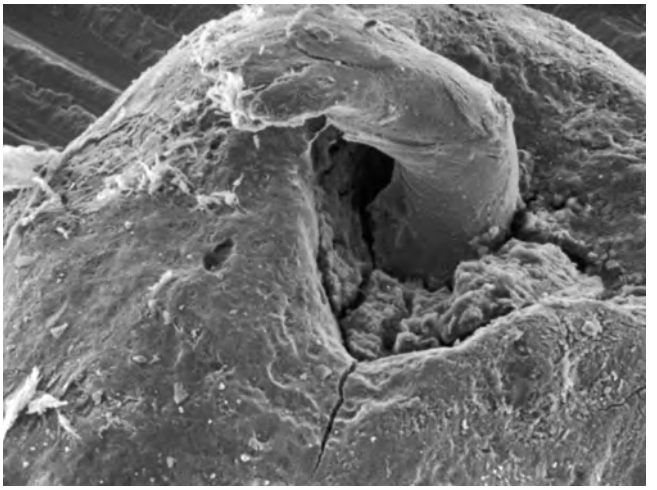

FIGURE 22-22 Scanning electron micrograph of a trasported root apex causing extrusion of the gutta-percha cone. The cone, although surrounded by sealer, is not sealing the foramen.

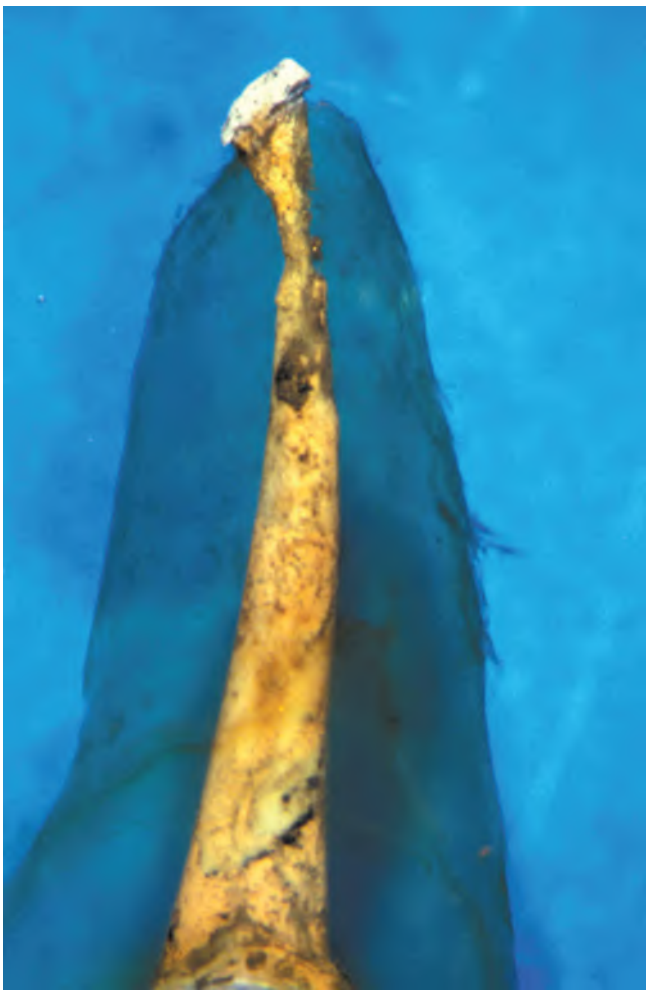

FIGURE 22-23 The root of this maxillary lateral incisor has been cleared. Transportation of the apical foramen, and extrusion of material through the tear-drop foramen had occured. Following such a mishap, properly sealing the apical foramen is very challenging.

REQUIREMENTS FOR AN IDEAL ROOT CANAL FILLING MATERIAL

Many substances have been used to obturate the root canals. Grossman,[71] in 1958, wrote: "I doubt very much whether there is any hollow cavity in the body that has been plugged with as many different materials as the root canal of a tooth."

Requirements for the ideal root canal filling material have been suggested.[72–76] According to West,[3] the ideal material must be:

1. Capable of being fully adapted to the prepared root canal walls
2. Dimensionally stable
3. Non-resorbable for an indefinite period of time
4. Non-irritating
5. Bacteriostatic, or at least should not encourage bacterial growth
6. Nonstaining to teeth
7. Preferably semisolid upon insertion and solid afterward
8. Capable of sealing canals laterally as well as apically
9. Impervious to moisture
10. Radiopaque
11. Sterile or sterilizable
12. Easily removed from the root canal, if necessary
13. Easily manipulable
14. Sticking to the canal walls
15. Nonconductor of thermal changes
16. Slightly expandable after placement
17. Able to set in a reasonable period of time.

Generally, root canal filling materials are divided into pastes and cements, solid materials and semi-solid materials.[77]

PASTES AND CEMENTS

Sealers are self-hardening ***cements*** used in conjunction with solid or semi-solid materials that serve as the core of the obturation. Sometimes, clinicians use certain ***cements*** to fill the entire root canal without another core obturation material. These cements set and transform into a firm mass after insertion in the canal. ***Pastes*** (such as Iodoformic paste or calcium hydroxyde) are used to fill the entire canal. In contrast to sealers/cements, however, they do not harden once placed in the canal and are easily resorbable. Consequently, they are not used as permanent filling materials.

Endodontic sealers are resorbable,[78] (some more than others) even within the root canal. Their use as the sole filling material is not encouraged.[79]

It would be desirable to fill the entire canal with a core solid or semi-solid material. In practice, however, this is not possible, because empty spaces (voids) will remain between the canal walls and the filling material. Sealers, therefore, have become indispensable to fill those voids and adhere the core obturation material to the canal walls.

Ideal sealers should be:

- easily manipuled with ample working time
- easily mixed in very fine powder particles and liquid form
- tacky when mixed and adhesive to the canal walls when set
- biocompatible
- expansible while setting
- inert
- physically stable (nonshrinking after setting)
- nonresorbable
- insoluble in tissue fluids
- radiopaque
- nonstaining of tooth structure
- bacteriostatic
- easily removable with common solvents, if necessary
- nonimmunogenic in the periapical tissues[80–82]
- neither mutagenic nor carcinogenic[83,84]

Sealers are designed to improve the seal (hence their name "sealers") provided by the core obturation material.[85] The sealer should be used in small amounts; as minimal as practical. It should be deposited on the canal walls as a microfilm of only a few microns in thickness.

Zinc Oxide-based Sealers

Zinc oxide is the major component of many sealers used in endodontics.[86] The most commonly used are *Grossman's Sealer*, *Roth's 801 Sealer*, and *Rickert's Sealer.*

Grossman's Sealer contains zinc oxide, staybelite resin, bismuth subcarbonate, barium sulfate, and sodium borate (anhydrous) with eugenol as the liquid component.[87] It has been marketed as Procosol Sealer, as well as under other commercial names. *Roth's 801 Sealer* is similar to the Grossman's sealer, containing bismuth subnitrate instead of bismuth subcarbonate.

Rickert's Sealer has been used for many years, meeting most of Grossman's requirements for an ideal sealer. For radiopacity, it contains silver and, therefore, may stain tooth structures.[88] It has been marketed as Kerr's Pulp Canal Sealer (Sybron Endo/Kerr, Orange, CA, U.S.A.). It's original formula had a rapid setting time, especially in hot and humid environments.[88] Subsequently, another formula became available under the name of Pulp Canal Sealer EWT (Extended Working Time) (Sybron Endo/Kerr, Orange, CA, U.S.A.) that provides a longer setting time.

Tubuli-Seal (Sybron Endo/Kerr, Orange, CA, U.S.A.) is a two-paste system contained in two separate tubes. It has been developed as a nonstaining alternative to the silver-containing Pulp Canal Sealer. Barium sulfate is used for radiopacity. Tubuli-Seal is easy to mix but has the disadvantage of rapid setting time.[88]

Resin-based Sealers

AH 26 (Dentsply Detrey, Konstanz, Germany) is a bisphenol epoxy resin sealer that, for polymerization, uses hexamethylenetetramine (methenamine). It is marketed as a liquid-powder system. It was claimed that this formula might release small amounts of formaldehyde during the setting phase.[86]

AH Plus or *ThermaSeal Plus* (Dentsply Detrey, Konstanz, Germany) is also an epoxy resin sealer that has been formulated to avoid the formation of formaldehyde.[89] It is marketed as a two-paste system (unlike AH 26). AH Plus has a working time of 4 hours and a setting time of 8 hours.

A resin sealer called "*Russian Red,*"[90] has been used as a permanent filling material in some countries in Eastern Europe, Asia, and the Pacific Rim. This is a resourcin-formaldehyde cement, a type of phenol-formaldehyde or Bakelite resin.[91] It can be toxic and has no efficient solvent. Its removal from the root canal for retreatment purposes can be extremely difficult; sometimes impossible.

Therefore, the use of this sealer and similar products is not recommended.[92]

Paraformaldehyde Sealers

The use of sealers/cements containing paraformaldehyde (just as, until a short time ago, were N-2, Rocanal, and Endomethasone) is not encouraged, especially if used as the sole canal filling material. N-2, like all other cements containing paraformaldehyde, is highly cytotoxic.[93–106] In 1974, the Council on Dental Therapeutics of the American Dental Association[107] classified N-2 as an "*unacceptable*" preparation, in light of the scientific studies indicating its potentially hazardous effects.[99,108–110]

Medicated Sealers

There are numerous medicated sealers in the market. Many contain iodoform[111] or calcium hydroxyde.[88] There is no biological justification for their use as permanent materials to seal root canals and they haven't shown any clinical advantage.

Mineral Trioxide Aggregate

Mineral Trioxide Aggregate (MTA) (ProRoot MTA, Dentsply Tulsa Dental Products, Tulsa, Oklahoma) (Figure 22-24) appears to possess many characteristics of an ideal cement and has been used for mechanical and carious pulp exposures,[112,113] iatrogenic perforations,[114–117] open apices,[118–120] resorbed apices, and as a root-end filling.[121–123]

MTA provides a good seal and is capable of stimulating healing and osteogenesis. It is a powder consisting of tricalcium oxide, silicate oxide, bismute oxide, tricalcium silicate, and tricalcium aluminate. Hydration of the powder results in formation of a colloidal gel with a pH of 12.5, that solidifies to a hard solid structure in approximately three to four hours.[112] MTA is biocompatible,[124–126] causes no cell damage to fibroblasts and osteoblasts, and can stimulate formation of dentin bridges when used for direct pulp capping. It has also been suggested that its sealing ability and biocompatibility are superior to many other commonly used materials.[121,127–131]

An important characteristic that distinguishes MTA from other materials used to date in endodontics is its hydrophilic properties. It sets in the presence of moisture. Moisture can affect the physical properties and sealing ability of restorative materials.[112] However, the presence or absence or blood doesn't seem to affect the sealing ability of the material.[129] In fact, MTA sets well only in the presence of moisture.[112]

Due to its sealing ability and biocompatibility, the use of MTA can be advantageous when more conventional core filling materials cannot be safely used (e.g., extensive root resorption or apical foramen that is too large) (Figure 22-25).

FIGURE 22-24 ***A***. Gray-color ProRoot MTA. ***B***. White-color ProRoot MTA (Dentsply Tulsa Dental Specialty, Tulsa, Oklahoma, U.S.A.).

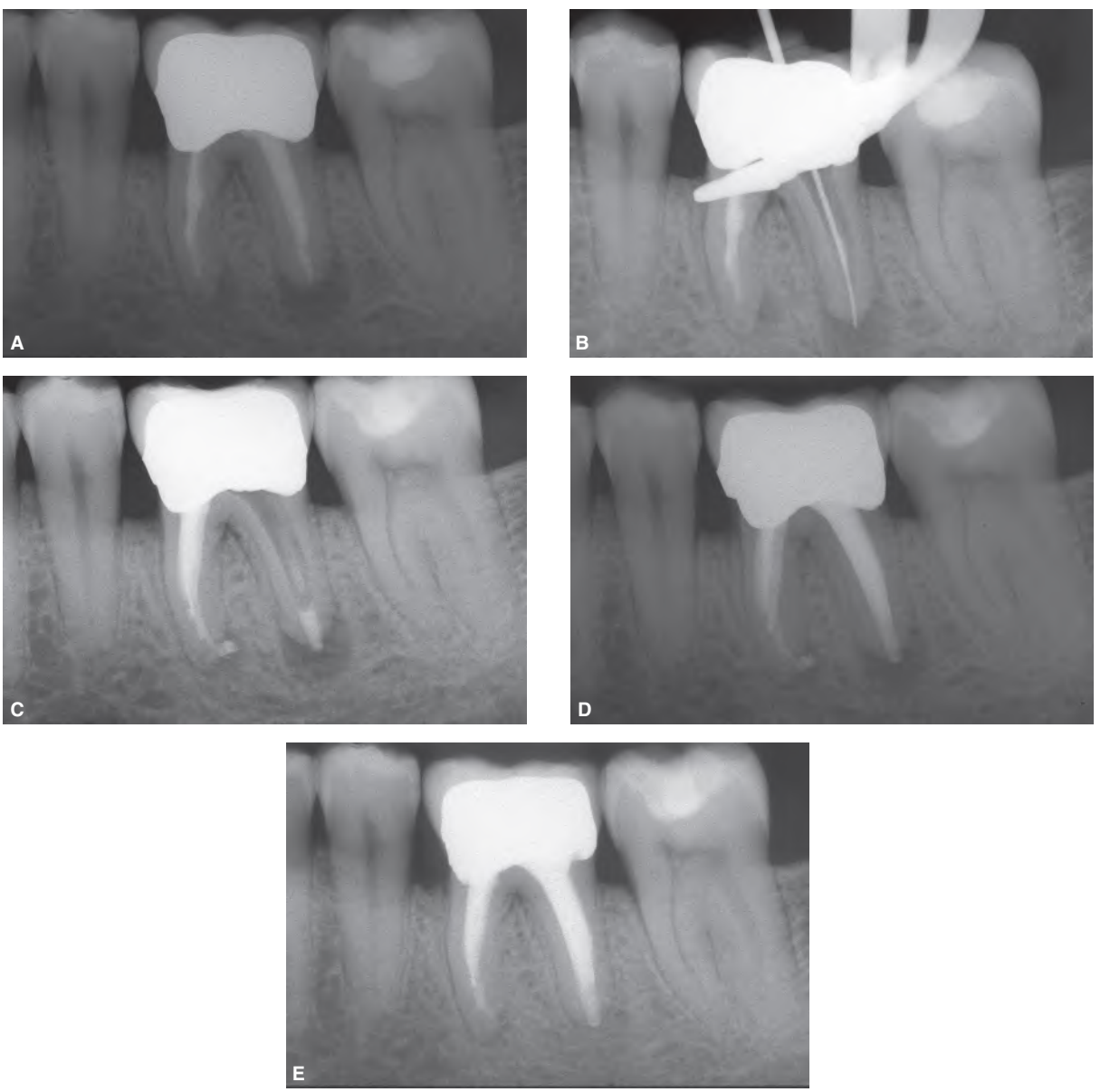

FIGURE 22-25 Endodontic retreatment in a mandibular left first molar using MTA. ***A***. Preoperative radiograph. ***B***. Working length of the distal canal. The size of the apical foramen is larger than a #50 instrument. ***C***. Three millimeters of gray MTA were placed at the apical end. ***D***. Postoperative radiograph showing the apical 3 mm of the root canal obturated with MTA. ***E***. One-year recall radiograph showing healing of the lesion.

SOLID MATERIALS

In the past, silver cones were among the most commonly used solid materials for root canal obturation. They were not the only solid materials used. Techniques of canal obturation that require the use of reamers, files, or endodontic probes intentionally separated in the root canal have been described. Silver points are not recommended for use in modern endodontic practice. They are briefly decribed here only for historical purposes and because clinicians may still encounter them in some retreatment cases.

Jasper[132] introduced the use of silver points to dentistry in 1933. He proposed that they be used to obturate very narrow and tortuous canals. It was conceived that, in such cases, a more rigid material than gutta-percha was needed to be delivered to the desired working length.

The ease of handling silver points, coupled with less canal enlargement, led to the misuse and abuse of silver points causing numerous treatment failures. This was mainly due to their introduction in canals that had not been sufficiently cleaned and shaped. Radiographically, however, they gave the appearance of a perfectly filled canal due to their high radiopacity. Nevertheless, silver cones couldn't fill or be compacted into all spaces within the root canal system. Leakage was also a major problem. It caused corrosion and production of cytotoxic silver salts.[133–137]

The main disadvantages of silver points are:

1. They do not adapt well to the inner canal anatomy. The canal will, therefore, remain inadequately obturated (Figure 22-26) or mostly filled with sealer.
2. The canal often has an irregular shape; it is elliptical, eccentric, and the apical foramen doesn't have a round cross-section like the silver point.[59,138] As a result, the silver point will only touch the walls in two points, will not be able to seal an elliptical foramen, and consequently filling mishaps may occur (Figure 22-27).
3. If the silver point is positioned at the apical foramen, or protruding beyond the apex,[135] (Figure 22-28) once the surrounding sealer has been resorbed, metallic corrosion sets in as a result of oxidation.[134,139] This leads to the formation of cytotoxic byproducts containing sulfur and chloride.[133] Corrosion results from the contact of the silver point with tissue fluids that penetrate from the apical foramen or from a deficient coronal restoration. It can also lead to disintegration of the metal.[140]

If the silver point has been sectioned at the canal orifice (this was used in several silver points techniques) or, worse yet, within the root canal itself, its removal can be very difficult, if not impossible.

The only advantage of silver points was their rigidity allowing their easy introduction into narrow and curved root canals. However, with the advent of advanced techniques that allow for successful obturation of smaller canals with gutta-percha, the use of silver points has declined substantially. Today, silver points are considered obsolete. There is no indication or justification for their use, in as much as they have been superceded by semi-solid materials, that offer a much higher versatility and predictable clinical results.[141]

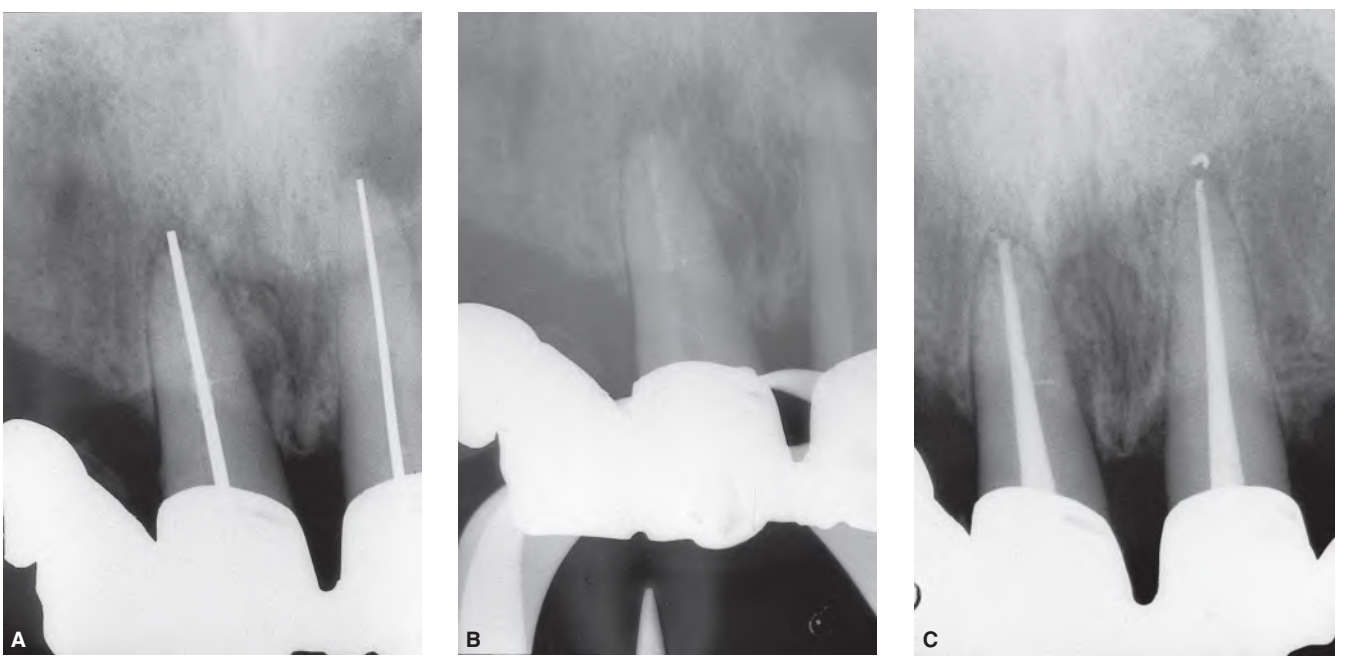

FIGURE 22-26 ***A***. A maxillary right central incisor obturated with a silver cone presenting periradicular radiolucencies. Note sealer extruding via a small lateral canal midway along the root. ***B***. The silver cone was removed. ***C***. Three years later, the periapical lesions associated with the two central incisors have resolved. However, the lateral lesion associated with the right central incisor has not completely resolved. It is possible that some infected material is still present within the lateral canal.

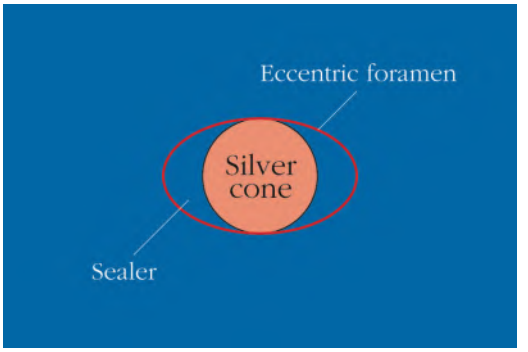

FIGURE 22-27 Schematic illustration of the relationship between a silver cone and an elliptical apical foramen.

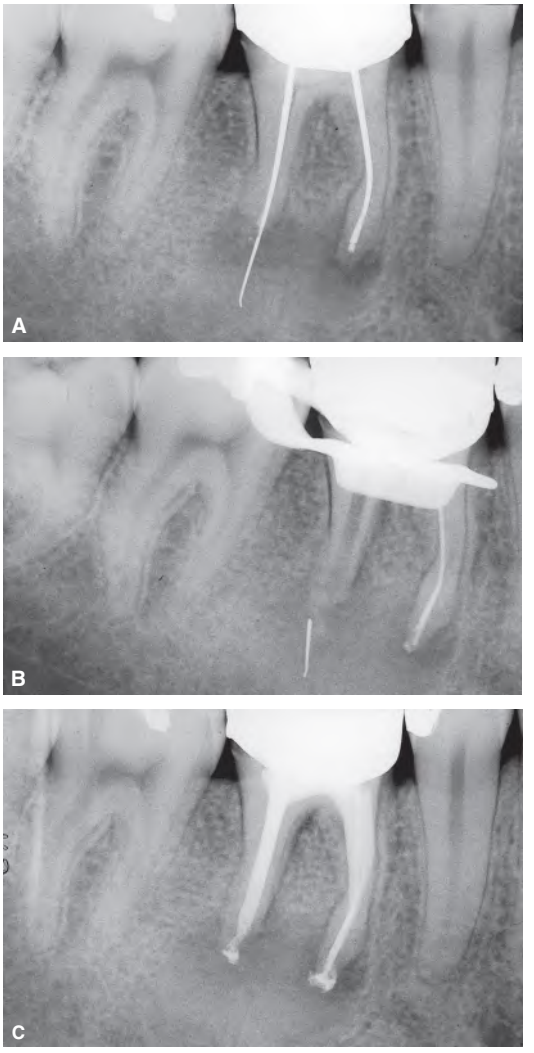

FIGURE 22-28 A silver cone is grossly extruding beyond the apex. It has become corroded, causing a separation of the cone into two segments at the level of the apical foramen. ***A***. Preoperative radiograph. ***B***. The silver cone has been removed from the distal canal, but the segment beyond the apex has remained. The patient developed an abscess that was left to drain from the tooth. ***C***. One week postoperative radiograph after obturation. The silver cone fragment was "drained" externally, together with the purulent exudate.

SEMI-SOLID MATERIALS

The semi-solid material most widely used in endodontics today is gutta-percha. It is derived from a rubber base obtained from certain tropical plants (from Malaysia, Borneo, Indonesia, South Africa, and Brazil) belonging to the genera Sapotaceae.[142] It has been successfully used in endodontics for over 100 years. While not perfect,[143,144] today, gutta-percha is still the material of choice for most canal obturation cases. Until a better material is found, the only issue is how to make better use of it.[40]

The composition of commercially available gutta-percha points consists of 18% to 22% gutta-percha, 59% to 76% zinc oxide, 1% to 4% waxes and resins, and 1% to 18% metal sulfates.[86] Although gutta-percha is not the major ingredient, it serves as the matrix. Zinc oxide acts as the filler, whereas the waxes and resins serve as plasticizers. Metal sulfates, such as barium sulfate, provide the radiopacity to identify the material radiographically. [86]

The advantages of gutta-percha are:

1. It adapts to the canal walls because of its compactability. In fact, gutta-percha is neither molecularly condensable nor compressible. However, once softened by heat, it can be compacted against the canal walls and collapse any voids present in the commercial product.[145]
2. Once set, gutta-percha is stable in size. It shrinks only when chemically softened (e.g., with chloroform) following evaporation of the solvent, or if it is physically softened (e.g., by heat), during the cooling phase. For these reasons, chemical softening of gutta-percha is to be avoided, since it may create voids. Softening by physical means must be accompanied by compaction of the material to compensate for the volumetric changes that occur during the cooling phase.
3. Although constituted primarily of zinc oxide, gutta-percha has a very low solubility rate. Nonetheless, there are some cases where gutta-percha, that had inadvertently been forced beyond the apex, was partially resorbed over the course of several years (Figure 22-15).
4. It is well tolerated by tissues.[57] Of all materials used in dentistry, gutta-percha is perhaps the most inert.[138]
5. It may have certain (weak) bacteriostatic activity, attributed to zinc oxide.
6. It is semi-solid when introduced in the canal. This allows easy manipulability. It becomes malleable if heated, so that it may assume any shape when compacted with appropriate instruments. This allows a three-dimensional filling of all canal spaces, both apically and laterally.
7. It is radiopaque because of its sulfate content (usually barium sulfate) and thus easily recognizable radiographically.
8. It can be easily disinfected. Immersion in 5%–6% sodium hypochlorite for 60 seconds is sufficient to eliminate both gram-positive and gram-negative microorganisms and even the most resistant *Bacillus subtilis* spores.[146,147]

9. If necessary, it can be easily removed from the root canal when endodontic retreatment is indicated. For more details, see Chapter 23, "Endodontic orthograde retreatment and management of mishaps." Solvents such as chloroform, chlorothene, eucalyptus, and rectified white turpentine have been used.[148–150]
10. Gutta-percha is a poor heat conductor. This implies optimal control of its plasticity in its most apical portion, when heated.
11. Once introduced into the root canal and heated, gutta-percha expands. This characteristic helps to ensure a tighter seal. As mentioned previously, gutta-percha shrinks during the cooling phase; thus, to compensate for thermal shrinkage, any technique requiring heating must also include a compaction phase.[151] In a study on the thermomechanical properties of gutta-percha, Schilder et al.[145] found that under clinical conditions, compaction, not compression, occurs. The reduction in volume that takes place during mechanical compaction of gutta-percha is due to the consolidation and collapse of internal voids in the material.

Disadvantges of gutta-percha points are:

1. Lack of sufficient rigidity especially in small sizes; it cannot always be pushed beyond a ledge that might be present.
2. It adheres to the dentin walls without establishing any bonds. To rectify this shortcoming, one must always use a sealer.

Gutta-percha points should be stored in a cool location with low humidity, otherwise they become brittle with time.

Gutta-percha is available commercially in the form of standardized ISO (International Standards Organization) sizes and in the so called "non-standardized points." Standardized points follow the same ISO numbers of root canal instruments. They are available in numbers 25 to 140, and their apical diameter and taper (0.02) correspond to those of the instrument of the same number. These points are indicated for the lateral condensation technique where the master cone is selected based on the last instrument used. In such cases, cone standartization is very useful (Figure 22-29).

Non-standardized cones, on the other hand, are much more conical and pointed. Rather than being distinguished by number, they are distinguished by size: extra-fine, fine-fine, fine, medium fine, fine-medium, medium, medium-large, large, and extra-large. These cones closely match the final shape and size of the instrumented root canal and are indicated for vertical compaction techniques, since their greater conicity allows for better adaptation to the tapered preparation (Figure 22-30).

Recently, new rotary shaping systems have been introduced, each having their own gutta-percha points to match the shape created by the particular rotary file system used. This is valid for many rotary or reciprocating NiTi File systems, like GTX Rotary, ProTaper Universal, WaveOne, ProTaper Next and similar ones (Figure 22-31).

FIGURE 22-29 ISO-standardized gutta-percha cones used with the lateral condensation technique.

FIGURE 22-30 Nonstandardized gutta-percha cones used with vertical condensation of warm gutta-percha.

With the thermoplasticized injectable techniques, the gutta-percha is produced in a pellet form that is loaded into a heating device like Obtura (Obtura Spartan, Earth City, Missouri) B&L Beta 2 (B&L Bio Tech, Seoul, Korea) (Figure 22-32), or as prepackaged cannulas or cartriges like Ultrafil (Coltene/Whaledent, Cuyahoga Falls, Ohio), Calamus (Dentsply Tulsa Dental Specialties, Tulsa Oklahoma), Elements, (SybronEndo, Orange, California) (Figure 22-33). Carrier-based gutta-percha products have gutta-percha around a plastic carrier (Thermafil, GT Obturators; Dentsply Tulsa Dental Specialties, Tulsa, Oklahoma) or around a cross linked gutta-percha carrier (GuttaCore; Dentsply Tulsa Dental).

The use of medicated gutta-percha cones containing iodoform,[111,152] calcium hydroxyde,[153] or chlorhexidine[154] is not encouraged and is not discussed in this chapter.

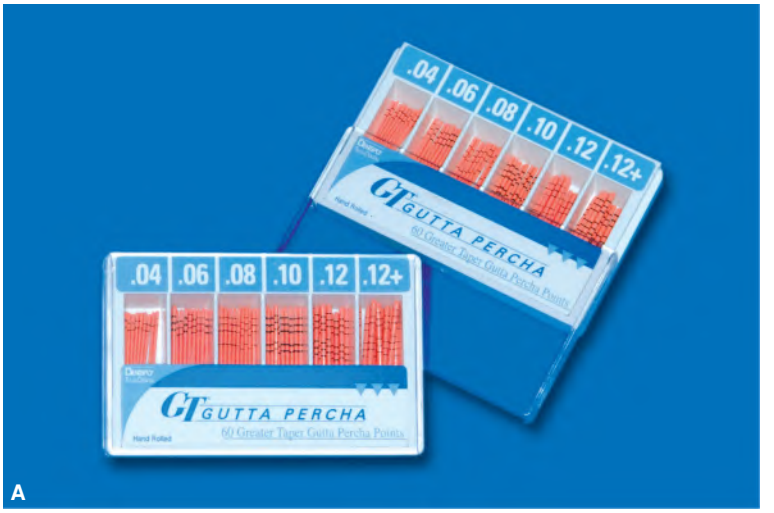

FIGURE 22-31 ***A***. GT gutta-percha cones used with vertical compaction of warm gutta-percha and with the single wave condensation technique. Cones have a standardized taper, corresponding to the GT rotary files. ***B***. Gutta-percha cones, used after shaping the root canal with rotary systems, such as ProTaper Universal and ProTaper Next, or with reciprocating systems such as WaveOne.

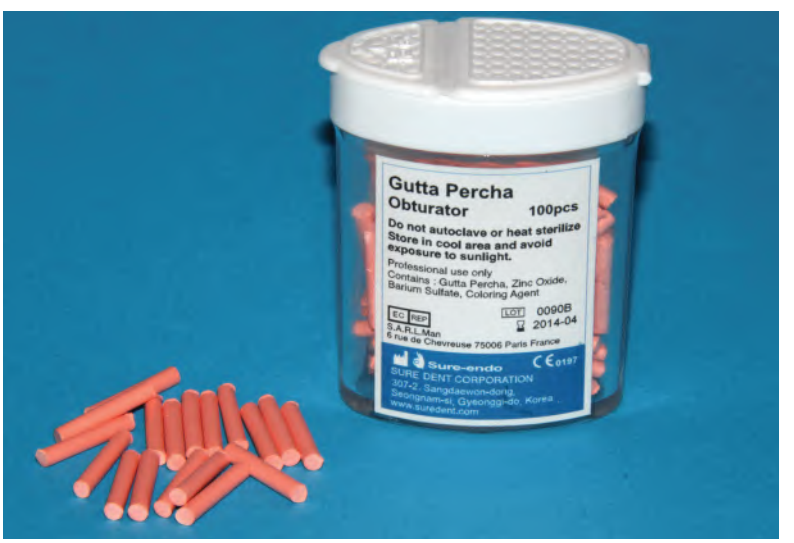

FIGURE 22-32 Gutta-percha pellets for the Beta 2 B&L (B&L Bio Tech, Seoul, Korea).

FIGURE 22-33 ***A,B***. Gutta-percha cartriges for the Calamus unit (Dentsply Tulsa Dental Specialties, Tulsa, Oklahoma, U.S.A.).

TECHNIQUES FOR ROOT CANAL OBTURATION WITH GUTTA-PERCHA

The most common obturation techniques using gutta-percha are ***lateral*** and ***vertical*** compaction methods, surrounding which there has been considerable debate for years. However, both "lateralists"[155] and "verticalists"[156] consider this debate artificial since it is physically impossible to condense either laterally or vertically alone[155] and on the other hand the softened gutta-percha mass, that is being compacted vertically into the conically shaped canal preparation, automatically assumes a lateral component of force. This follows routine laws of physics and requires no lateral direction of the instrument on the part of the operator.[156] One can discuss which technique is superior, but should not do so in terms of "lateral" or "vertical" forces, but rather of "cold compaction" versus "warm compaction."

VERTICAL COMPACTION OF WARM GUTTA-PERCHA

Many endodontists prefer to be familiar with several techniques of canal obturation, since each technique is indicated in certain situations.[21,155] Weine[138] discussed the various techniques of gutta-percha use, emphasizing that vertical compaction is to be preferred in certain well-defined cases, such as when the fitting of a conventional master cone to the apical portion of a canal is impossible, or when there is a ledge formation, perforation, or unusual canal curvatures, internal resorptions, or large lateral canals. The vertical compaction method then affords the operator a chance to achieve success in a case that might fail if any other kind of treatment were used.

The vertical compaction technique of warm gutta-percha, conceived and described by Schilder[40] in 1967, combines a reasonable ease of execution with maximal efficacy in obturating both simple and more complex canals.[157] Gutta-percha softened by heat is vertically compacted into the conically shaped canal preparation.

Armamentarium

The instruments used for compacting the gutta-percha in this technique are similar to amalgam compactors. However, they differ from them by being longer and narrower. These instruments are referred to as compactors or "pluggers" (Figure 22-34). Serrations at five millimeter intervals help to assess the working depth each instrument specifically made to prevent it coming into contact with the dentin. Particularly useful are the double ended pluggers: one end is in NiTi, smaller and flexible, and the other end is stainless steel, larger and rigid, like the ones manufactured by Obtura Spartan (0.80, 1.00, and 1.20 mm in the stainless steel end and 0.40, 0.50, and 0.60 in the NiTi end) and by B&L (0.70, 0.80, 1.00, and 1.20 mm in the stainless steel end and 0.35, 0.40, 0.50, and 0.60 in the NiTi end) (Figure 22-35).

The other instrument that this technique requires is the "heat carrier," that is used to deliver heat to the gutta-percha cone in the root canal. Several types of heat carriers are available, like the Touch 'n Heat (SybronEndo), the Elements (SybronEndo), the Calamus (Dentsply Tulsa Dental), or the new cordless Alpha 2 from B&L (Figure 22-36). These instruments are provided with tips of different tip diameter and different taper.

Gutta-percha Cones

The gutta-percha cones indicated for this technique are tapered. Although they have the same chemical composition

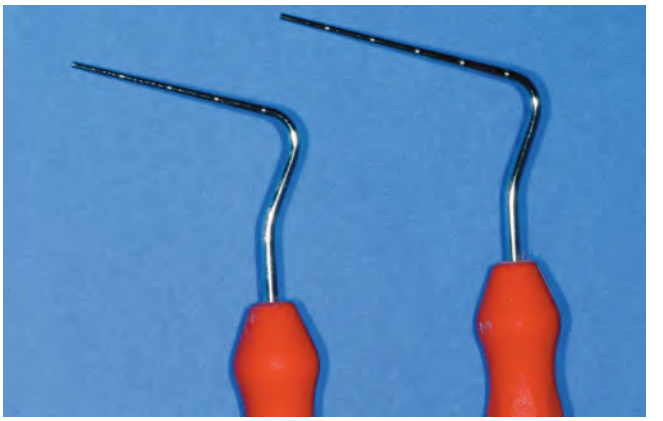

FIGURE 22-34 A plugger of the "Posterior" series, for teeth of 25 mm or less (left). A plugger of the "Anterior" series, for teeth of 30 mm (right) (Dentsply Maillefer, Ballaigues, Switzerland).

as the standardized ones, they are more conical and thus provide a larger amount of gutta-percha on which to exert vertical force with the working surface of the pluggers. In the original Schilder technique the sizes most commonly used were "fine," "fine-medium," "medium," and "medium large" (Figure 22-30). Today, many shaping systems are available each having gutta-percha points that correspond to its shaping files (Figure 22-31).

FIGURE 22-35 The three most frequently used pluggers from B&L (B&L Bio Tech, Seoul, Korea).

Sealer

A minimal amount of sealer is necessary to ensure better adaptation of gutta-percha to the canal walls. Therefore, the ideal sealer for this technique is one that can be spread onto the canal walls as a microfilm of few microns in thickness.[156,158] Many commercially available endodontic sealers can be used, as long as they are inert, biocompatible (or at least well tolerated by the tissues), nonshrinking, and nonresorbable.

Weiner and Schilder[159] found that there was a correlation between the sealer's setting time, its resorbability, and its

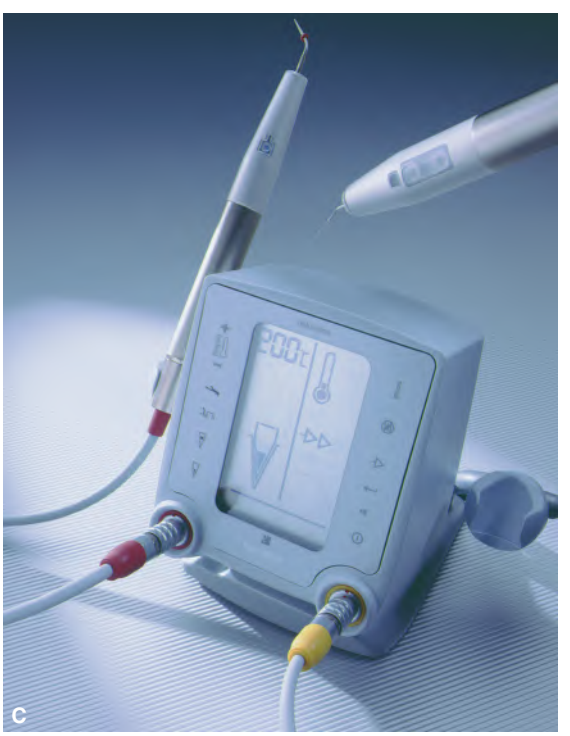

FIGURE 22-36 ***A***. The "Touch 'n Heat" carrier (SybronEndo). ***B***. The "Alpha 2" heat carrier (B&L). ***C***. The Elements Obturation Unit (SybronEndo). ***D***. The Calamus (Dentsply/Tulsa Dental).

shrinkability; the longer the setting time, the easier the sealer will shrink and be resorbed. Sealers that appear to be the least affected are the Pulp Canal Sealer (Kerr), that has a relatively short setting time (Figure 22-37A), and the Pulp Canal Sealer E.W.T. (Kerr), that allows an easier manipulation due to its extended working time (Figure 22-37B). The composition of this sealer, in Rickert's 1931 formulation, is:[160]

Powder (%)	
Silver	24.74
Zinc oxide	34
Thymol iodide	10.55
Oleoresin	30.71
Liquid (%)	
Eugenol	78
Balsam of Canada	22

Phases of obturation

Cone Fit

If feathered gutta-percha cones are used, they must be slightly shortened (Figure 22-38) up to the size of the apical foramen, and a radiograph taken to check the cone fit. The gutta-percha point should reach the working length and show to have retention inside the root canal (tug-back). After the appropriate cone fit has been confirmed on the radiograph, the tip of the cone is cut so as to be slightly short with respect to the preparation (Figure 22-39).

In accordance with the second mechanical objective of shaping descibed by Schilder,[161] the apical cross-sectional diameters of the prepared root canal must diminish coronoapically. As a consequence, the cross sectional diameter of the shortened gutta-percha point is bigger than the cross

FIGURE 22-37 The Pulp Canal Sealer ***A***. and the Pulp Canal Sealer EWT (Kerr Corporation, Orange, California, U.S.A.) ***B***. are best suited for the Schilder technique.

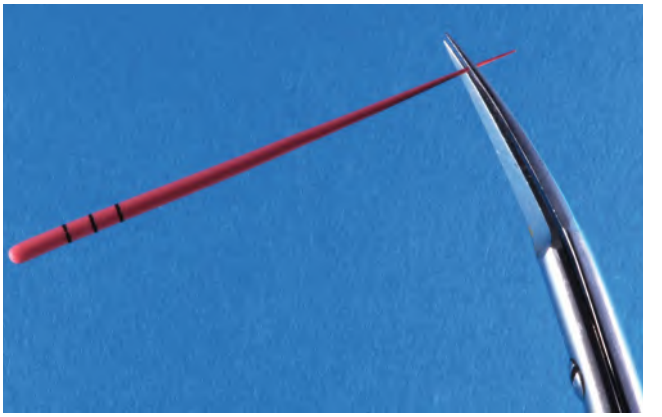

FIGURE 22-38 The feathered gutta-percha cone just removed from its wrapper must always be blunted with a pair of scissors.

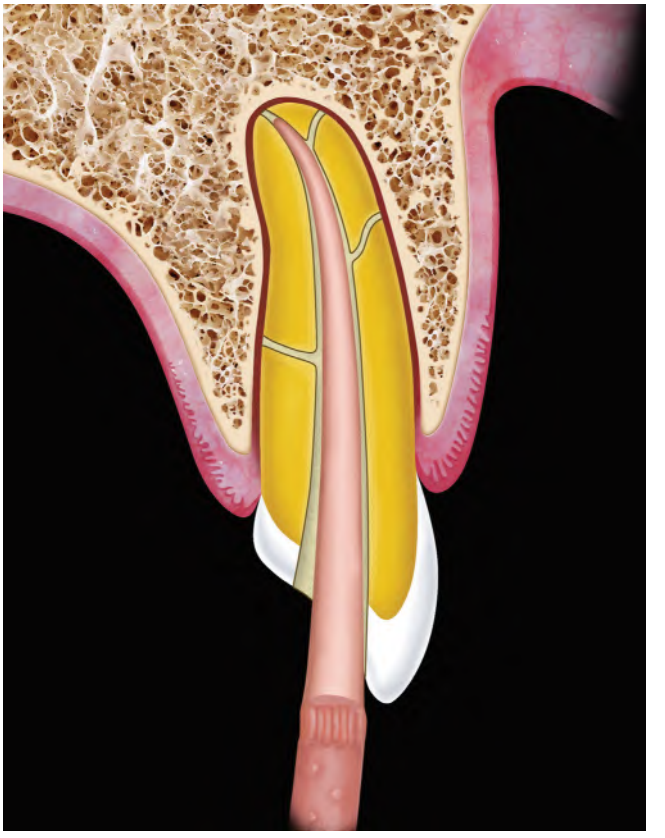

FIGURE 22-39 The cone must be slightly shorter than the preparation. A notch is made by cotton pliers at the level of the reference point.

sectional diameter of the foramen. When one begins to compact and push the cone vertically, the cone moves apically, and its tip will become engaged in progressively smaller portions of canal. This will lead to deformation of the cone ensuring a good seal and a good apical control of the obturating material. On the other hand, if the gutta-percha cones are not shortened, they will protrude beyond the apical foramen as a result of the vertical force until its cross-section is almost equal to, if not greater than, the diameter of the apical foramen.

The tip of the gutta-percha cone is shortened by using a special gauge (Figure 22-40) or small scissors and selectively removing varying amounts of the apical tip until it enters the canal comfortably and binds. This must occur at the working length and the depth is marked on the cone with a notch made by cotton pliers at the reference point of the stop. The fit is initially checked by comparing the length of the cone with that of the last instrument at the terminus of the respective canal (Figure 22-41), followed by a control radiograph.

If the canal was prepared using NiTi instruments systems like GTX Rotary Files, ProTaper Universal, ProTaper Next, WaveOne, and similar ones, the cone fit is much easier and the clinician will choose the cone corresponding to the taper prepared in the root canal (Figures 22-30 and 22-31).

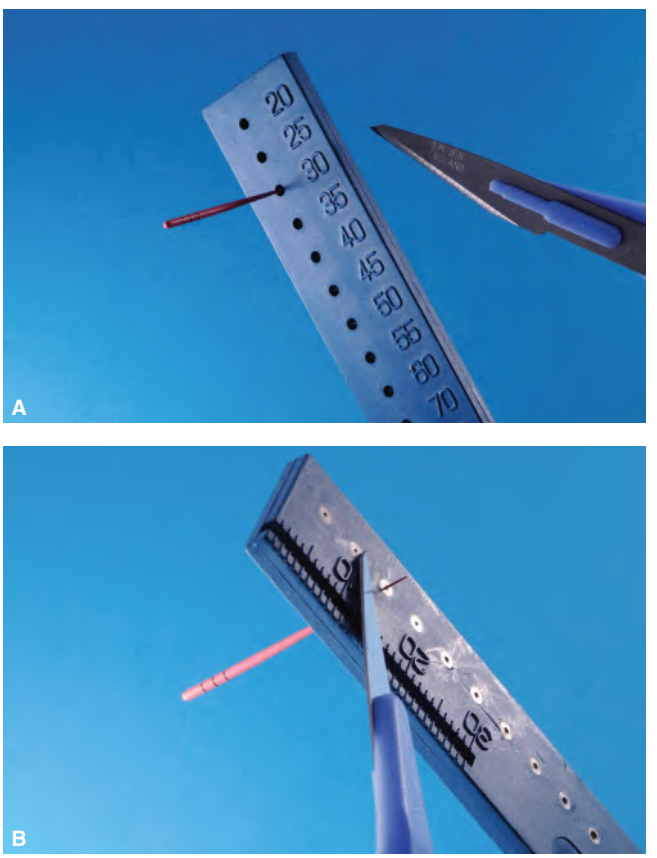

FIGURE 22-40 ***A,B***. The gutta-percha cone is cut off using a special gauge (Dentsply, Maillefer) for gutta-percha points. This gauge is very useful to cut off the gutta-percha cones to the desired diameter.

However, the principles are exactly the same: the cone should have an adequate tug-back, should fit slightly short of the working length, and should then reach the apical foramen during compaction, to assure an adequate apical seal of the root canal system.

Prefitting of Pluggers

The pluggers must be prefitted in the canal to determine the depth to which they can be introduced without touching the dentin walls. The essence of the technique is to capture the maximal cushion of softened gutta-percha and compact it vertically.[162] Starting with a sufficiently wide plugger, that descends in the coronal one third of the canal (e.g., a Maillefer #10 or a blue B&L) and advancing to smaller sizes. If the plugger has been supplied with a rubber stop, the instrument is introduced into the canal and the stop is positioned just before the instrument touches the dentin walls (Figure 22-42A). Next, a second plugger (e.g., #9 or a yellow B&L) is selected, that descends into the middle one third of the canal, again adjusting its rubber stop (Figure 22-42B).

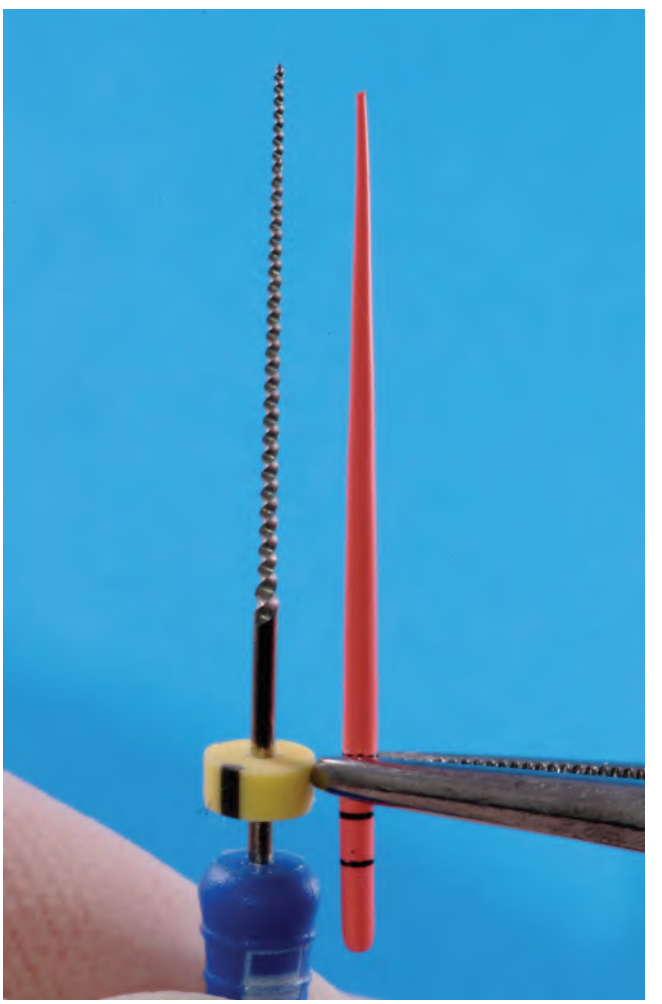

FIGURE 22-41 The relation between the gutta-percha cone and the reference instrument informs the operator how short the cone is relative to the canal preparation.

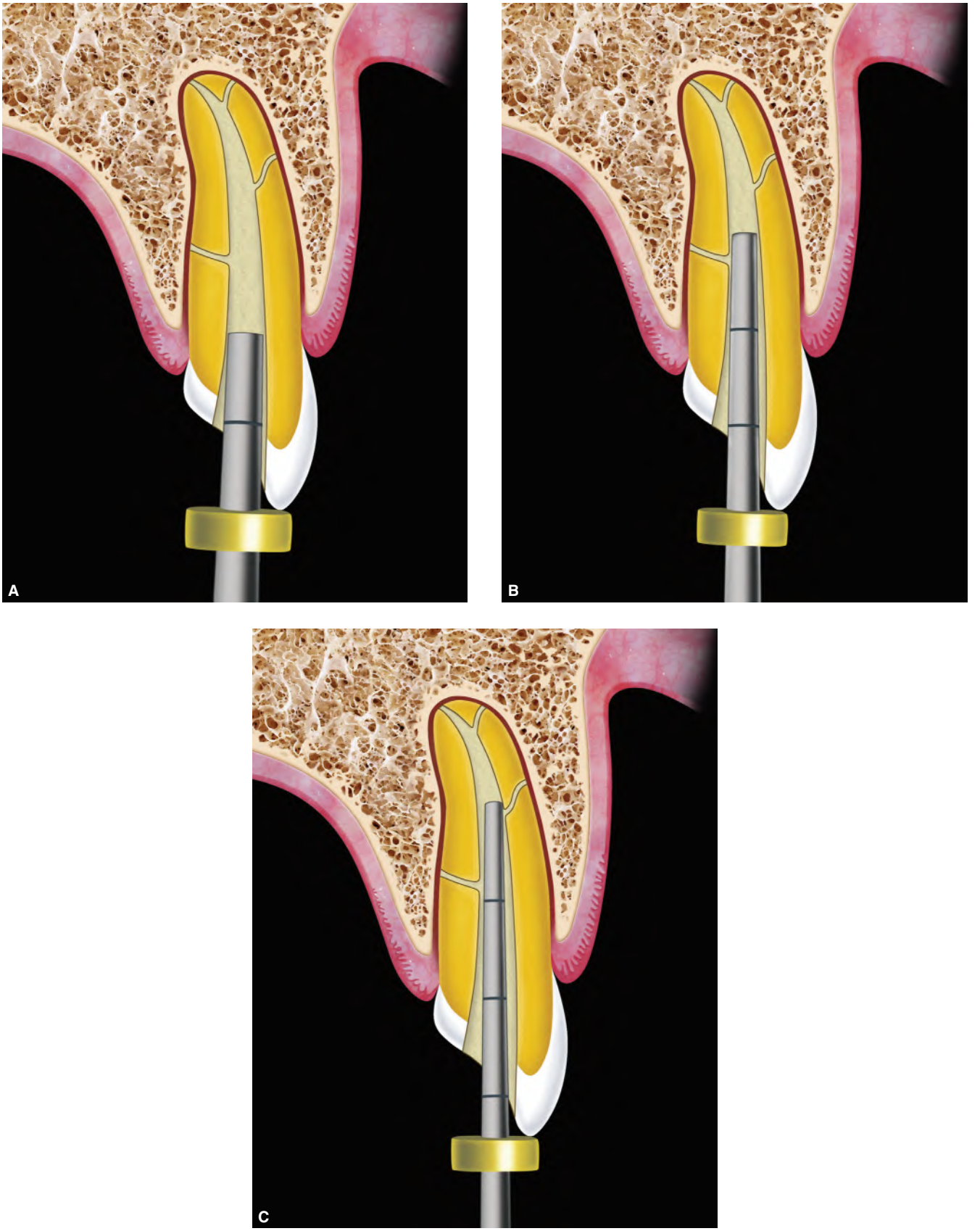

FIGURE 22-42 ***A***. Prefitting the first plugger in the coronal one third. The rubber stop is positioned immediately and before the tip of the instrument makes contact with the dentin walls. ***B***. Prefitting the second plugger in the middle one third. ***C***. Prefitting the smallest plugger. Its working depth is about 5 mm from the apical foramen.

Finally, a third, narrower plugger, that descends into the apical one third is advanced to a point about 5 mm from the apical foramen (e.g., a #8 or 81/2 or a black B&L), and its stop is also adjusted (Figure 22-42C). The pluggers must never touch the dentin walls, since by doing so it will no longer exert any pressure on the gutta-percha for compaction purposes while potentially causing root damage (e.g., crack or fracture) by their wedging action.

Sealer Preparation

A good type of sealer for this technique is the Pulp Canal Sealer E.W.T. (Kerr). Commercially, this sealer is marketed in 2 bottles; one with powder and one with liquid. It should be prepared according to the manufacturer's instructions. A study by Casanova[163] demonstrated that an increase of the powder/liquid ratio diminishes the solubility and resorbability of the sealer without affecting its fluidity and thus its capacity to spread as a microfilm. It is therefore advisable to prepare some extra amount of powder on a glass slab and two drops of liquid (Figure 22-43). At least two-thirds of the powder is dissolved in one drop of liquid to obtain a uniform, creamy mixture. Freshly-prepared sealer must be viscous enough to exhibit at least a 10 cm of "string" (Figure 22-44). The powder remaining on the glass slab can be applied to the tip of the plugger to prevent the heat-softened gutta-percha cone from adhering to it. This reduces the risk of the gutta-percha being pulled out the root canal during the process. The second drop of liquid can be used in case the mixture inadvertently became too thick.

Sealer and Master Cone Placement

The prefitted master cone is soaked in alcohol and dried with a sterile gauze. The entire length of the cone, that will contact dentin in the root canal, is coated with a thin uniform layer of sealer (Figure 22-45). It is then placed in the canal orifice and, with gentle pushing strokes, is made slide slowly and carefully to the maximal depth designated by the notch. The slow, careful introduction of the cone into the canal causes the air and any excess sealer apical to the cone to exit laterally and coronally, without creating any increased pressure or pushing sealer into the periapical area.

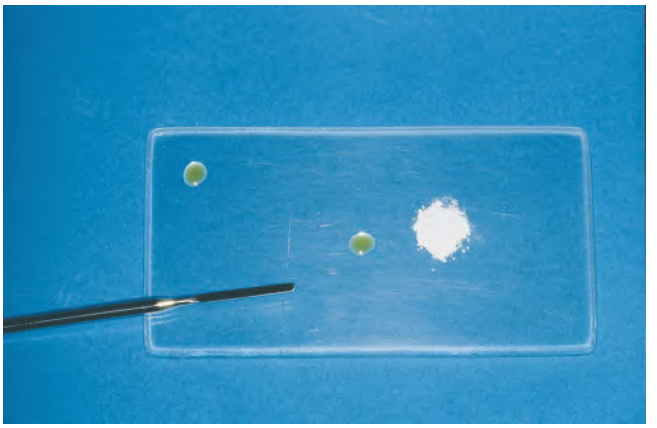

FIGURE 22-43 On a sterile glass slab, a small amount of powder and two drops of liquid (one of which is kept in reserve) are prepared. It is then mixed to achieve the desired consistency.

FIGURE 22-44 Properly prepared sealer must not "drip," but make at least a 8–10 cm "string."

FIGURE 22-45 The entire surface of the cone entering the root canal is coated with a thin layer of sealer.

If the canal has an extreme elliptical shape in its coronal third, an auxiliary cone is introduced alongside the master cone (Figure 22-46). The purpose is not to condense it laterally but to have a greater mass of gutta-percha to compact apically.

Down-packing

The heat-carrier is used to remove the excessive portion of gutta-percha extruding in the pulp chamber (Figure 22-47). Next, the wider prefitted plugger (after having powdered its tip with sealer powder to prevent adhesion to the tacky gutta-percha and sealer) is introduced to initiate the vertical compaction (Figure 22-48). The plugger is then returned, while the heat-carrier is handed back. It is introduced for 3–4 mm into the gutta-percha mass (without touching the dentin walls), where it remains for a fraction of a second (Figure 22-49).[162] The heating element is then deactivated and released after a quick short pause. The instrument is removed, along with some remnants of gutta-percha (Figure 22-50). In this manner, the gutta-percha is heated

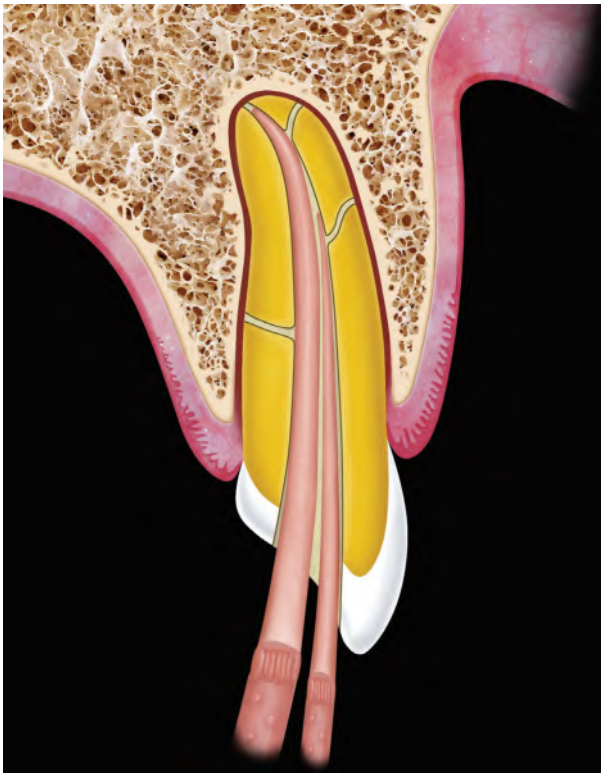

FIGURE 22-46 In very wide or elliptical canals, it is advisable to introduce a supplemental cone(s) alongside the master cone for added bulk of gutta-percha to be compacted apically.

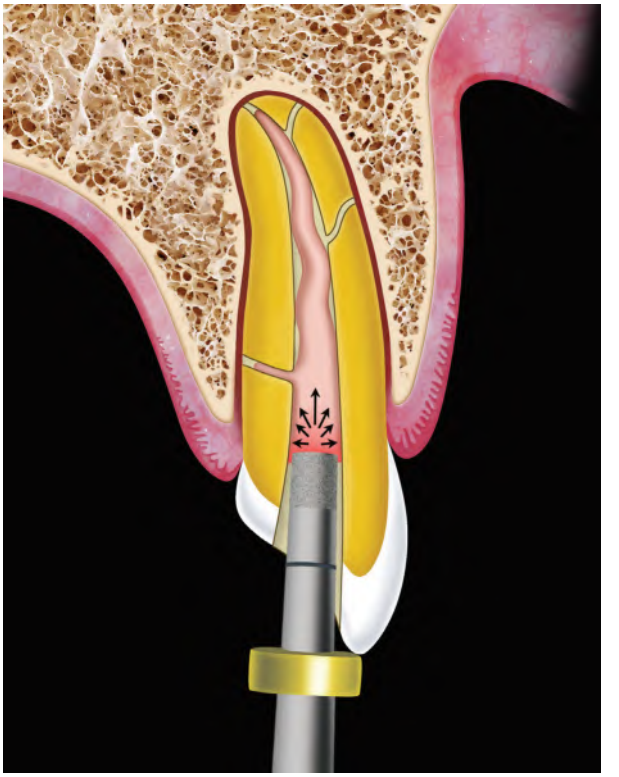

FIGURE 22-48 The first plugger (whose tip is powdered with left-over sealer so the gutta-percha cone does not attach to it) initiates the vertical compaction of the gutta-percha just heated by the preceding heat carrier.

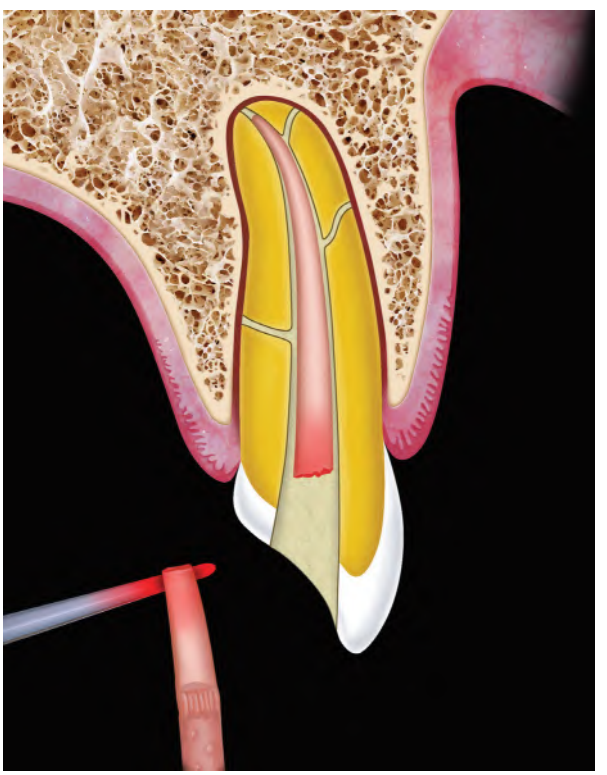

FIGURE 22-47 The gutta-percha that protrudes into the pulp chamber is seared off with a heat carrier.

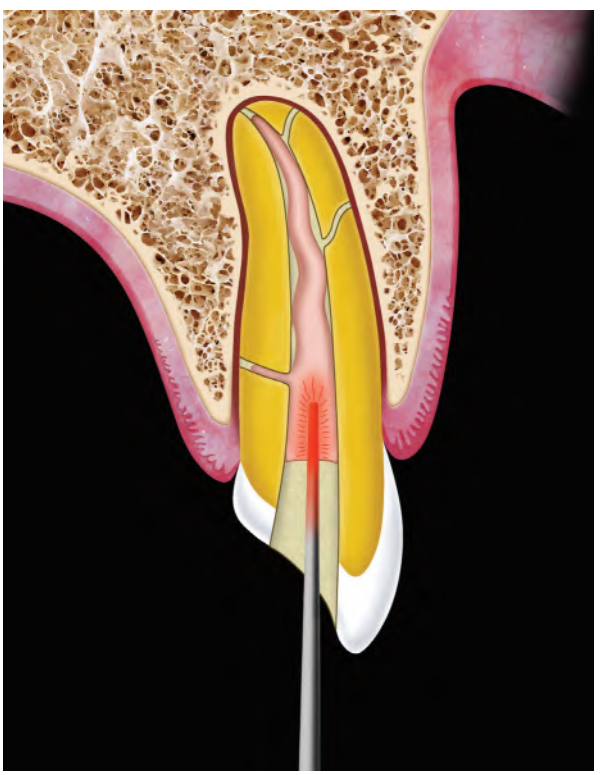

FIGURE 22-49 The heat-carrier is introduced into the center of the gutta-percha cone in the root canal.

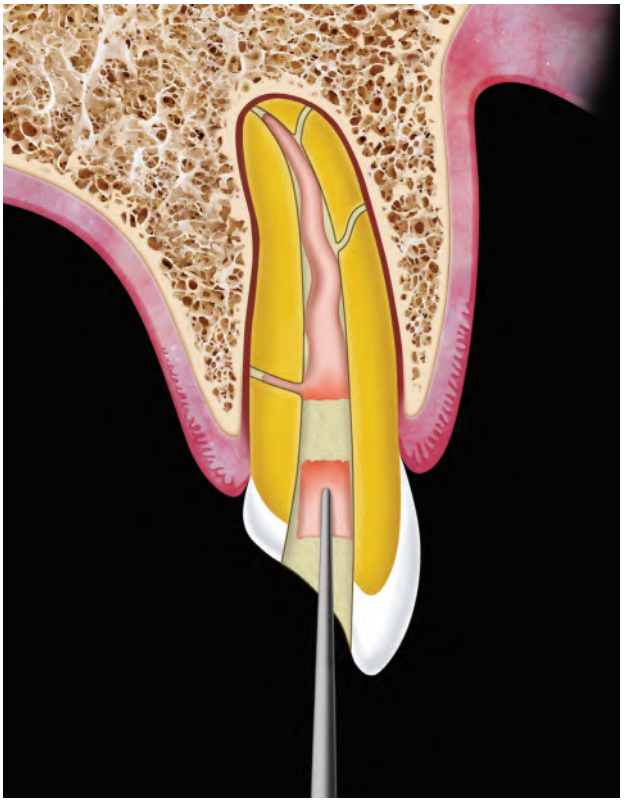

FIGURE 22-50 The heat-carrier is heating the gutta-percha apically to its tip and removes the surrounding material from the canal.

around the heat-carrier and about 3–4 mm apically (no more, because gutta-percha is not a conductor of heat).[164]

When the instrument is withdrawn from the root canal, the attached remnants of gutta-percha are removed and the instrument cleaned (Figure 22-51). Meanwhile, the prefitted plugger is introduced again to compact and push apically the gutta-percha that has just been heated in the root canal (Figure 22-52). Next, the heat carrier is again introduced into the canal to a depth of 3–4 mm and is removed once again covered with remnants of gutta-percha (Figure 22-53) and the same prefitted plugger is used to compact the softened gutta-percha apically as well as laterally. The cyclic use of heat carrier and pluggers continues until the smallest plugger compacts the material at about 5 mm from the working length (Figures 22-54 to 22-57).

The cyclic use of the heat-carrier and pluggers casues the following phenomena:[156]

1. From the very beginning, the force is exerted in a "closed" space where a significant hydraulic effect is produced. Coronally, the plugger of adequate size exerts its pressure against the maximal amount of heat-softened gutta-percha that, compacted, will make immediate contact with the dentin walls, while at the other end the cone, by virtue of its "tug-back," occludes the apical foramen. Thus the force is dissipated in a closed environment and the hydraulic

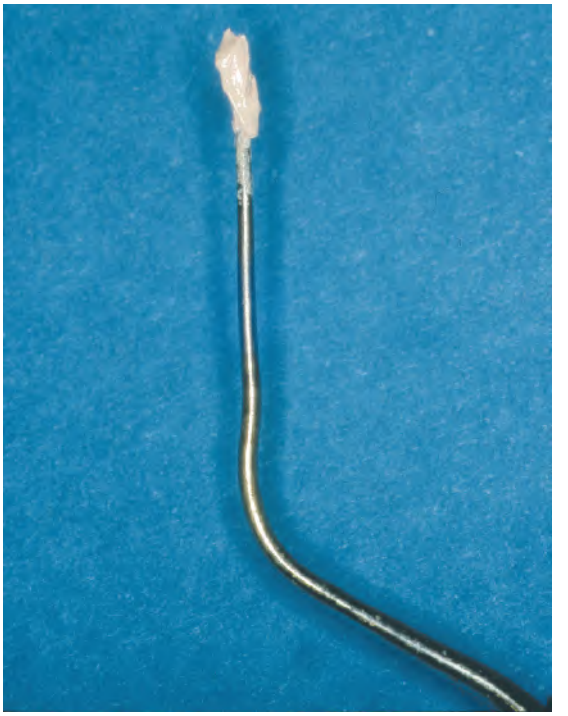

FIGURE 22-51 The heat-carrier is withdrawn, carrying out the cooled gutta-percha around it. This way, the level of the next compaction will be more apical.

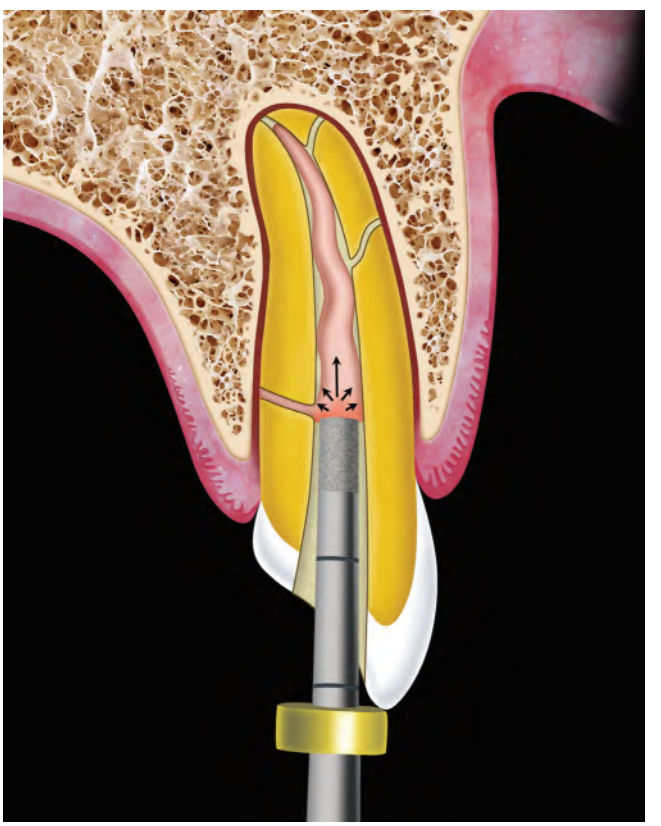

FIGURE 22-52 The first plugger repeats and completes the vertical compaction process in the coronal third of the root. Once the rubber stop is approaching the reference point, it is time to select a plugger of slightly smaller diameter.

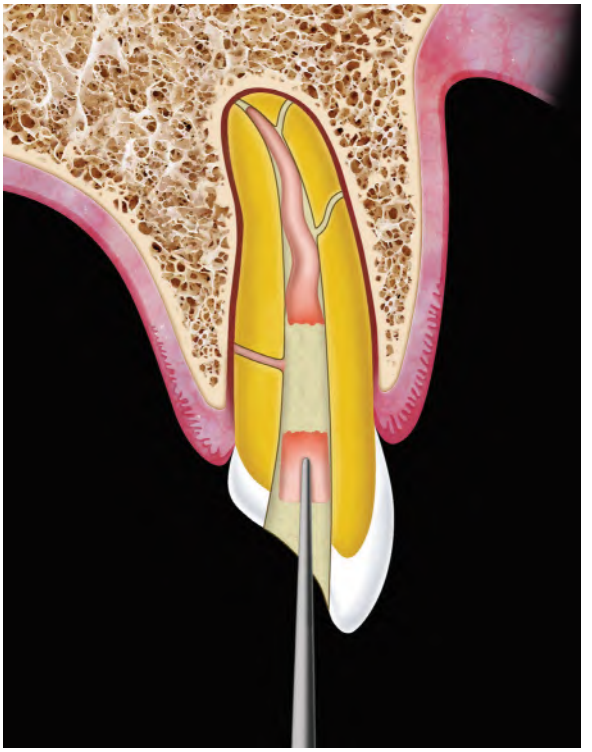

FIGURE 22-53 The heat-carrier is returned and is activated for just one second once it makes contact with the gutta-percha inside the root canal. It is then inserted directly into the central portion of the gutta-percha mass to a depth of 3–4 mm and quickly inactivated and withdrawn while cooling, removing another piece of gutta-percha from the root canal.

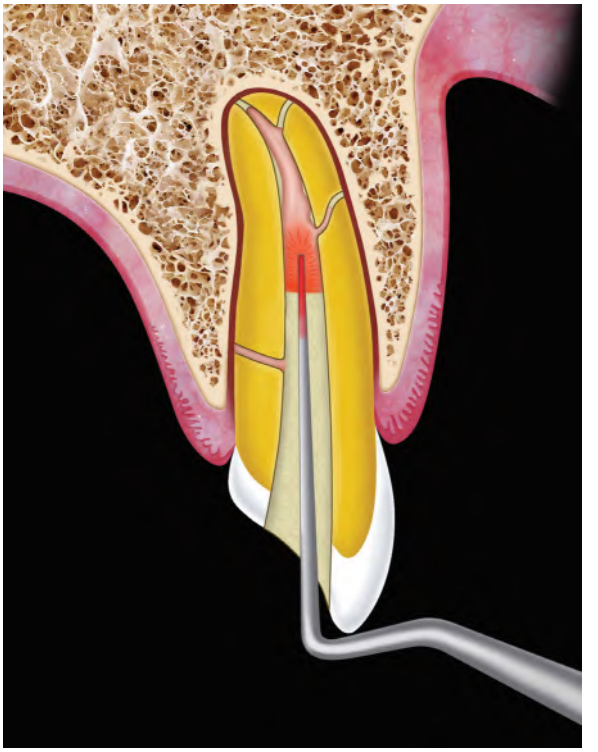

FIGURE 22-55 Reintroduction of the heat-carrier is softening the gutta-percha in the apical one third.

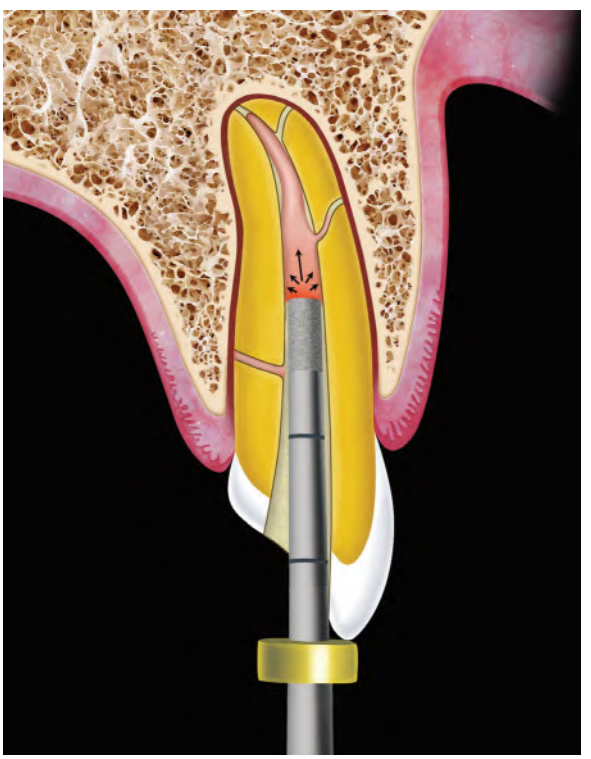

FIGURE 22-54 The second plugger has completed its vertical compaction to the maximal working depth.

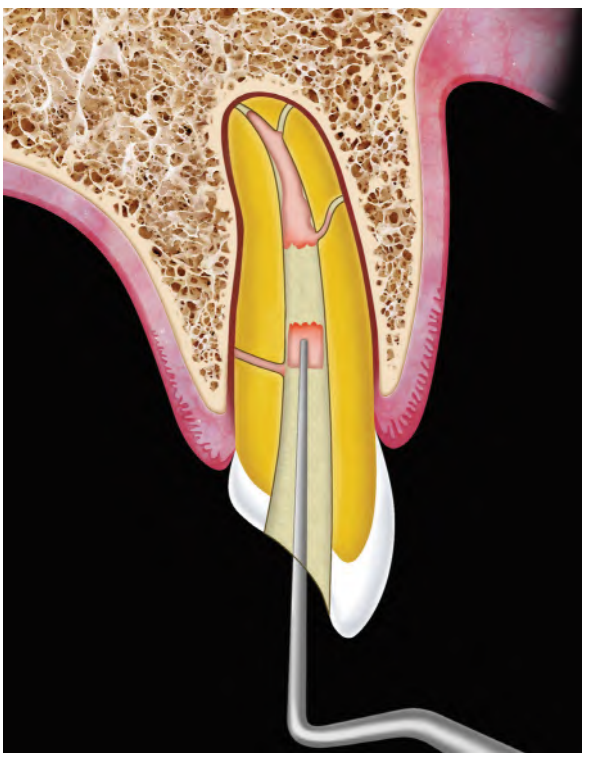

FIGURE 22-56 Another piece of gutta-percha has been removed. Now it is time to use the smallest plugger.

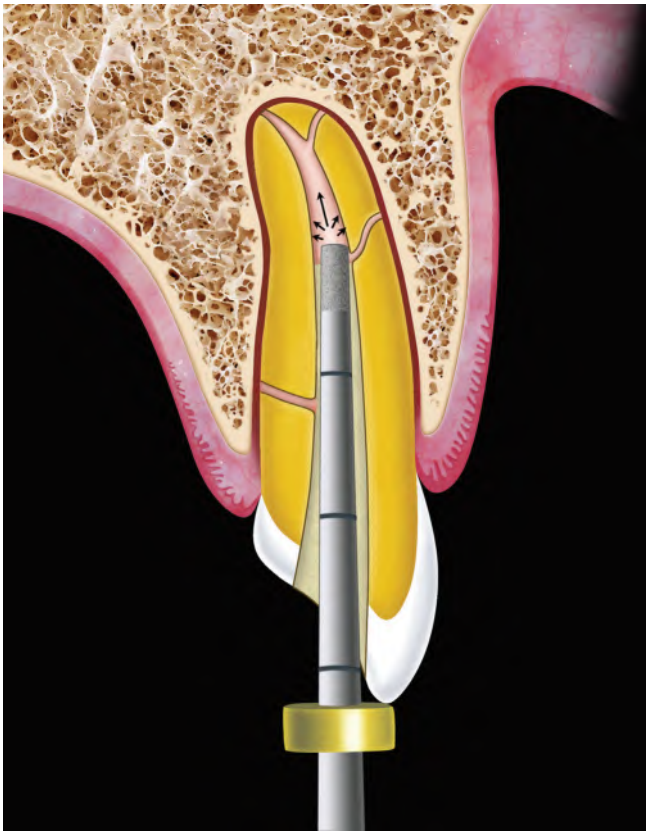

FIGURE 22-57 The smallest plugger has advanced, compacting the gutta-percha mass about 5 mm from the canal terminus.

pressure that develops within causes the sealer and the heat-softened gutta-percha to fill all the available spaces in a true corono-apical "wave of condensation." The more coronal lateral canals are filled first, then gradually as the condensation wave moves apically, those of the middle third of the canal and finally those of the apical third and the ramifications of the delta. This hydraulic pressure is absent in the lateral condensation.

2. The working level of compaction of the gutta-percha moves increasingly apically for two reasons. The first is that the softened gutta-percha mass, which is being compacted verticallyinto the conically shaped canal preparation, automatically assumes a lateral component of forces. This follows routine laws of physics and requires no lateral direction of the instrument on the part of the operator.[156] Second, each introduction of the heat-carrier in the root canal is followed by the removal of a certain amount of gutta-percha that remained attached to the tip of the heating instrument as it begins to cool. Consequently, the wave of condensation and the working level of the pluggers are displaced increasingly apically and it becomes necessary to advance to the plugger of narrower size.
3. The temperature of the apical gutta-percha gradually increases by a few degrees above body temperature. Since gutta-percha is a poor conductor of heat, its temperature will not rise immediately after the first or second application of heat. It will require several applications and even then the temperature will not rise to hazardous levels. A thermocouple study[165] has demonstrated that the apical gutta-percha becomes moldable at a temperature range of 3°C-7°C above body temperature and will not be elevated more than 9°C above the initial temperature.[168] Once the heat-carrier followed by the narrower plugger have descended to a depth of about 5 mm from the apical foramen, thermal increase occurs gradually in concomitance with the various applications of heat (Figure 22-58). If heat is not intentionally added, the gutta-percha returns to body temperature and will resist further movement beyond the terminus of the canal preparation.[165,166] Apical control of the obturation is thus excellent, since the gutta-percha at the apex is never molten.

It is contraindicated to descend further apically with the heat-carrier to heat the gutta-percha 2–3 mm from the apical foramen.[167] Overheating and excessively plasticizing the apical gutta-percha increase the possibility of gutta-percha extruding beyond the terminus of the preparation.[59] Stopping 5–6 mm short is more than sufficient to obtain an effective sealing or "corkage" of the canal preparation and filling of small lateral canals (Figure 22-59) or the apical delta (Figure 22-60) with gutta-percha and/or sealer.

Reverse Filling ("back-packing")

Once the gutta-percha has been compacted to about 5–6 mm from the terminus of the preparation, a radiograph is taken to verify that the gutta-percha is seated at the desired location and that the obturation is well compacted (Figures 22-61 and 22-62). The remainder of the canal (middle and coronal thirds) is filled in a backwards fashion. It can be achieved via two different methods:

1. Introducing small pieces of gutta-percha into the root canal, and rhythmically heating and compacting and them.
2. Using thermoplastic gutta-percha.

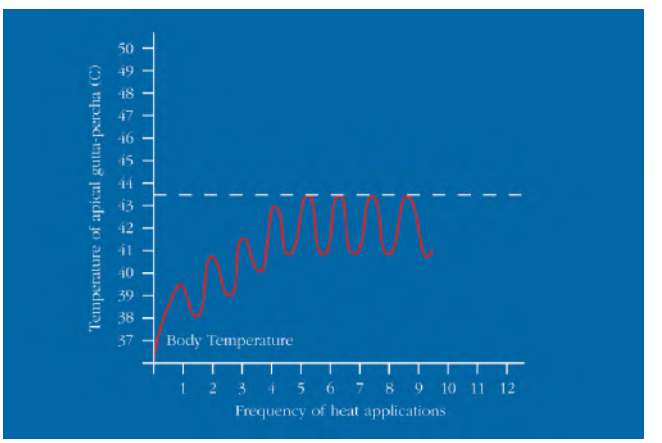

FIGURE 22-58 Thermal profile of gutta-percha in the apical portion of the canal preparation.[168]

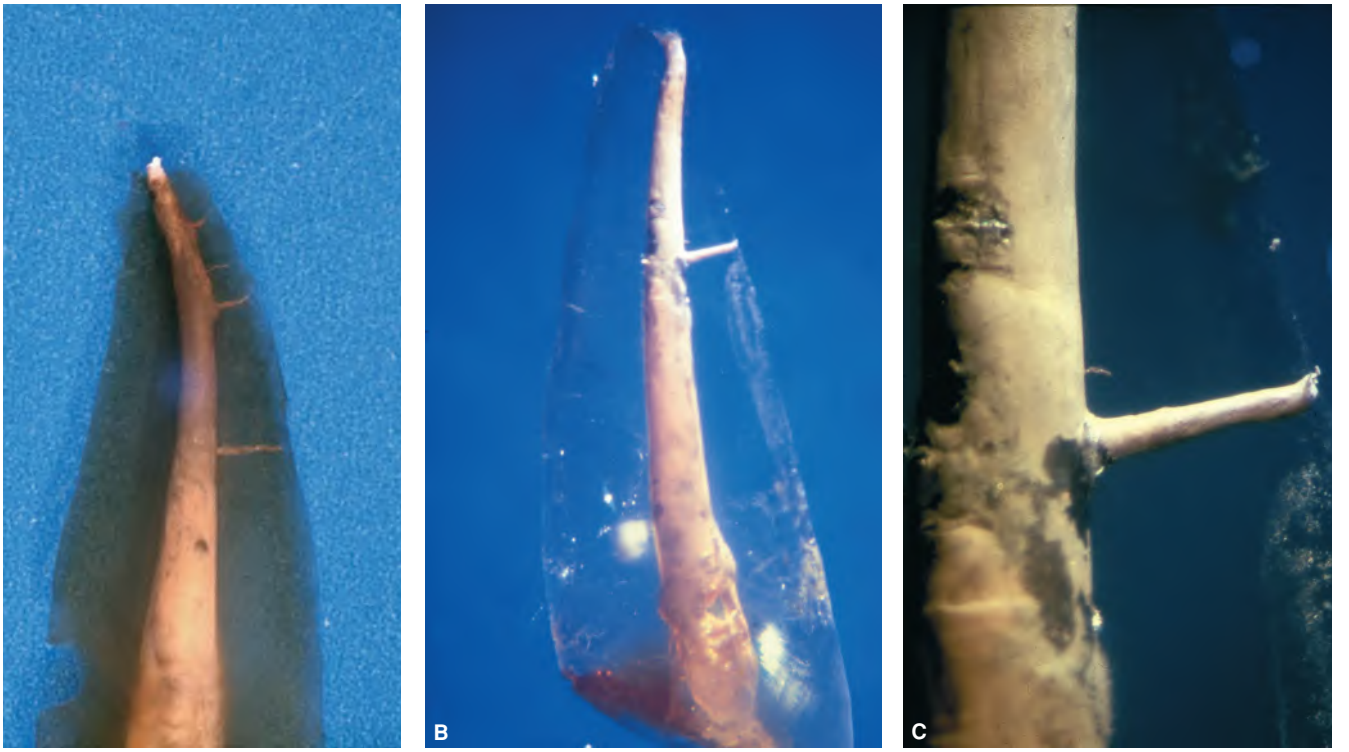

FIGURE 22-59 ***A***. An endodontically-treated maxillary central incisor extracted for periodontal reasons and cleared. Four lateral canals filled mostly with gutta-percha are evident. In this case, the compaction halted 5 mm from the canal terminus. ***B***. Maxillary premolar treated endodontically *in vitro* and then cleared. A lateral canal present about 4 mm from the apical foramen, is completely filled with gutta-percha. Heating and compacting was carried up to 5 mm from the terminus. ***C***. Higher magnification of 22-59B.

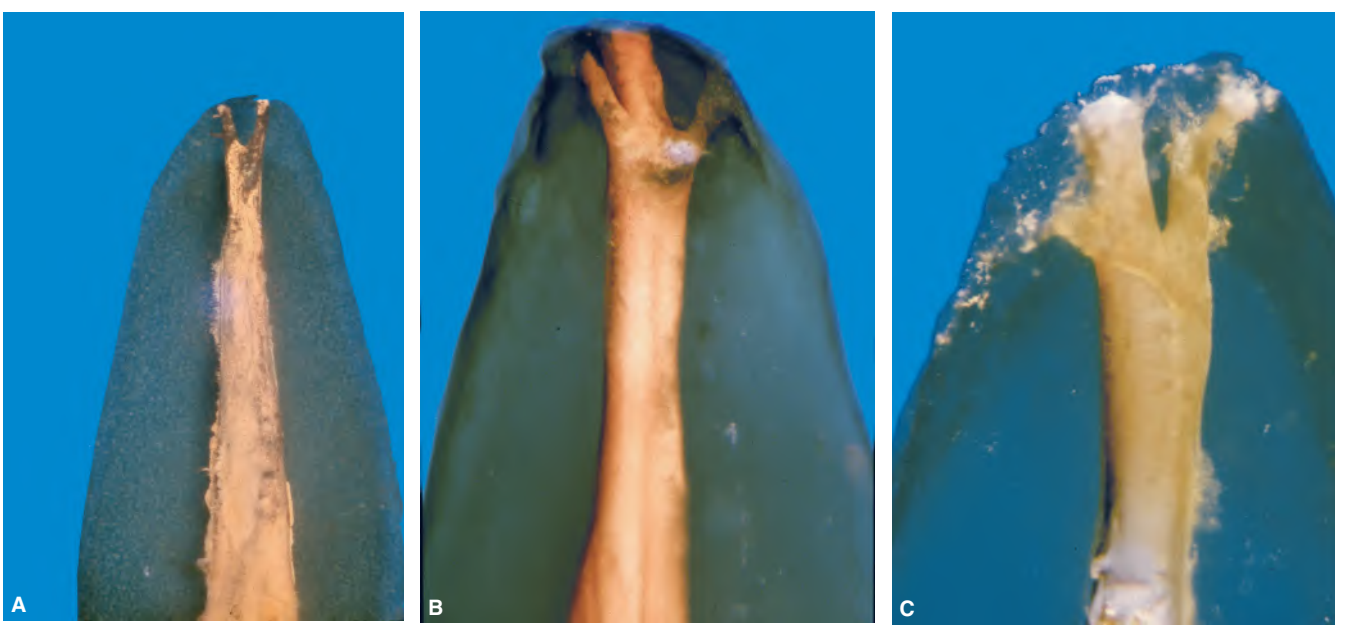

FIGURE 22-60 ***A***. A cleared root demonstrates a small apical bifurcation filled with gutta-percha and sealer. The compaction occurred 5 mm from the canal terminus. ***B,C***. Other examples of apical delta filled mainly with gutta-percha, heated, and compacted about 5 mm from the end of the canal preparation.

If remnants of gutta-percha or sealer are left attached to the dentin walls from the previous phase of compaction, the gutta-percha used for the back-packing phase may adhere to this material before fusing with the apical mass of gutta-percha, thus leaving empty spaces affecting the uniformity of the obturation (Figure 22-63).[157]

If post space or canal retention are needed for prosthetic purposes, the clinician may choose to terminate the

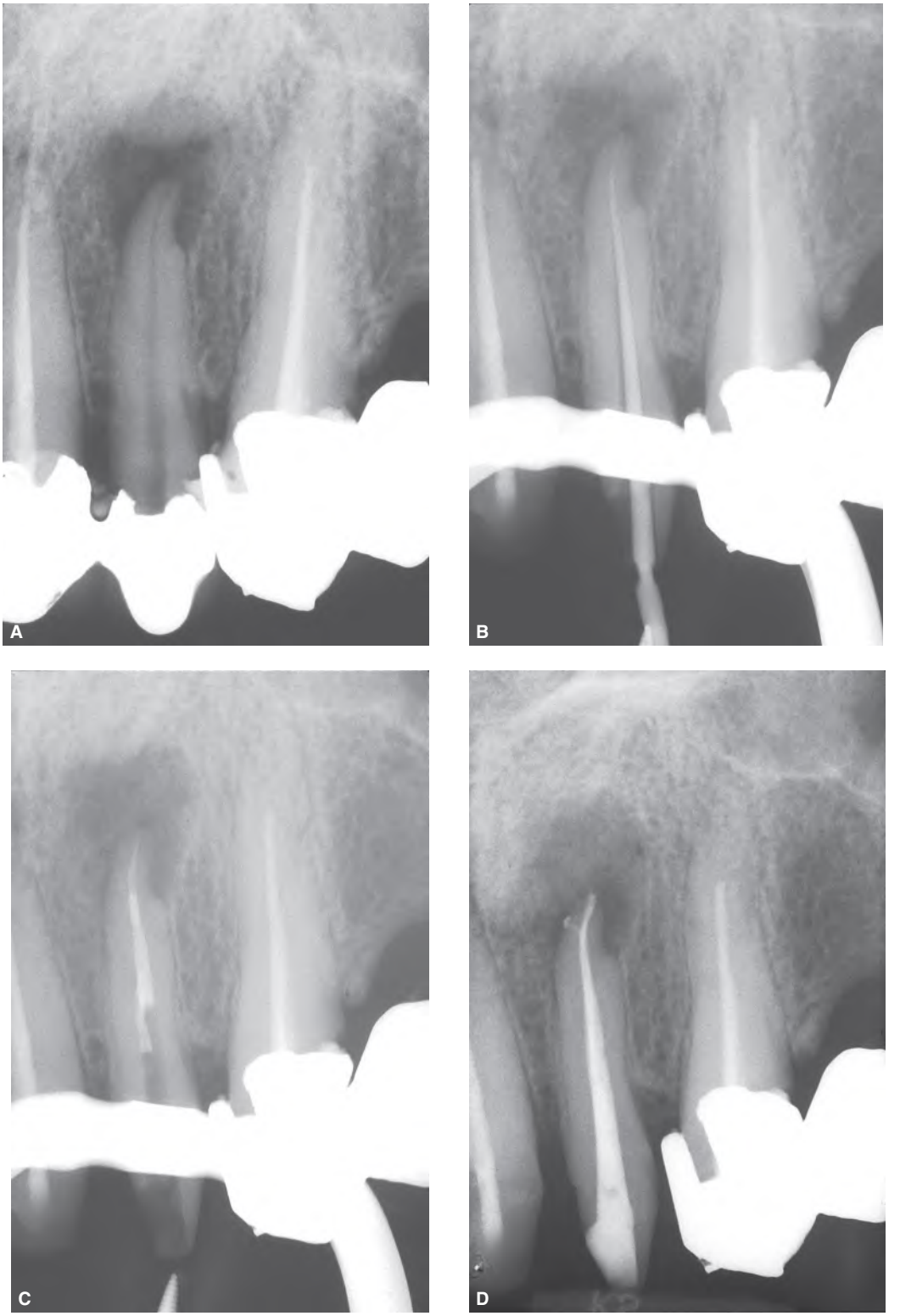

FIGURE 22-61 ***A***. Preoperative radiograph of maxillary left lateral incisor. ***B***. Intraoperative radiograph of the cone fit. ***C***. Radiograph of the apical compaction. The gutta-percha appears to be compacted for about 4 mm and then seems to fold. This is because the material has not been modified in the most apical portion. It is necessary to advance more apically with the heat-carrier and better compact the gutta-percha. ***D***. Postoperative radiograph showing the obturation material being homogeneous and well compacted. The presence of a lateral canal has also been demonstrated by the filling material.

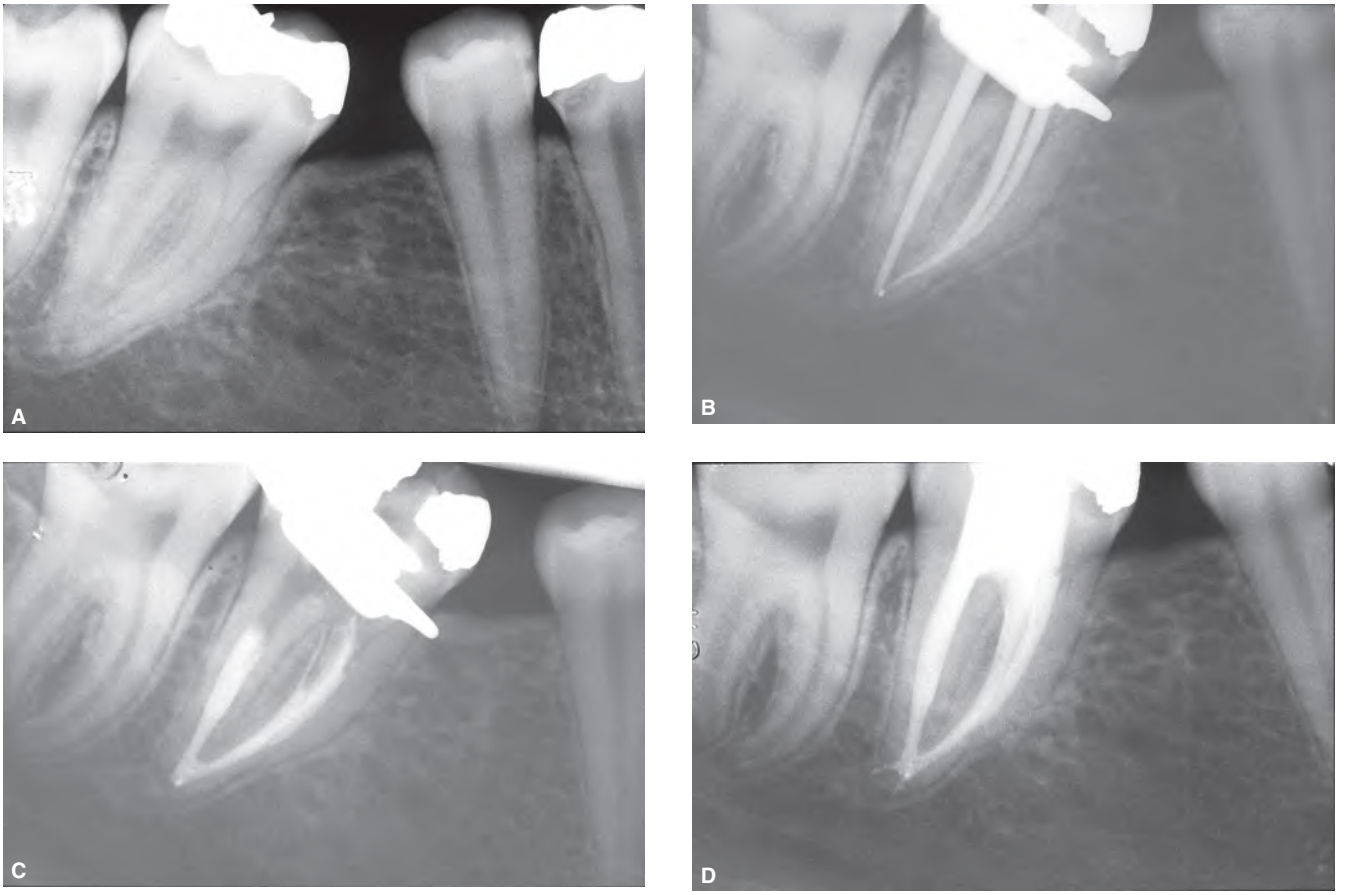

FIGURE 22-62 ***A***. Preoperative radiograph of a mandibular right second molar. ***B***. Cone fit. ***C***. Intraoperative radiograph of the apical compaction. The material appears to be homogeneous, but the obturation is slightly short. It is necessary to descend further apically (5 mm from the working length) with the heat-carrier and compact the gutta-percha better. ***D***. Postoperative radiograph showing filling material also in the small apical bifurcation.

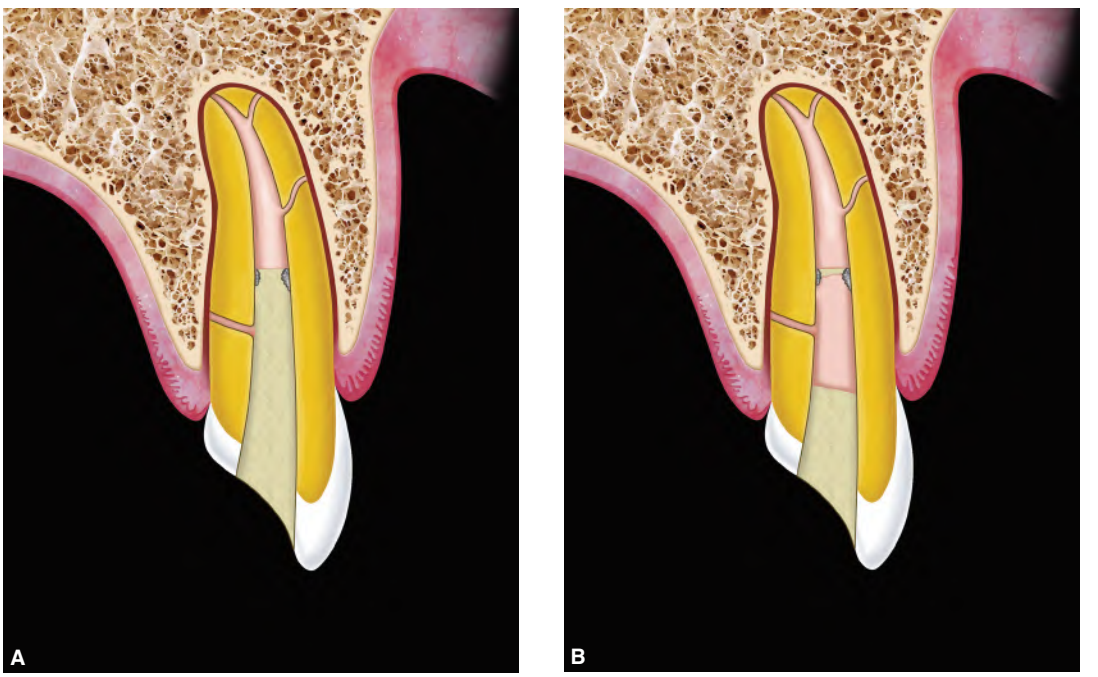

FIGURE 22-63 ***A***. During the initial phase of compaction, the plugger can leave streaks of gutta-percha along the walls (gray in the illustration). They must be re-directed toward the center of the root canal. ***B***. Streaks left behind can create voids during the "back-packing" phase.

obturation at the deepest point of the apical compaction or at any point of the reverse filling. Otherwise, the obturation can be completed up to the canal orifice and later partially removed to allow the necessary prosthetic space. In the single rooted tooth, the "back-packing" can be completed at any level since lateral canals that may have been present in the coronal or middle third of the canal have already been addressed during the downpacking (Figure 22-64). Furthermore, in anterior teeth, it is important to limit the backfilling to at least 2 mm below the gingival margin to prevent discoloration. Gutta-percha colorants reflecting through the coronal root and crown are unesthetic.

In multirooted tooth, however, complete "back-packing" and obturation of the canals and pulp chamber are imperative due to the presence of accessory canals on the floor of the pulp chamber. In these cases, once the canals have been filled, it is also necessary to seal the pulp chamber. If left unsealed it may cause an inflammatory reaction in the tissues associated with the furcation area.[160,168] For more details, see Chapter 36.

"Back-packing" with Thermoplastic Gutta-percha

When using thermoplastic gutta-percha such as Obtura III (Spartan Corporation), Beta 2 (B&L) (Figure 22-65), Elements (SybronEndo), Calamus (Dentsply Tulsa Dental) (Figure 22-36C & D), the reverse filling of the canal can be performed effectively in a significantly shorter time, especially in multirooted teeth.

This procedure is performed by introducing the narrowest needle (#23 or #25) into the root canal, and squeezing the trigger of the gutta-percha gun gently but decisively so as to feel the pressure of the gutta-percha. The pressure of the gutta-percha will push the operator's hand coronally while filling the canal. After the introduction of a small amount of gutta-percha, it is compacted by a corresponding size plugger. It is then re-heated by the heat-carrier and compacted again (Figure 22-66).

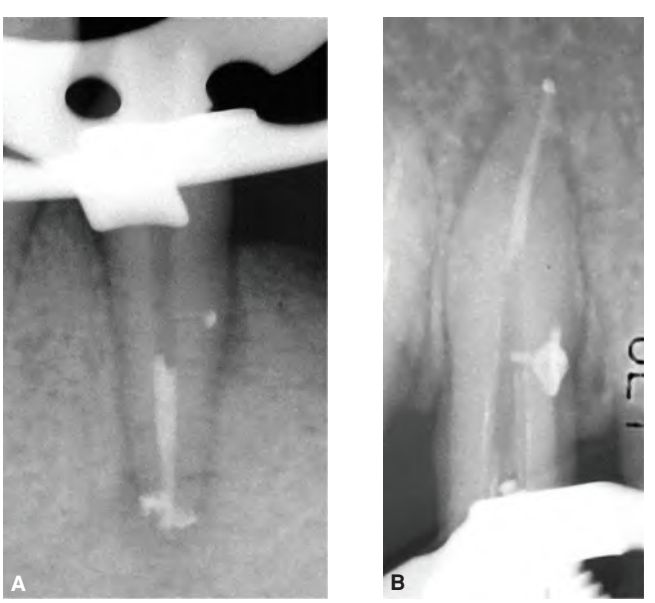

FIGURE 22-64 ***A,B***. Lateral canals can be filled during the downpacking.

FIGURE 22-65 ***A***. The Obtura II (Obtura Spartan Corporation, Earth City, MO, U.S.A.). ***B***. The Beta 2 (B&L Bio Tech, Seoul, Korea).

In general, with three successive introductions of material, "back-packing" of a canal is complete. It is always advisable to perform canal filling with thermoplastic gutta-percha in three or more injections (sectional technique), since better results are obtained (no entrappment of air bubbles) as compared to fillings performed in a single injection (single technique).[169] The single injection technique is more prone to leave voids.

LATERAL CONDENSATION OF GUTTA-PERCHA

Once the root canal has been cleaned and shaped, a gutta-percha cone is selected that fits well to the apical preparation (master cone). Usually, standard cones are peferred because they have less taper and permit a deeper penetration of the spreader (see later). The master cone should fit perfectly at the working length and show resistance to displacement ("tug back") (Figure 22-67). After the canal is irrigated and

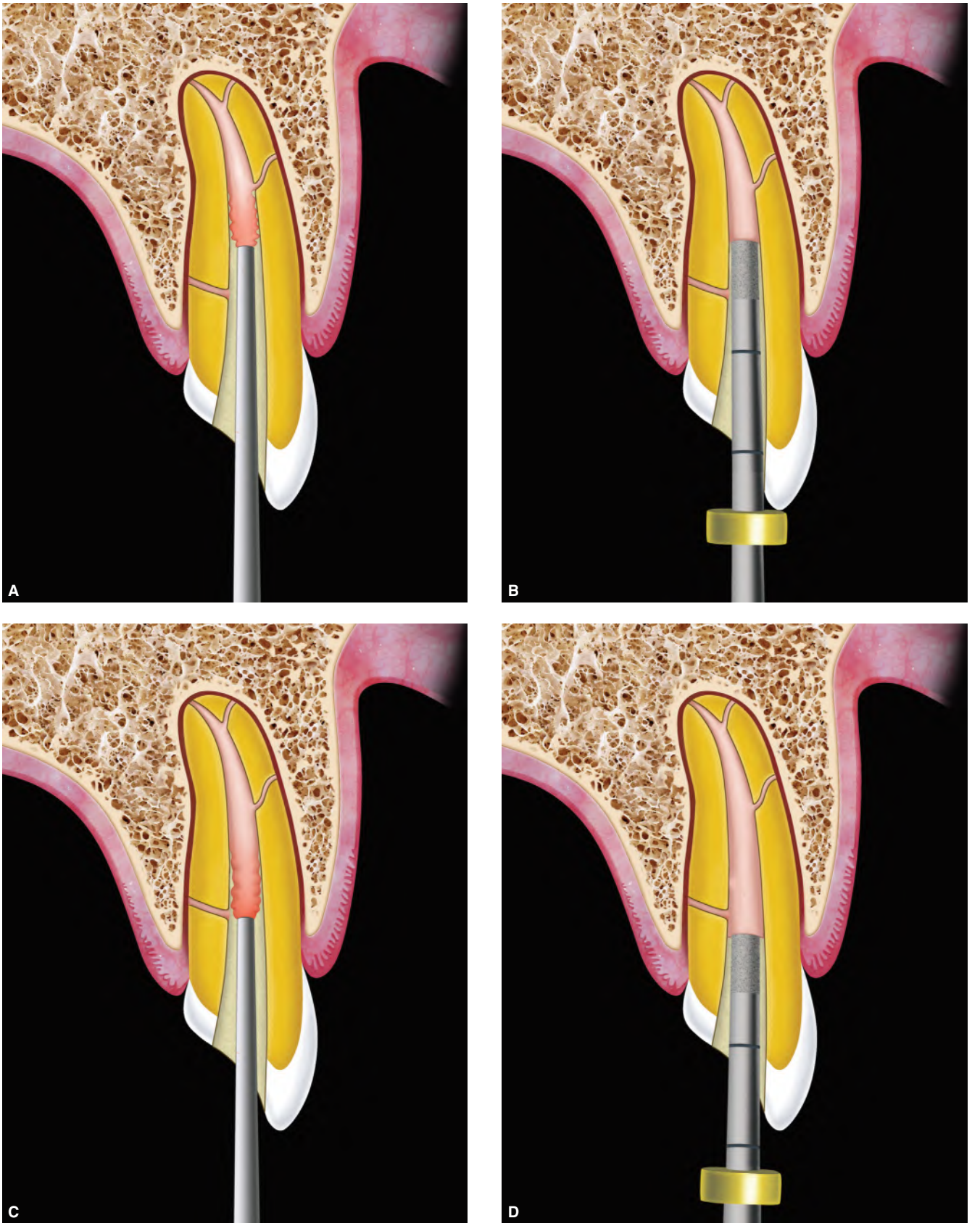

FIGURE 22-66 ***A***. The needle of the device touches and heats the apically compacted gutta-percha. Then a small amount of material is injected, in order to fill no more than 5 mm of space at a time. ***B***. The plugger, previously used in the middle third, is now used to firmly compact the material until the gutta-percha is plastic. ***C***. Additional 5 mm of gutta-percha are injected into the root canal. ***D***. The compaction is completed using the larger plugger.

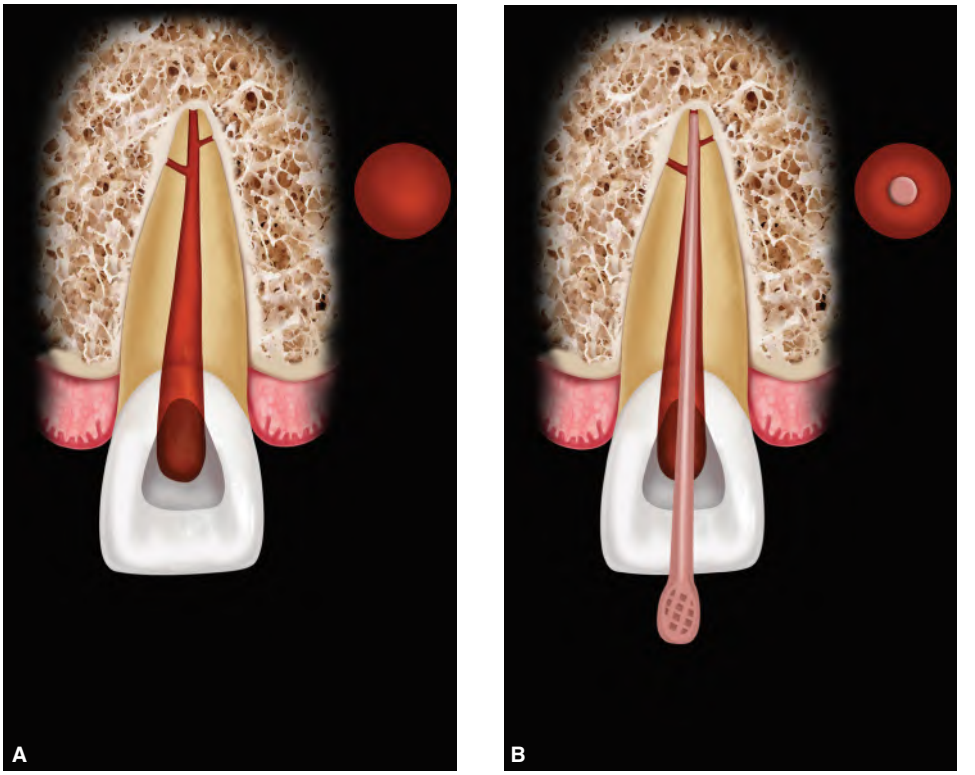

FIGURE 22-67 ***A***. Schematic illustration of the prepared root canal. ***B***. Fitting of the master cone. The cone is binding only in the apical portion of the prepared root canal.

dried with paper points, the master cone fit is confirmed with a radiograph.

A spreader is pre-fitted in the canal allowing its insertion to within 1.0–2.0 mm from the working length. Auxiliary cones are also selected to closely match the size of the spreader. The use of finger spreaders is suggested since they provide a better tactile sensation and are less likely to exhert excessive force on the root than the more traditional D-11T hand spreaders.[170–172] Spreaders are also available in nickel-titanium. These spreaders show increased flexibility,[173] reduce stress,[174] and provide deeper penetration as compared to stainless steel instruments.[175,176] The spreader should fit to within 1 mm to 2 mm of the working length (Figure 22-68) and when first introduced into the root canal alongside the master cone should penetrate up to 2 mm of the working length.[177]

After introducing a small amount of sealer in the prepared root canal, the master cone is slowly inserted to the working length. The spreader is then introduced between the master cone and the dentin walls to make room for the first auxiliary cone (Figure 22-69). The master cone is now laterally (and somewhat vertically) compacted, rotating the instrument in 180-degree motions.[178] During this movement the cone is compacted against a canal wall while, at the same time, space is created laterally to the master cone for the auxiliary cones. The spreader is then removed by rotating it back and forth as it is withdrawn from the canal and an auxiliary cone is inserted in the empty space (Figure 22-70). The spreader is now reinserted

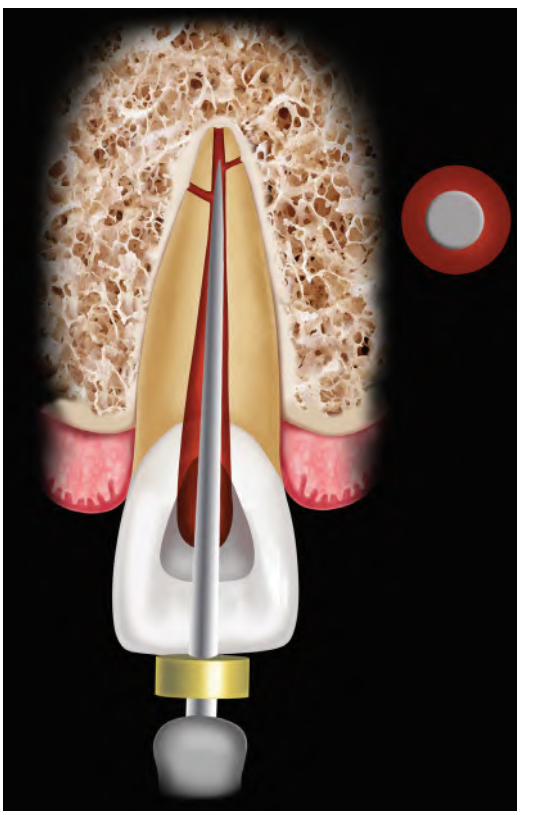

FIGURE 22-68 Spreader fit within 2 mm of the prepared canal without binding. Note the space available adjacent to the spreader.

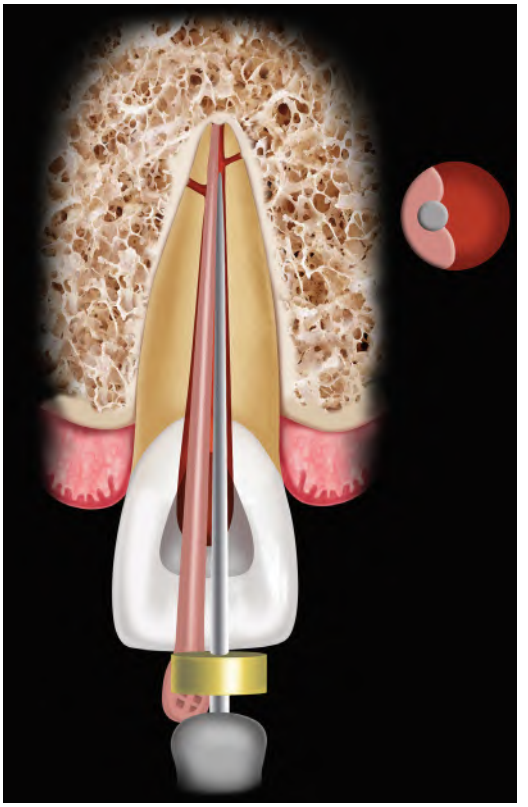

FIGURE 22-69 The spreader is introduced alongside the master cone to the proper apical depth.

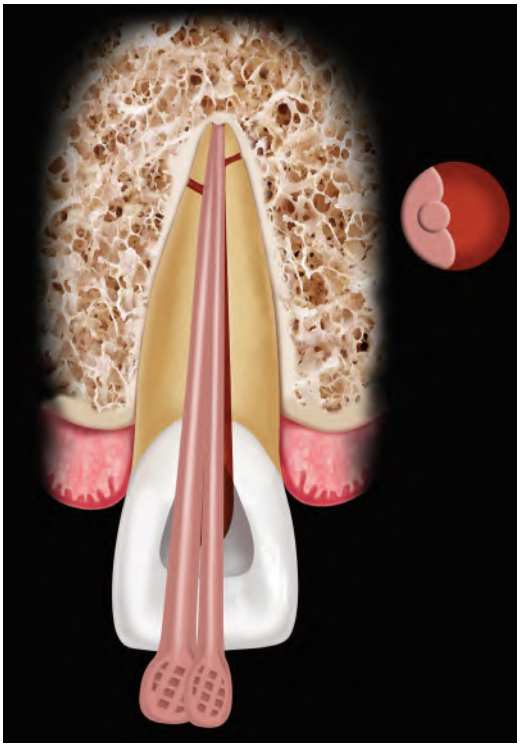

FIGURE 22-70 After careful removal of the spreader, an accessory cone, lightly coated with sealer, is introduced to the apical depth created by the spreader.

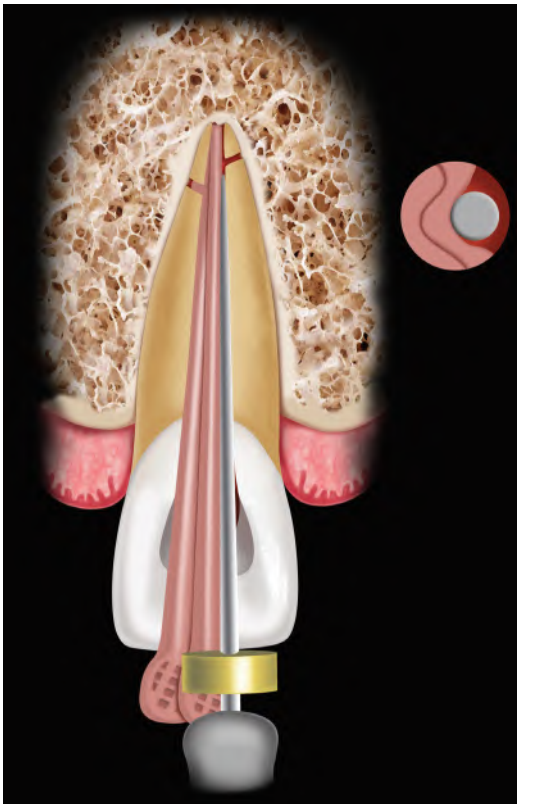

FIGURE 22-71 The spreader is reinserted to make room for an additional accessory cone.

(Figure 22-71) to make room for the next auxiliary cone. This sequence is repeated until there is no more room for insertion of the spreader or for additional auxiliary cones (Figure 22-72).

The auxiliary cones are selected based on the size of the spreader, the size of the canal, and the position of the space created inside the canal. Prior to their insertion, the auxiliary cones are lightly coated with sealer and are introduced to the same depth that the spreader has last reached. When the spreader can only penetrate 2 or 3 mm into the canal orifice, the obtutation is completed. The pressure exerted during lateral condensation should be very light because gutta-percha is not compressible, and since as little as 1.5 kg of pressure is capable of fracturing the root.[179]

Finally, the excess of gutta-percha protruding in the pulp chamber is seared off with a heat carrier and is vertically compacted with prefitted pluggers. The pulp chamber is cleaned with cotton pellets soaked in alcohol to remove any residual particles of gutta-percha or sealer.

The lateral condensation technique has several drawbacks:

1. Being a "cold" technique, the gutta-percha cones don't form a homogeneous, compact mass. The final filling will comprise of a number of gutta-percha cones separated by a greater or lesser amount of sealer,[180] depending on the clinician's profeciency with the technique.

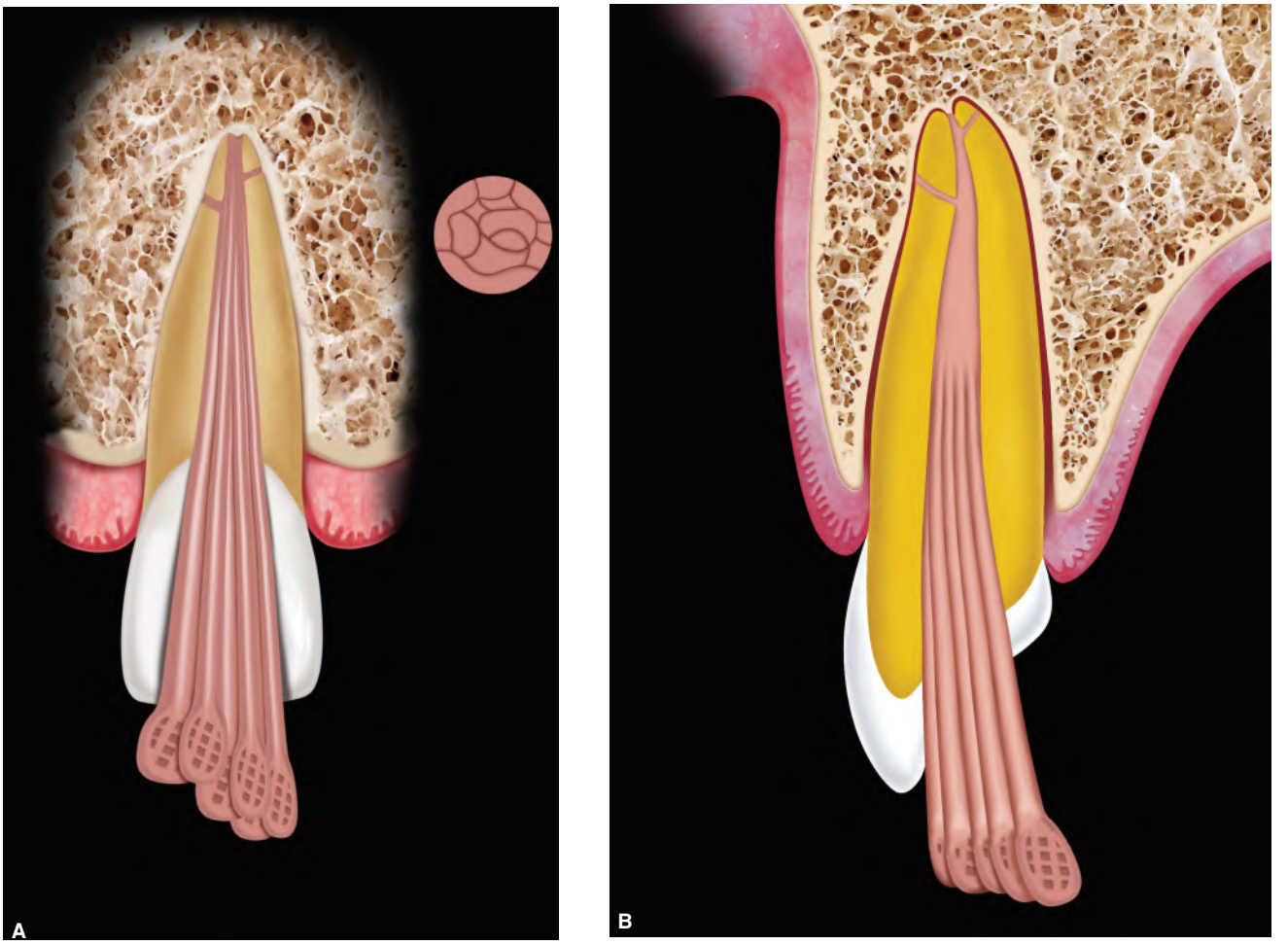

FIGURE 22-72 ***A***. The obturation process is continued until there is no more room for additional insertions of the spreader or further condensation of accessory cones (Facial view). At this point, the obturation is completed. ***B***. Proximal view of the obturated canal.

2. Filling of lateral portals of exit occurs less frequently than when vertical compaction is performed and is always constituted of sealer, not gutta-percha.[181]
3. The spreader should be placed within 1 mm to 2 mm of the working length.[88] If not carefully done, it may exhert excessive lateral forces, increasing the risk of root fracture.[182] For more details, see Chapter 14, "Vertical root fractures."
4. When considering that heat is not used, the most apical portion of the master cone does not undergo significant modification. The seal in this area is, therefore, mainly entrusted to the sealer.

In vitro studies[183,184] demonstrated that cold lateral condensation may show more volumetric leakage as compared to other techniques. Gilhooly et al.[185] found increased apical leakage to dye when teeth were obturated, with lateral condensation, *in vitro*, as compared to heated multiphase gutta-percha obturation. Other studies regarding density and fill by weight of obturation showed heat-related techniques of compacting gutta-percha to have advantages over lateral compaction as well as gutta-percha to sealer ratio.[186]

Studies by Gimlin et al.[182] demonstrated that the stress produced in the apical area is greater during lateral than vertical condensation. Furthermore, when forces are applied to create identical apical stress, the average force registered throughout the entire length of the canal, is higher in vertical than lateral condensation thereby producing forces that are uniformly distributed along the entire root surface.[181,182] On the other hand, lateral condensation may create stress only in a small area, near the tip of the spreader. Such concentration of stress may cause fractures, if the technique is not applied correctly (Figure 22-73).

While one study[187] reported a 10% greater success rate with warm vertical compaction versus cold lateral compaction in cases with apical periodontitis, not many studies support this finding but rather show no difference. For more details, see Chapter 33, "Outcome of endodontic therapy."

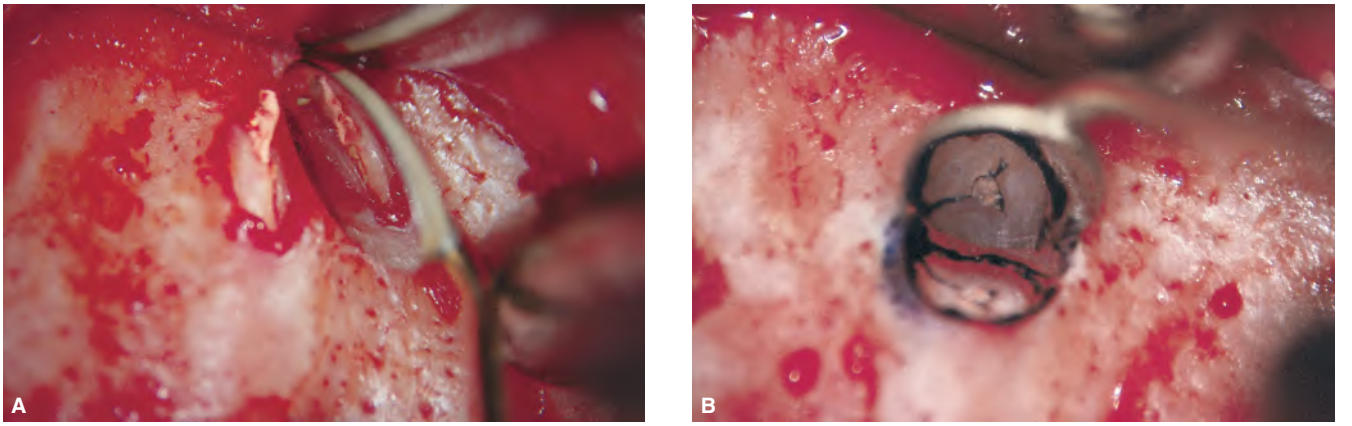

FIGURE 22-73 ***A***. Intraoperative photograph taken during surgical endodontic treatment in a maxillary first premolar, previously obturated by lateral condensation. The clinician probably used excessive force during obturation. An apical vertical root fracture is present. ***B***. After removal of the apical fractured fragment, a fracture line is still present on the bevel of the root (stained with methylen blue). The tooth had no coronal probing, confirming that the fracture was apically induced.

THERMOPLASTIC GUTTA-PERCHA TECHNIQUES

The thermoplastic gutta-percha technique was introduced by Yee et al.,[188] Torabinejad et al.,[183] and Marlin et al.[189] It consists of injecting gutta-percha, heated by an electrical device, into a prepared root canal. The instrument has a gun-like shape (Figure 22-65) whose cartridges are small gutta-percha cylinders that are heated to a temperature regulated by the user. Exerting pressure on the "trigger" activates a piston that presses the gutta-percha toward the tip of the instrument. The gutta-percha is then conveyed through a thin silver needle that, when appropriately bent, allows its operation in root canals of various sectors of the mouth.

This technique uses sealer that also lubricates the plastic material on its path toward the apex,[148] in addition to ensuring a better seal.[190] Yee et al.[188] demonstrated that it is possible to obtain dense obturation, without entrapped air bubbles, if the technique is accompanied by the use of a sealer. They also demonstrated the presence of filling material in lateral canals, in addition to a good apical seal.

Torabinejad et al.[183] observed that, in this technique, the gutta-percha adapts well to the dentin walls. The adaptation was similar to that obtained with other conventional techniques, apart from the presence of some small voids, that were not visible radiographically. Weller et al.[191] observed that the thermoplasticized injectable technique exhibited the best adaptation to the prepared root canal walls, when compared to Thermafil and cold lateral condensation. Marlin et al.[189] showed that this technique produces good clinical results and a significant reduction in working time. The same authors also emphasized the importance of combining gutta-percha with a sealer, also suggested by subsequent studies,[192–194] and warn against the danger of extrusion of material beyond the apex.

A drawback of this technique is that it lacks good and predictable apical control during obturation.[195–197] The gutta-percha injected from the syringe is already warm and plastic. Moreover, the user does not have precise control on either the pressure that is exerted or the amount of gutta-percha being introduced into the root canal. To prevent it, the operator must form, during the shaping procedure, a good "apical barrier"[198] to avoid extrusion of excessive material into the periodontium.[199] The needle of the syringe should be positioned no less than 4–6 mm from the end of the preparation, otherwise, a high percentage of underextended obturation will result.[200]

The technique may be confidently used only when there is no risk of introducing material beyond the apex, in the following circumstances:

1. Back-packing, to fill the coronal aspects of the canal following the initial placement and compaction of gutta-percha in the apical portion of the canal.
2. Unnegotiable canals where it is necessary to fill the canal space as much as possible, via coronal approach, prior to surgical root-end filling (Figure 22-74).
3. In partially unnegotiable canals when sometimes, thermoplastic gutta-percha succeeds in obturating portions of canal that had remained unnegotiable to endodontic instruments. In many cases, endodontic surgery or root amputation may not be necessary[201] (Figure 22-75).
4. After closure and maturation of the root following apexification (Figure 22-76).
5. After obtaining an apical barrier with MTA[202] (Figure 22-77).
6. Root canals with internal resorption, after the apical third of the root canal has been obturated in the traditional manner (Figure 22-78).
7. Root canals in which, due to inadequate previous shape, the operator is unable to fit a traditional gutta-percha cone with the tug-back in the appropriate position

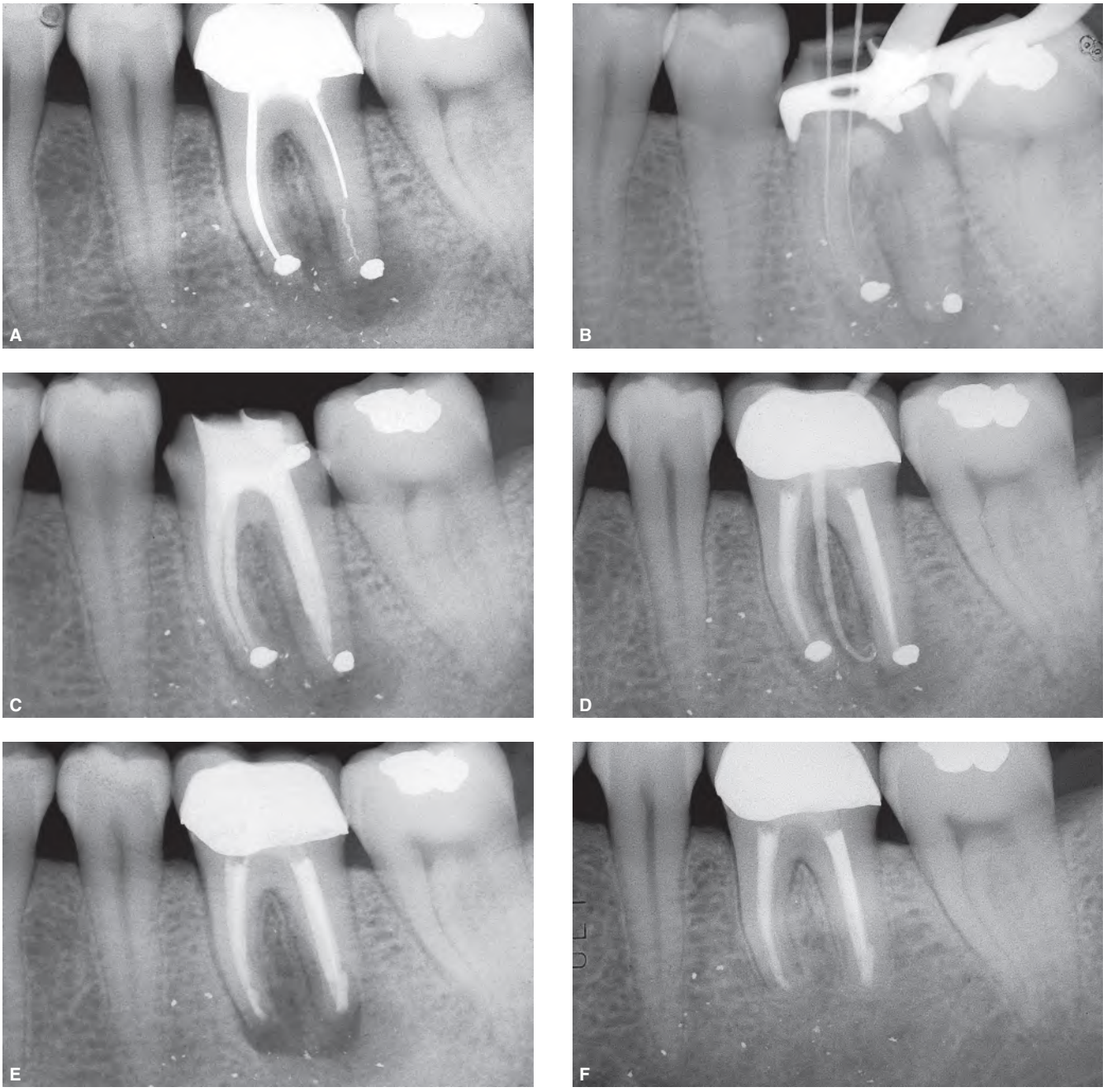

FIGURE 22-74 ***A***. Preoperative radiograph of a mandibular left first molar. The tooth was obturated with silver cones. A separated instrument is also present in the distal root. ***B***. Intraoperative radiograph during non-surgical retreatment. The silver cones and the separated instrument were successfully removed. ***C***. Immediate postoperative radiograph. ***D***. Six-month follow-up radiograph. A sinus tract is now present. At this point, surgical retreatment was carried out. ***E***. Postoperative radiograph following surgery. ***F***. Two-year recall radiograph showing a healing of the bony lesion.

(Figure 22-79), or root canals in which, due to unusual anatomy or presence of a ledge, it is very challenging to introduce a traditional gutta-percha cone (Figure 22-80).

8. Root canals where a perforation is present in the apical third of the root (Figure 22-81).
9. During endodontic surgical procedures,[203] applying obturation of the root canal from the apical end, before positioning a root-end filling (Figure 22-82).
10. In non-surgical retreatment of surgical failures (Figure 22-83).

Another limitation of this technique is the high temperature that the gutta-percha reaches within the syringe (160°C or more) before being introduced into the root canal. Techniques using heat and compaction, for example, the Schilder's warm gutta-percha technique, require substantially less heat (45°C to 55°C).[151, 204]

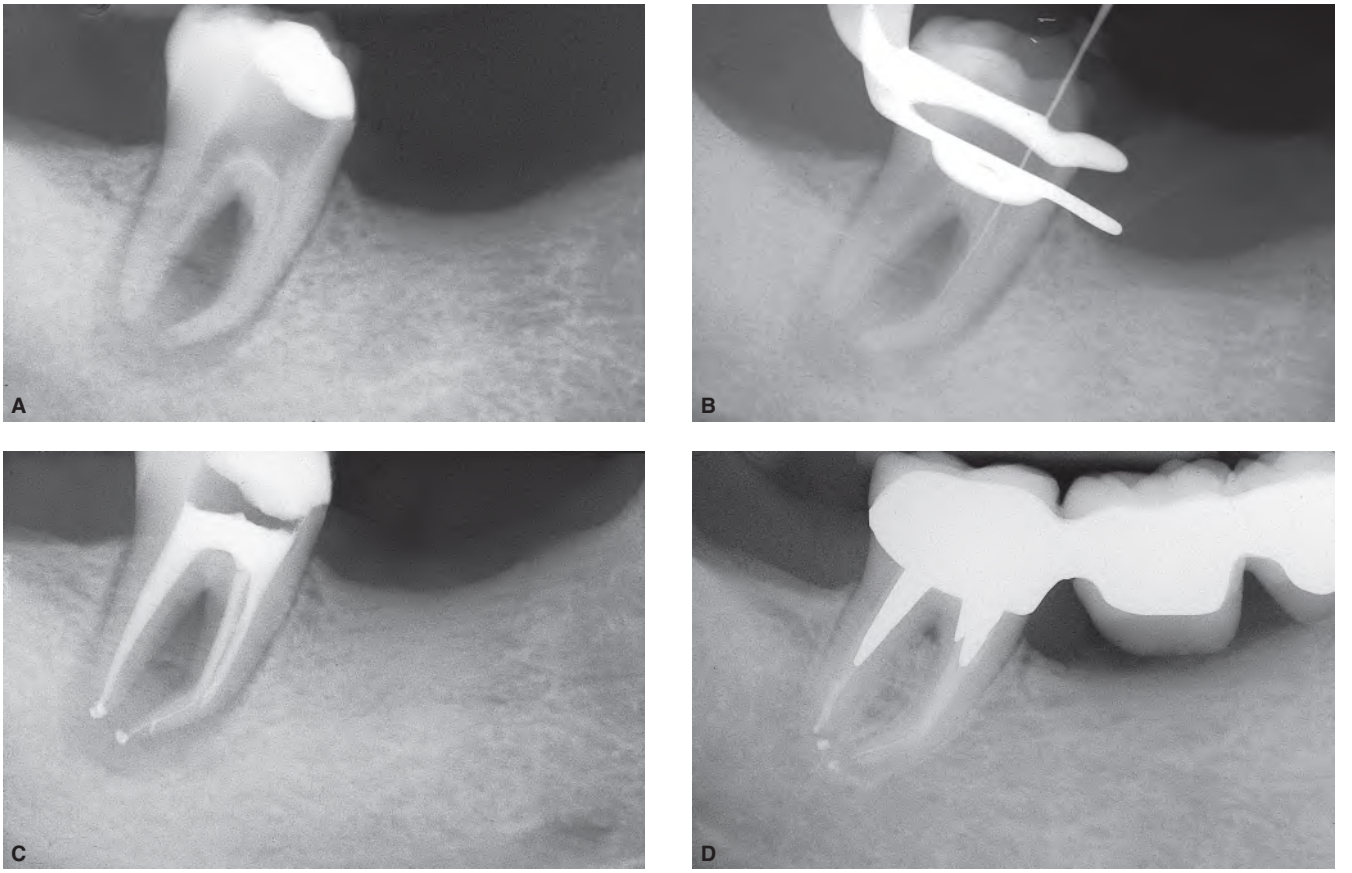

FIGURE 22-75 ***A***. Preoperative radiograph of a mandibular right second molar. The patient was referred for treatment after the distal canal had been prepared and the mesial canals were only partially negotiable. ***B***. A ledge was present at the beginning of the curvature of the mesial canals. ***C***. Postoperative radiograph. The distal canal was obturated with warm gutta-percha, and the mesial canals with thermoplastic gutta-percha. Note the filling of the non-instrumented portion of the mesial canals. ***D***. Three-year recall radiograph showing successful results.

Commercial gutta-percha is found in the "beta" molecular configuration at 37˚C and begins to transform into the "alpha" configuration when heated to 42˚C–49˚C. It then passes to an amorphous phase when heated to 53˚C–59˚C.[205] During cooling, amorphous gutta-percha crystallizes (with a consequent diminution of volume) and returns to the "beta" configuration between 37˚C and 40˚C, without passing through the "alpha" configuration, unless it is slowly cooled. Under clinical conditions, cooling of thermoplastic gutta-percha occurs rapidly. Consequently, there is a diminution of volume in the recrystallization phase.[204] Therefore, it is important that vertical pressure be applied during cooling to compact the gutta-percha and compensate for its volumentric diminution.[151] Studies[198–208] have not demonstrated any damage to the periodontal tissues. This is because the heated gutta-percha cools rapidly once introduced into the root canal.[209]

Voids may be formed while using this technique. In order to avoid them, the operator's hand should be driven coronally by a light pressure exerted by the gutta-percha as it accumulates in the root canal (passive injection). A steady force can push the material beyond the apex. On the other hand, if the gun is withdrawn coronally too quickly, voids can be formed within the obturation. Another cause for voids formation is the injection of gutta-percha that is not sufficiently warm or plastic.

To avoid voids or entrapment of air during obturation, the following is recommended:

- Before injecting into the canal, squeeze the gutta-percha out of the needle, so that the material introduced in the canal comes directly from the heating chamber and, therefore, is warmer than the material inside the needle.
- Insert the needle into the canal orifice and wait for a few seconds before injecting. The needle will lose some heat after insertion, so it is necessary to wait until it becomes warm again.
- Inject small amounts of gutta-percha, no more than five millimiters each time. Only 5 mm of gutta-percha can be effectively compacted at a time.

Michanowicz et al.[210] introduced a device for thermoplastic gutta-percha at a temperature of 70˚C (Figure 22-84). The instrument resembles the Peripress syringe for intraligamental anesthesia. It is loaded with carpules of gutta-percha with needles and placed in a device that externally heats the carpule and the needle to 70˚C. The gutta-percha used

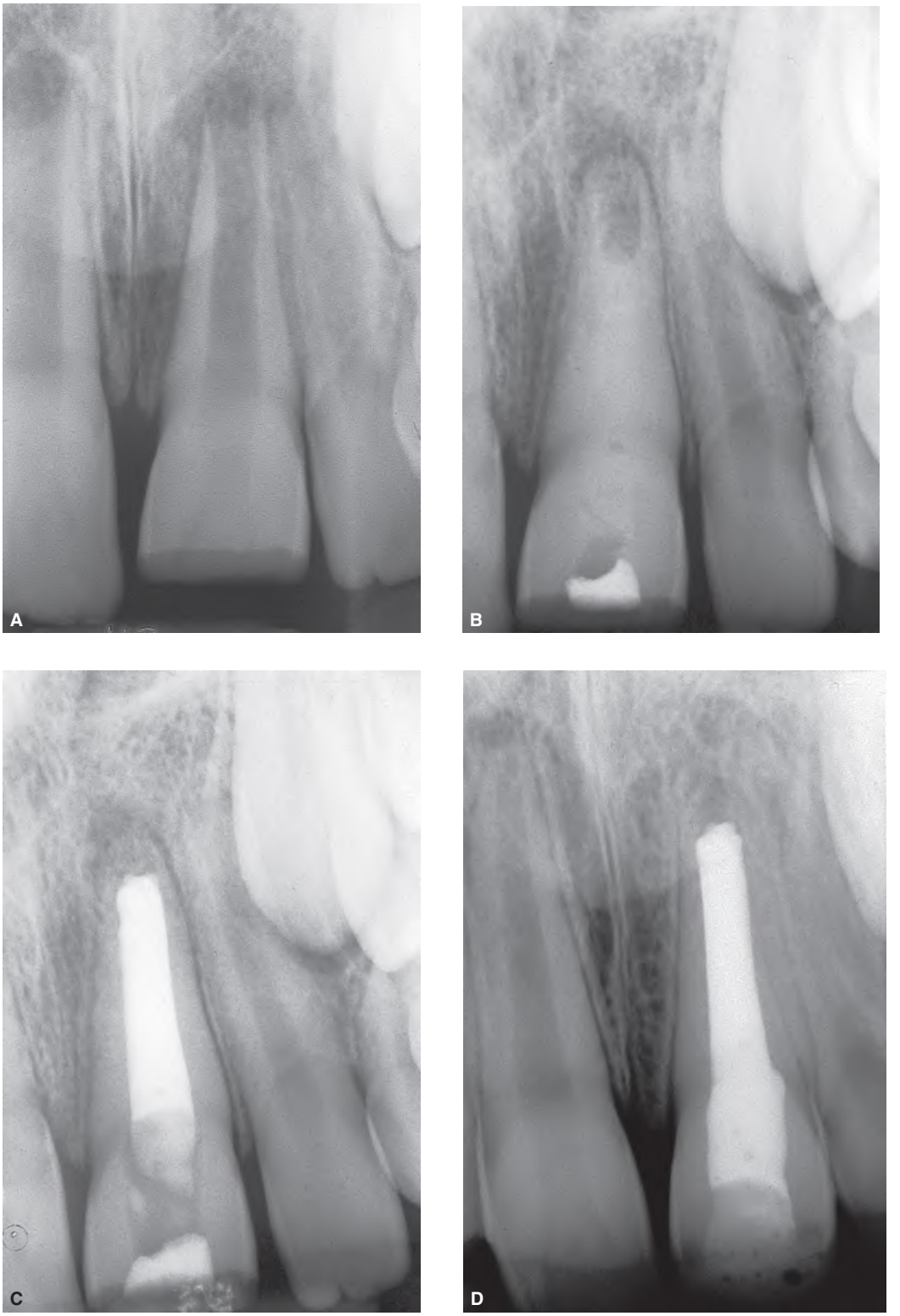

FIGURE 22-76 ***A***. Preoperative radiograph of a maxillary left central incisor with an immature apex and necrotic pulp in a 9-year-old patient. ***B***. Radiograph taken after six months of therapy with calcium hydroxide. Apical barrier is evident. ***C***. Postoperative radiograph following obturation with thermoplastic gutta-percha. ***D***. Two-year recall radiograph showing successful results.

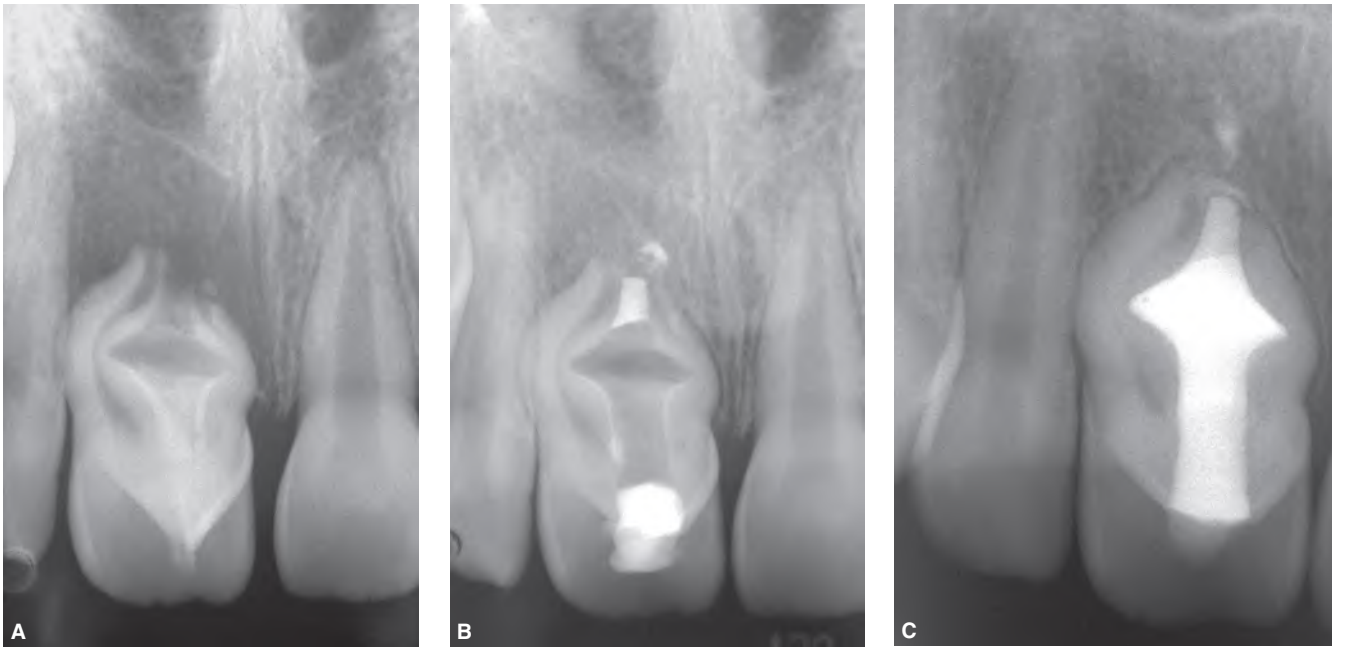

FIGURE 22-77 ***A***. Preoperative radiograph of a maxillary right central incisor. Note the periapical radiolucency, the unusual anatomy, and the wide open apex. ***B***. Three millimeters of MTA were placed at the foramen, using the apical barrier technique. ***C***. 2-year recall radiograph showing successful results.

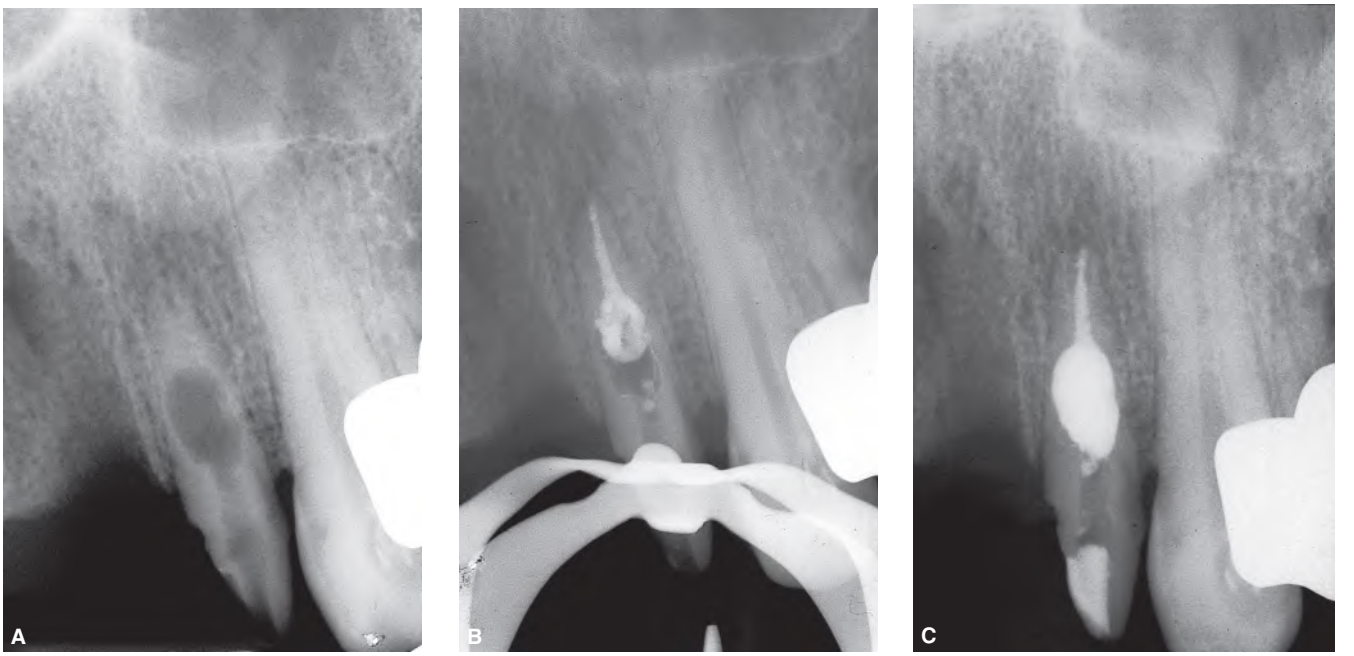

FIGURE 22-78 ***A***. Preoperative radiograph of a maxillary left lateral incisor with a large internal resorption defect. Once this type of resorption is diagnosed, it is necessary to intervene immediately because the lesion tends to progress. ***B***. Intraoperative radiograph of the apical condensation. ***C***. Postoperative radiograph. The area of internal resorption has been filled with thermoplastic gutta-percha.

in this case differs from other commerical gutta-percha by its higher paraffin content. This contributes to rendering it malleable at a lower temperature than that required by the Obtura or the B&L syringe. On the other hand, it is more difficult to compact.

Michanowicz et al.[211] suggested that this technique can be more successful in filling the dentinal tubules of the coronal and middle thirds of the canal with gutta-percha. However, in contrast to the preceding instrument, that was ready for use with one minute after being turned on, and

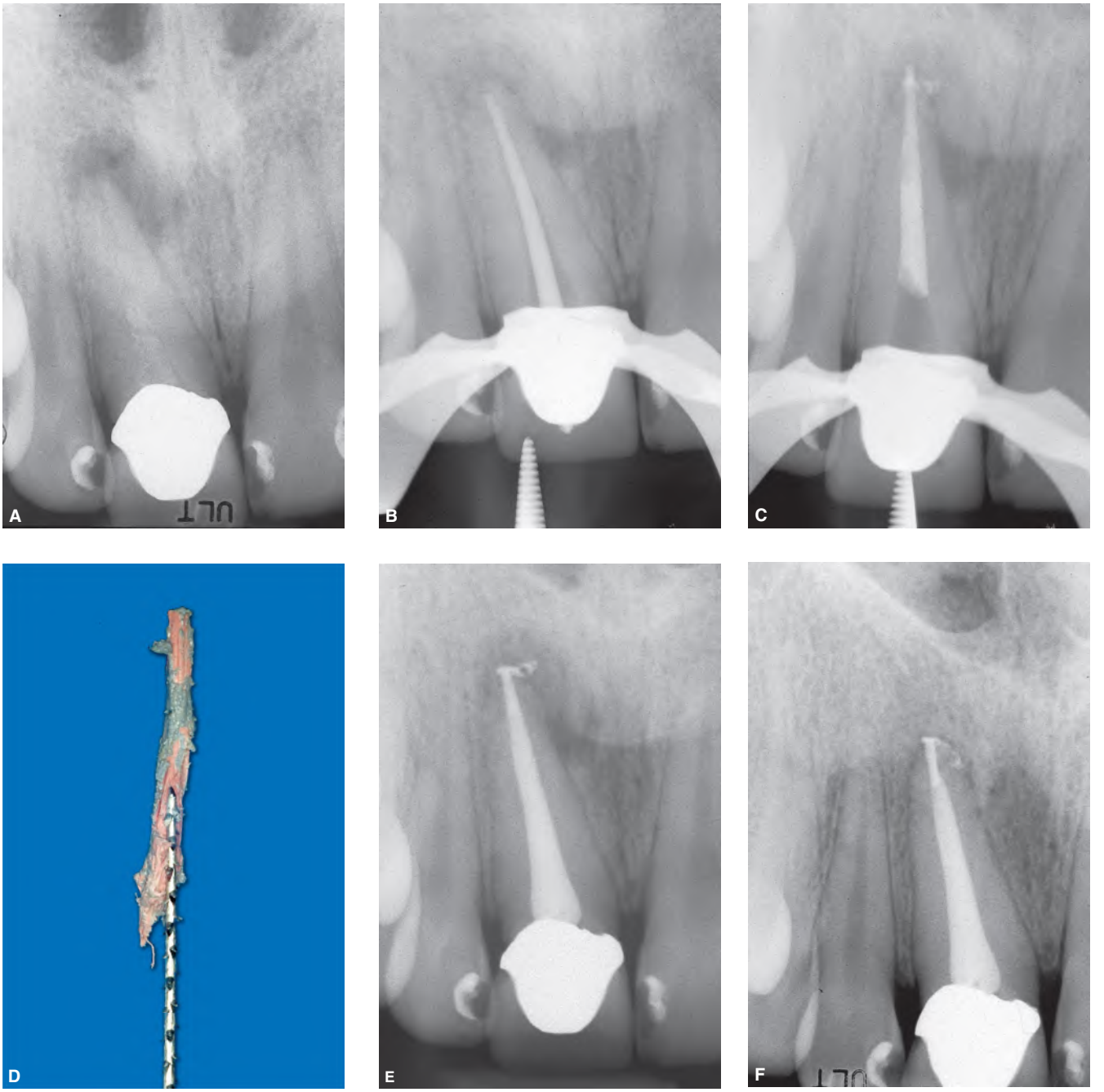

FIGURE 22-79 Owing to a previous improperly performed shaping, the master cone had a tug back only in the middle third of the canal, resulting in an inevitable extrusion of filling material beyond the apical foramen. The extruded gutta-percha was removed and the obturation completed with an Obtura syringe, using the sectional technique. ***A***. Preoperative radiograph. ***B***. Radiograph of the cone fit. Clinically, the cone presented a tug-back. ***C***. Evidently, the tug-back was in the middle third of the canal. ***D***. Using a large barbed broach, the gutta-percha cone was removed from the canal. ***E***. Postoperative radiograph following obturation with the sectional technique of thermoplastic gutta-percha. ***F***. Two-year recall radiograph showing successful results.

could have been left in the unit constatntly, this instrument requires heating for 15 minutes, and the syringe cannot remain in its hub for more than 4 hours.[199] Furthermore, once the syringe has been removed from the heating device, it can only be used for about one minute, after which the gutta-percha cools off and is no longer flowable. Several minutes are required to re-heat it.

The first commercially available thermoplasticized gutta-percha system was the Obtura system. Recently, another device became available on the market, the B&L Beta 2, that appears to be easier to handle as it is cordless. It is fast in reaching the selected temperature and has a silver needle that can be rotated and oriented without any risk of breakage. It uses the same type of gutta-percha pellets as the original

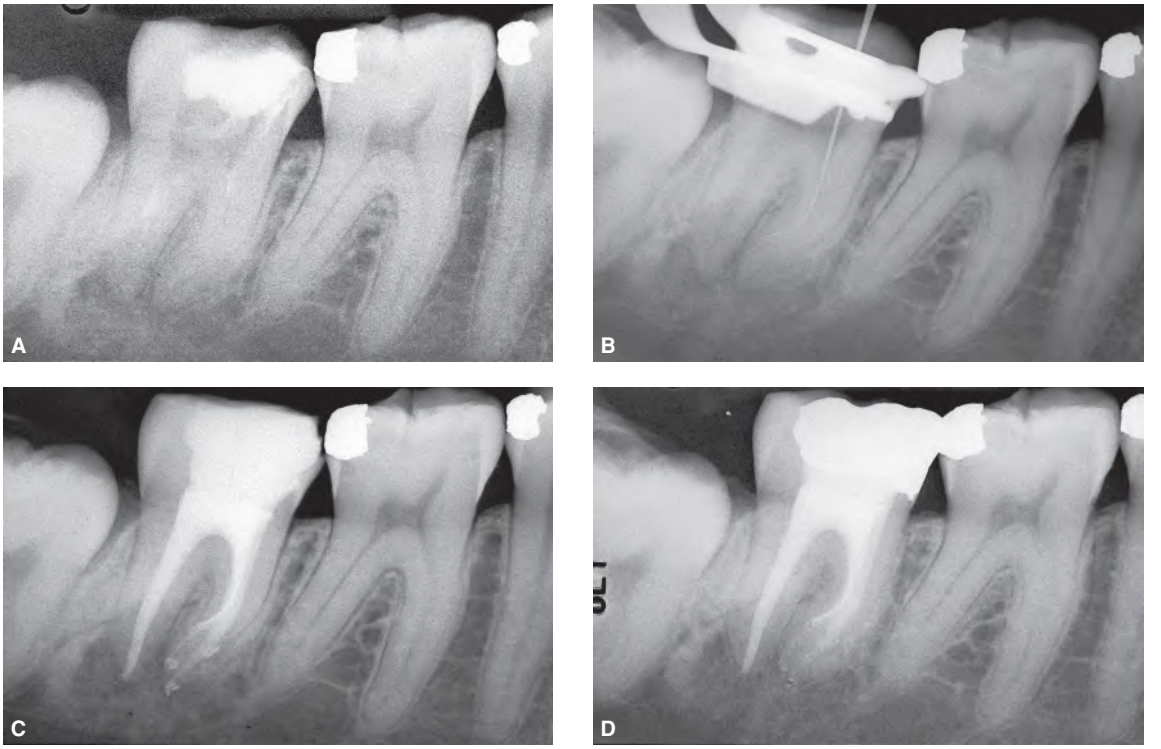

FIGURE 22-80 ***A***. Preoperative radiograph of a mandibular right second molar requiring retreatment. ***B***. A marked bayonette curvature of the mesiolingual canal was detected. The apical third of this canal was cleaned and shaped only with #08 and 10 K-type files. ***C***. Postoperative radiograph. The distal and mesiobuccal canals have been obturated using the Schilder's warm gutta-percha technique, while the mesiolingual canal was filled with thermoplastic gutta-percha. ***D***. Three-year follow-up radiograph showing successful results.

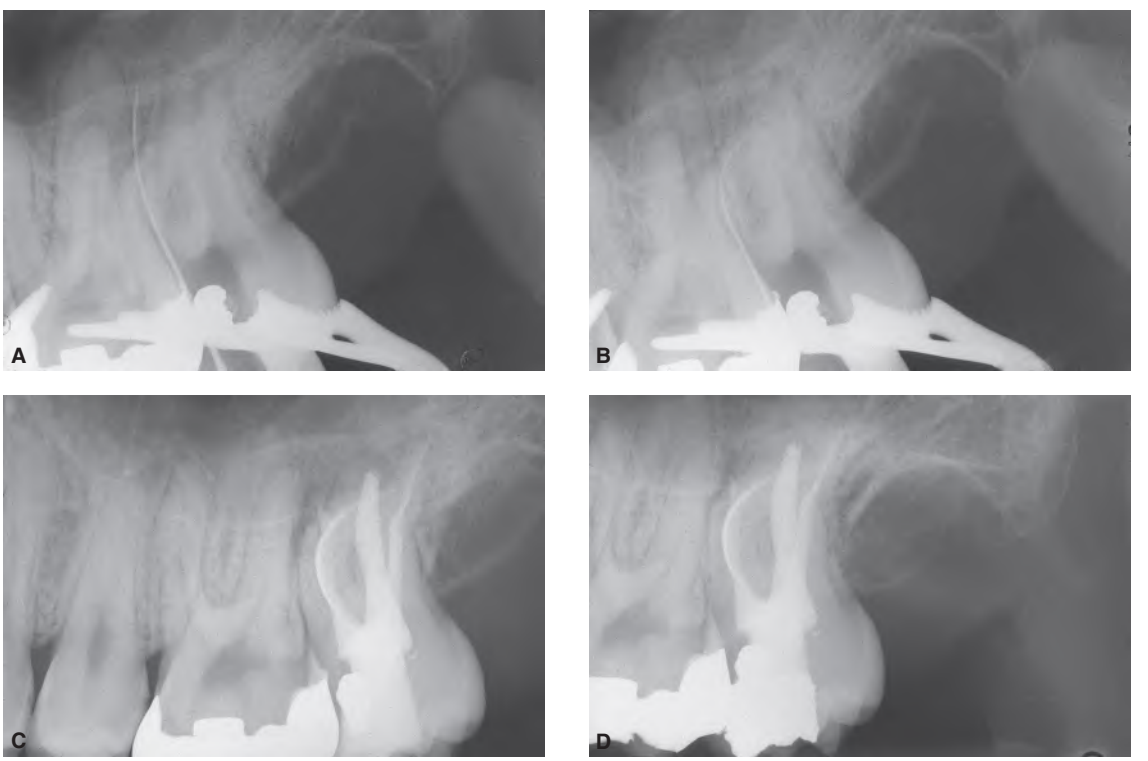

FIGURE 22-81 ***A***. A #20 file demonstrating the presence of a root perforation. ***B***. Using a #08 file, the original path of the canal was located. Cleaning and shaping can therefore be completed. ***C***. Postoperative radiograph. The perforation was obturated as if it were a lateral canal, using thermoplastic gutta-percha. ***D***. One-year follow-up radiograph showing successful results.

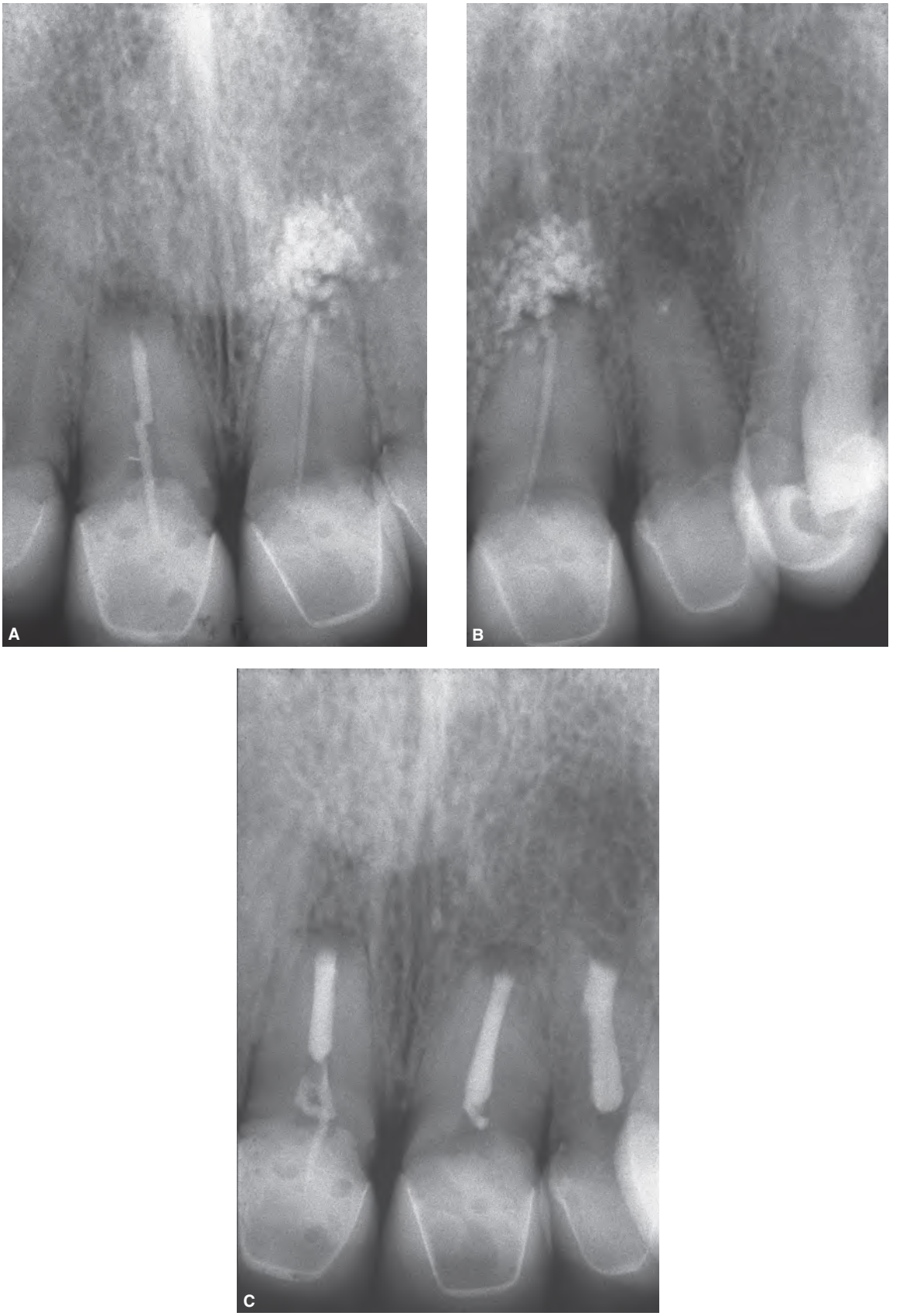

FIGURE 22-82 ***A,B***, Preoperative radiographs of the maxillary central incisors and the left lateral incisor. Surgical endodontic treatment was done. The thermoplastic gutta-percha was introduced to each root canal during the surgical procedure, before root-end filling with white-color MTA. ***C***. Seven-month recall radiograph.

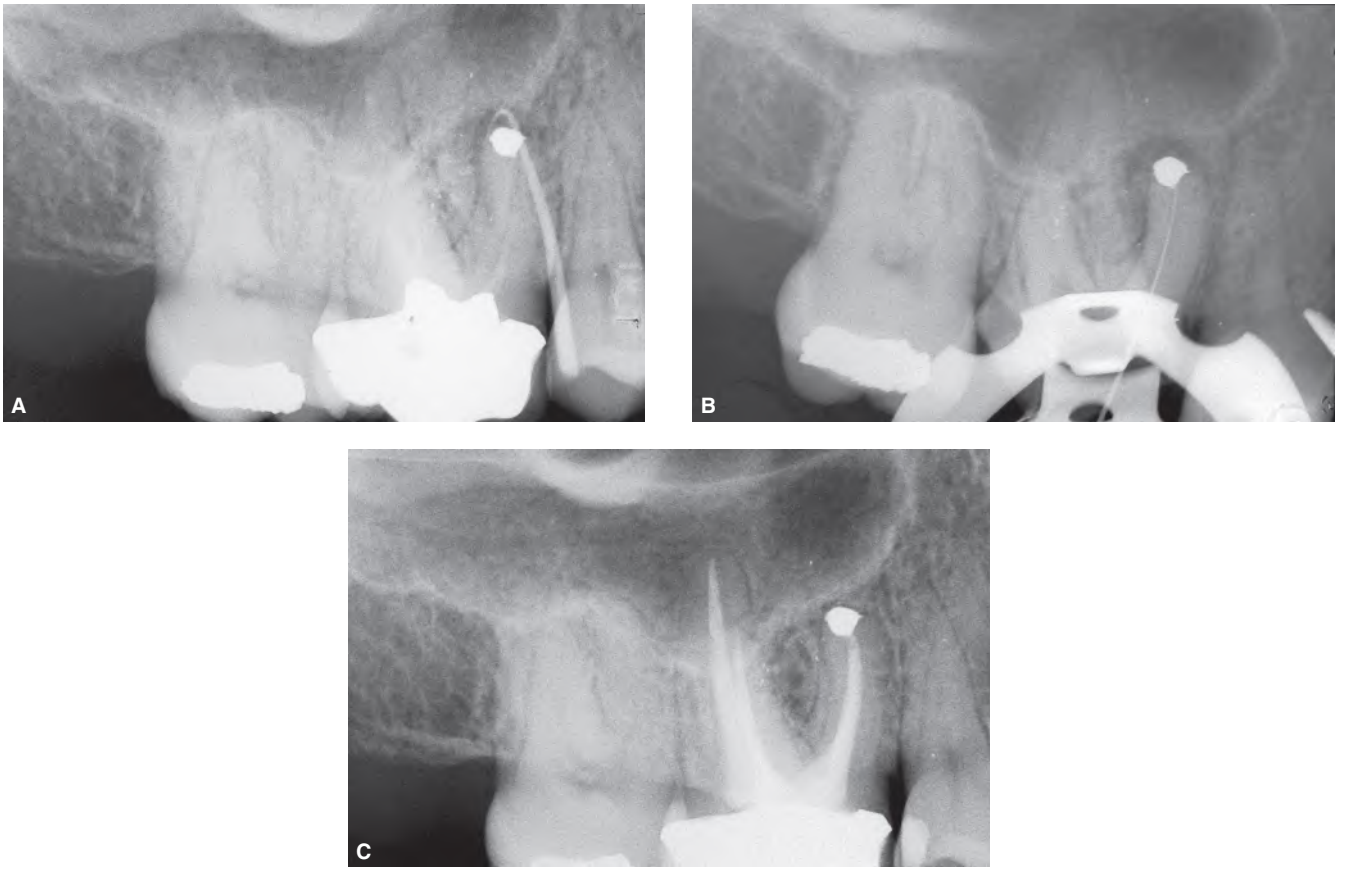

FIGURE 22-83 Nonsurgical retreatment of a failed endodontic surgery. ***A***. Preoperative radiograph of a maxillary right first molar. Gutta-percha cone indicates the presence of a sinus tract. ***B***. Intraoperative radiograph. The obturation of the MB-1 and MB-2 canals was done using thermoplastic gutta-percha. The previous amalgam root-end filling served as apical stop. Careful execution will not push filling material to the periapical tissues. ***C***. Two-year recall radiograph showing therapy has been successful.

FIGURE 22-84 The Ultrafil syringe (Coltene, Whaledent, Cuyahoga Falls, Ohio, U.S.A.).

Obtura. The temperature varies from 150°C to 230°C, and the needles are of different sizes: 20-, 23-, or 25-gauge. Like the Obtura, it can also be used with Resilon pellets (see later in this chapter). Lower temperatures are required to thermally soften Resilon.

Other high-temperature thermoplastcized gutta-percha systems are currently available; Elements System (Sybron Endo, Orange, California) (Figure 22-65C) and the Calamus System (Tulsa Dental Products, Tulsa, Oklahoma) (Figure 22-65D). These units use preloaded gutta-percha cartridges, and the heated material is extruded by an activation button.

In the author's opinion, these units have a few disadvantages when compared to the use of syringes like Obtura or B&L Beta 2. For example:

- The cartridges are more expensive compared to the simple gutta-percha pellets.
- The operator needs to electrically extrude the piston from the used cartrige, insert a new cartrige, and wait unit the material is ready for use. This is more time consuming.

The Ultrafil (Hygienic Corp., Akron, Ohio) is a low-temperature thermoplasticized gutta-percha delivery system that contains prepackaged cannulas with attached 22-gauge needles. The gutta-percha is prepared in the alpha phase

form so that it softens in the heating unit at temperatures between 70°C to 90°C. According to Lipski,[212] the temperature transmitted to the periodontal ligament with the Ultrafil system is below the level that can cause damage or injury.

THERMOMECHANICAL COMPACTION OF GUTTA-PERCHA

This technique was first introduced by McSpadden about 40 years ago. In this technique a new instrument was used to condense the gutta-percha—the McSpadden compactor. It resembled a reverse Hedstrom file, having the blades turned towards the tip instead of towards the shaft. The compactor, mounted on a 1:1 contra-angled handpiece, was inserted in the canal next to a gutta-percha cone and rotated at up to 20,000 rpm. Heat generated by friction softened the gutta-percha and the blades pushed the material apically. This allowed the adaptation of the obturation material three dimentially to the canal walls.[213–217]

Later, McSpadden developed a new technique, combining the principle of the thermomechanical compaction with new materials. The previous compactor was replaced with Nickel Titanium Condensers, of different diameters and tapers, and with "alpha phase" gutta-percha. This gutta-percha was preplasticized in a MicroSeal heater and then introduced into the canal with a rotating instrument, at lower speeds (1,000 rpm to 4,000 rpm) than the original McSpadden Compactor. This system uses MicroSeal gutta-percha master cones and a specially formulated Alpha Phase gutta-percha (low-fusing or ultra-low-fusing) in a cartridge, that is heated in the MicroSeal heater. The NiTi condenser is coated with heated gutta-percha and inserted into the canal next to the master cone. The rotating instrument generates heat from the friction that, in turn, thermally softens the master cone. It also flows laterally, by centrifugal force, into all areas of the canal.[218] The material is therefore pushed both apically and laterally, providing a three-dimensional obturation of the root canal system.

The most important characteristics of this technique are:[205,219–221]

- Lower point of fusion of the gutta-percha
- Longer working time
- Less shrinkage during cooling
- High fluidity of the gutta-percha allowing for a good adaptation of the material to the canal complexities.[222]

On the other hand, it requires good apical control of the obturating material.

CARRIER-BASED GUTTA-PERCHA

The Thermafil obturation technique was developed by W.B. Johnson in 1978.[223] Originally, the first Thermafil obturators were stainless steel K-files covered with a uniform layer of gutta-percha. The obturators were then heated and placed in the canal, already coated with sealer, and sectioned at the level of the pulp floor. The final obturation was characterized by the presence of a stainless steel instrument surrounded by gutta-percha and sealer, with consequent difficulties in retreatments, preparation of a post space or surgical root-end filling. Later, the Thermafil obturators were modified and the K-file was replaced by a grooved plastic carrier made from a biocompatible radio-opaque plastic. The purpose of the groove was to increase flexibility and facilitate retreatment.

The product is marketed today in different versions based on the size and shape of the root canal system.[224] Classic Thermafil obturators were available in 17 sizes with tip diameter ranging from 0.20 to 1.40 mm and taper that varied between .04 and .05. Later, every rotary shaping system has introduced different obturators, such as GT and GTX obturators (Tulsa Dental Products, Tulsa, OK, U.S.A.), designed to be in conjunction with GT or GTX rotary files; ProTaper obturators (Tulsa Dental Products), to be used in conjunction with ProTaper Universal rotary files; WaveOne obturators (Dentsply Maillefer, Ballaigues, Switzerland), and similar systems.

Recently, the plastic carrier was replaced with a gutta-percha carrier and the obturator is now called GuttaCore (Dentsply Tulsa Dental, Tulsa, Oklahoma). This product has the carrier made from a cross linked gutta-percha that is intimately adherent to the surrounding gutta-percha. The advantage is eliminating the existing gap between the carrier and the gutta-percha. GuttaCore obturators are available to be used in conjunction with ProTaper Next and ProTaper Gold.

Thermafil Gutta-percha

The Thermafil gutta-percha covers the carrier for approximately 16 mm overextending the tip of the core by about 1 mm. It is hard and friable in the solid state. When heated, it becomes sticky, shiny, and expanded, assuming excellent adhesive and flow characteristics.[225,226] Because of its properties, in particular its low viscosity, Thermafil gutta-percha was initially considered as Alpha gutta-percha (compared to the Beta gutta-percha of conventional cones), elastic and malleable in the solid state but more viscose and less adhesive once thermoplasticized.[219,221] However, fusion temperature analysis[221] for Thermafil gutta-percha shows a fusion temperature of about 56°C, that is 14°C less than the fusion temperature (70°C) required from Alpha gutta-percha.[219,221] Furthermore, once plasticized, Thermafil gutta-percha resolidifies after 90 seconds, reassuming the initial characteristics of friability and hardness. However, if reheated again, it does not show any changes in physical properties. It therefore appears then that Thermafil gutta-percha is not a stereo isomeric crystalline Alpha form but has the physical behaviour of the Alpha type.[224] Due to its low viscosity, Thermafil gutta-percha has a better capacity to penetrate dentinal tubules (Figure 22-85).[226] It should be noted that the clinical significance of obturating dentinal tubules with gutta-percha has not yet been demonstrated. It may play a role in blocking the ingress of microorganisms and their byproducts[227] and

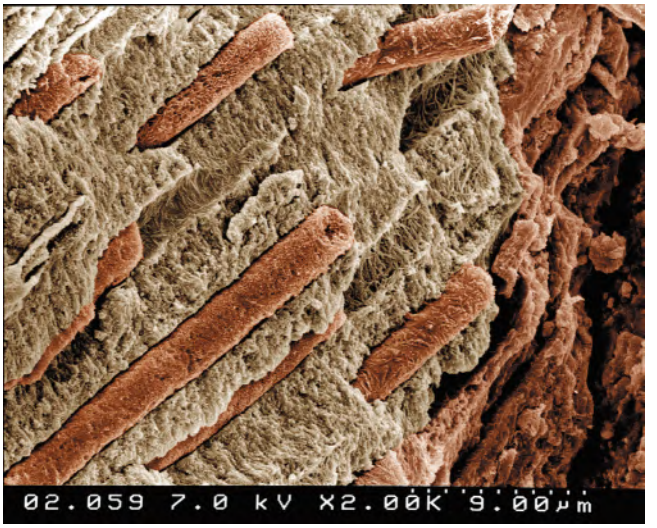

FIGURE 22-85 Scanning electron microscopy analysis of extracted teeth obturated with Thermafil without sealer (×1000). Penetration of Thermafil gutta-percha into the dentinal tubules is evident. (Courtesy of Dr. Giuseppe Cantatore, Rome, Italy.)

possibly improve the seal of the obturation. However, this remains to be further investigated.

Size Verifiers

The best method to select the correct size of Thermafil is to remove the gutta-percha from the plastic carrier and check the plastic carrier itself inside the root canal. The plastic carrier that stops about 1 mm short of the working length should be selected and checked radiographically. However, this is not possible when using the new GuttaCore system, since the gutta-percha carrier is intimately adhered to the surrounding gutta-percha so that it cannot be removed. In such cases, a size verifier is used. This comprises of a nickel titanium instrument, similar to a hand Profile, with a non-cutting tip and radial lands.[224]

The Oven

The Thermaprep oven enables one to thermoplasticize two Thermafil obturators in a few seconds (Figure 22-86). It requires inserting the obturators in the necessary inserts, selecting the diameter of the obturator and initiation of the heating process. Consequently, the thermoplasticized gutta-percha is ready for use in a few seconds. Once the obturators have been removed from the oven, the operator should check if the gutta-percha is ready for use by appearing expanded, shiny and sticky. If not, the heating cycle should be repeated.[224]

Blum et al.[228] studied the condensation forces that develop during Thermafil obturation and found that they were lower than those produced during lateral condensation, vertical condensation and System-B. Dulak et al.[229] and Goldberg et al.[230] compared Thermafil obturation with other techniques regarding its capacity to obturate lateral canals and found that Thermafil was equal to vertical condensation and System-B, but significantly superior to lateral condensation, warm lateral condensation and Obtura.

FIGURE 22-86 The heater for thermoplastisizing Thermafil obturators (Dentsply Tulsa Dental Specialties, Tulsa, Oklahoma, U.S.A.).

The relationship between gutta-percha, sealer, carrier, and the canal walls in Thermafil obturation has been the subject of numerous "morphological" studies.[191,231–238] The results of these studies show that the Thermafil technique has a good ability to completely adapt to the canal walls, comparable to the Obtura II injectable technique and superior to the lateral condensation.[191] Coronal micro-infiltration tests indicated that Thermafil behave better than cold lateral condensation, especially in the short term. After four months the two techniques tend to show the same results.[239–241] Other studies suggested that Thermafil provided a better seal than laterally condensed gutta-percha and was similar to vertical compaction (Figure 22-87).[241,242]

Regarding apical leakage, the following should be noted:

- Thermafil obturation, notwithstanding the excellent characteristics of its gutta-percha, requires sealer, otherwise a drastic increase in microleakage results.[224] The role of the sealer is twofold: (1) to lubricate the canal walls aiding the flow of the gutta-percha, that otherwise tends to adhere to the dentin, and (2) to compensate for the cooling contraction of the gutta-percha.[191,243,244]
- Thermafil obturation appears to exhibit superior results when compared to laterally condensed gutta-percha.[238,245–251]
- Compared to warm vertical condensation and System-B, the Thermafil technique behaves equally well; the difference between the three methods was not found to be statistically significant.[252–254]
- Temperatures produced by heated carrier-based gutta-percha were found to be at safe levels and did not cause damage to bone and periodontal ligament.[255,256]

Thermafil obturation is best indicated in narrow, curved canals where spreaders, heat carriers, and pluggers are difficult, or impossibile, to use in the apical one-third. On the other hand, the technique should not be used in cases of large, short root canals, open apices, bi- or tri-forcations of the middle and apical thirds of the root canal, internal resorption or anatomical variations like C-shaped canals.

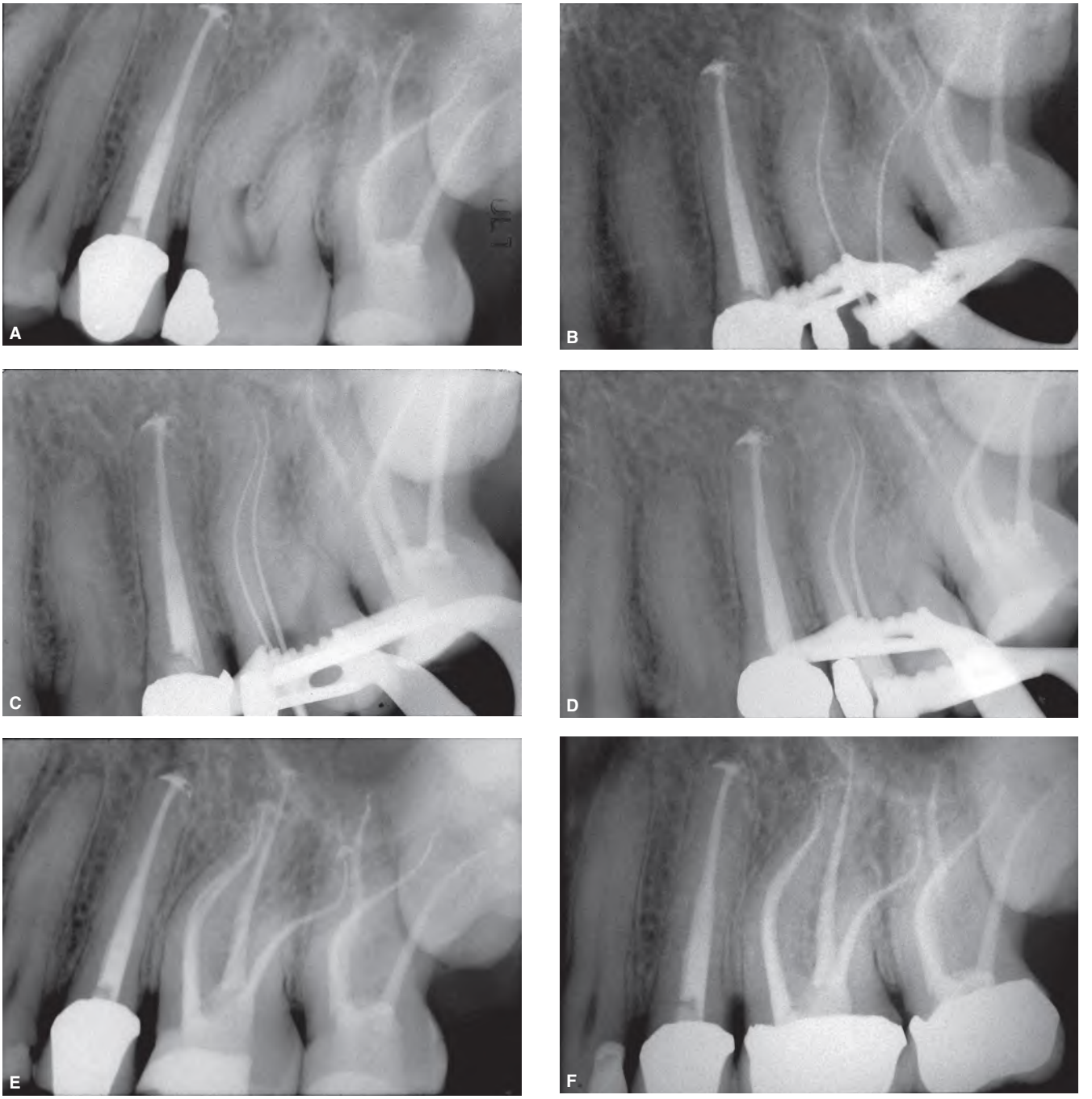

FIGURE 22-87 ***A***. Preoperative radiograph of the maxillary left first molar. The second molar has already been obturated with Thermafil. ***B***. Working length of the MB-1 and DB canals. ***C***. Working length of the MB-1 and MB-2 canal. ***D***. Plastic carriers in the MB-1 and MB-2. ***E***. Postoperative radiograph. ***F***. Two-year recall radiograph showing that the therapy has been successful. (Courtesy of Dr. Giuseppe Cantatore, Rome, Italy.)

SYSTEM B TECHNIQUE

The System B technique or "single wave of condensation technique" is an aditional heat-assisted gutta-percha compaction technique introduced by Buchanan in 1996.[257] It can be considered a variation of the Schilder technique, where instead of using a heat carrier and cold cylindrical pluggers only one tapered heated plugger is used during the down-packing. The hydraulics that develop here are higher then in the Schilder technique because of the wedging effect produced by the heated tapered plugger introduced in the gutta-percha cone in the tapered preparation of the root canal.

Pommel and Camps[254] studied, *in vitro*, the apical leakage of this technique and found that System B was as effective as vertical condensation and Thermafil in resisting apical leakage.

After one month, System B, Thermafil and vertical condensation showed less leakage than lateral condensation and single cone technique. Jacobson et al.[258] evaluated the coronal bacterial leakage in teeth obturated with the System B continuous wave of condensation technique and compared it to Obtura II backfill, and lateral condensation technique. The results indicated that microbial coronal leakage occurs more quickly when using lateral condensation than when the System B continuous wave of condensation technique and Obtura II backfill were used.

Goldberg et al.,[259] using simulated lateral canals, found that a greater number of lateral canals were obturated using Ultrafil, Thermafil and System B + Obtura II in comparison to the hybrid technique, Obtura II or lateral compaction of gutta-percha. The difference was statistically significant.

Romero et al.[260] studied the heat transfer to the periodontal ligament during root obturation. They found that the "Continuous Wave Technique," when performed with a controllable heat source, such as System B, causes only negligible temperature increases in the PDL. Guess et al.[261] studied the effect of System B plugger depth on gutta-percha filling adaptation to the canal walls using the continuous wave technique, and found that the best results were obtained when pluggers were used at a depth of 3 mm to 4.5 mm from the working length.

Lea et al.[262] compared quantitatively the density of standard cold lateral gutta-percha compaction and warm vertical compaction using the continuous wave of condensation technique. They concluded that warm vertical compaction, using the System B, resulted in a greater gutta-percha fill by weight, as well as a significantly greater density compared to standard cold lateral gutta-percha compaction.

Galvao Barbosa et al.[263] compared the frequency, location, and direction of accessory canals filled with the Schilder technique and with System B and didn't find any difference between the two filling techniques.

CHEMICAL SOFTENING OF GUTTA-PERCHA

This technique was described by Callahan and Johnston at the beginning of the last century. It used chloroform to chemically soften gutta-percha. The technique is still practiced today with various types of chloroform sealers. Gutta-percha particles are added to chloroform to produce a sealer (chloropercha), that has the same color as gutta-percha. The mixture can then be used as a sealer with gutta-percha cones for obturation of the canal. There is more shrinkage with the chloroform solvent techniques and this often translates into leakage, with the material pulling away from the canal walls as it shrinks thereby creating voids.[266]

The technique has several drawbacks:

1. When the chloroform evaporates, the material undergoes a significant shrinkage, compromising the long-term apical seal.[267] If the root canal is filled with chloropercha alone, two thirds of the material will be lost once evaporation of the chloroform has occurred.[268]
2. The operator must be very careful to avoid overfilling, because of the reported tissue toxicity of chloroform.[110]

Studies by the American Food and Drug Administration[269,270] have demonstrated that chloroform is potentially carcinogenic. Consequently, the Council on Dental Therapeutics of the American Dental Association[107] has decided to withdraw chloroform from *Accepted Dental Therapeutics*. Chloroform can be substituted with eucalyptol,[271] an organic solvent derived from eucalyptus trees. Its tissue toxicity is much lower than chloroform, and it is used in medicine as a decongestant and rubefacient.[21] Eucalyptol was also reported to possess antibacterial action and antiinflammatory properties.[272]

TECHNIQUE OF WARM LATERAL CONDENSATION: THE ENDOTEC

This technique was introduced by Martin in 1987.[273] It uses a special spreader whose tip is electrically heated transmitting heat to the gutta-percha while laterally condensing (Figure 22-88). While the lateral condensation technique is based on pressure that only causes a stratification of the gutta-percha cones, the Endotec system permits the coalescense and fusion of the various cones into a dense, homogeneous mass of gutta-percha,[274–276] with less stress on the dentinal walls.[277] This is a significantly simpler technique to execute.[278]

To date, there are insufficient experimental studies on this technique. Kersten[279] found that the lateral condensation technique appeared to be markedly improved by use of the Endotec. Luccy et al.[280] have claimed positive results following *in vitro* experimentation, as well as Castelli et al.[281] and Guldener.[282]

More recently, the instrument has been modified.[283] The Endotec II is a new, advanced, endodontic condenser incorporating several clinical benefits. The resistor has been placed within the body of the handle, allowing for cost reduction of the condenser tip. The heating element temperature has been raised to ensure proper thermosoftening of the gutta-percha.

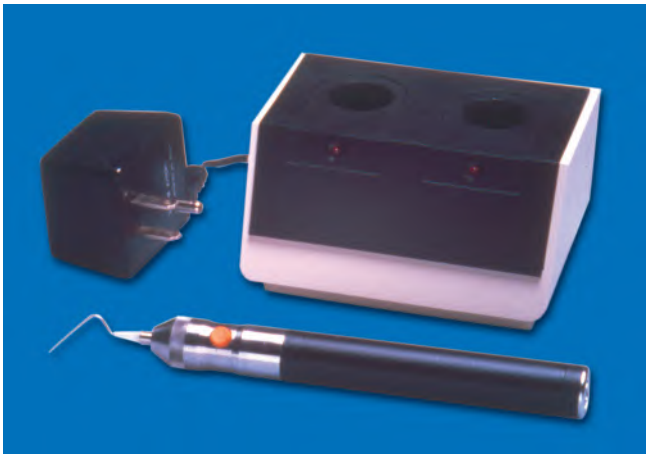

FIGURE 22-88 The Endotec unit (Medidenta, Woodside, New York, U.S.A.).

The quick-change condenser tips are now autoclavable, and can provide an angle of canal insertion ranging from 90° to 180°. Furthermore, charged by two AA batteries inserted into the handle, the unit does not require constant recharging. In the author's opinion, the Endotec II system is a marked improvement allowing for predictable softening of the gutta-percha with heat and subjecting the mass to vertical or lateral pressure.

THE ENDOTWINN

Recently, a new device has been developed for both warm lateral and vertical condensation, the EndoTwinn (MDCL N.V. Corporation, Amsterdam, The Netherlands) (Figure 22-89). This device combines application of heat and vibration. Like the Endotec, the EndoTwinn is a hand-held, self-contained, heat-carrying instrument with spreader or plugger tips. Sonic vibrations are also incorporated to augment the compaction and obturation effectiveness of EndoTwinn's heated tips.[284]

The EndoTwinn system comes with one rechargeable cordless handpiece and several pluggers for compacting, softening and cutting gutta-percha cones. The pluggers are available in the standard series with 0.5 mm diameter and a range of sizes F, FM, ML, and L, and in the Ultrasoft series, that can be easily pre-bent before entering the root canal. For narrow and curved canals, another tip is available with a 0.3 mm tip diameter and .04 taper. The kit also includes a "tip spoon" specifically designed for cutting plastic carriers.

The EndoTwinn tips can be used with and without vibrations. The combination of heat and vibration seems to provide better results. Presumably, the low vibration, in combination with heat, provides the gutta-percha better flow properties. Several studies have reported, that when using this feature, the average percentage of gutta-percha in the canal space can increase significantly, especially in narrow tapered canals.[285–287]

In early 2007, efforts to improve and refine the EndoTwinn led to the introduction of the DownPak (Hu-Friedy, Chicago, Illinois). The DownPak system enables the clinician to employ variable temperature settings and to turn the vibration feature on or off.[288] The variable temperature settings are useful when different obturation materials are used. For example, Resilon is softened at a lower temperature than gutta-percha. The DownPak is cordless and light weight, with an ergonomically balanced hand-held grasp; all of the switches and adjustments are easily accessible on the handle.[284]

RESILON

Resilon obturating points (Pentron Clinical Technologies, Wallingford, Connecticut) and the resin sealer Epiphany or RealSeal (Sybron Endo) have been proposed to be used in combination with a self etch primer after smear layer removal, to allow creation of a solid "monoblock" (a material that is contiguous from its resin tags in cleared dentinal tubules through sealer to the core canal filler).[289] The material seems to fully obturate canals with anatomical variations (especially through the compaction of warm obturation techniques). Resilon Products (RP) are marketed as RealSealTM (SybronEndo, Orange, California), and Epiphany (Pentron, Wallingford, Connecticut) (Figure 22-90).

Resilon is a polycaprolactone core material with difunctional methacrylate resin, bioactive glass, and radio opaque fillers.[86] The polyester core material is available in ISO-sized points with accessory points for lateral compaction and warm vertical techniques, or pellets for use in thermplasticized techniques.[91]

The RealSeal points handle and feel like gutta-percha. It can be used with all current obturation techniques (vertical compaction of warm gutta-percha, cold lateral condensation, lateral/vertical combinations). This allows the clinician to use this new technology with only two added steps relative to common endodontic treatment regimens, clearing the smear layer and placing the self etch primer. The only difference between RealSeal and gutta-percha is that the temperatures used in the thermoplasticized techniques are lower than

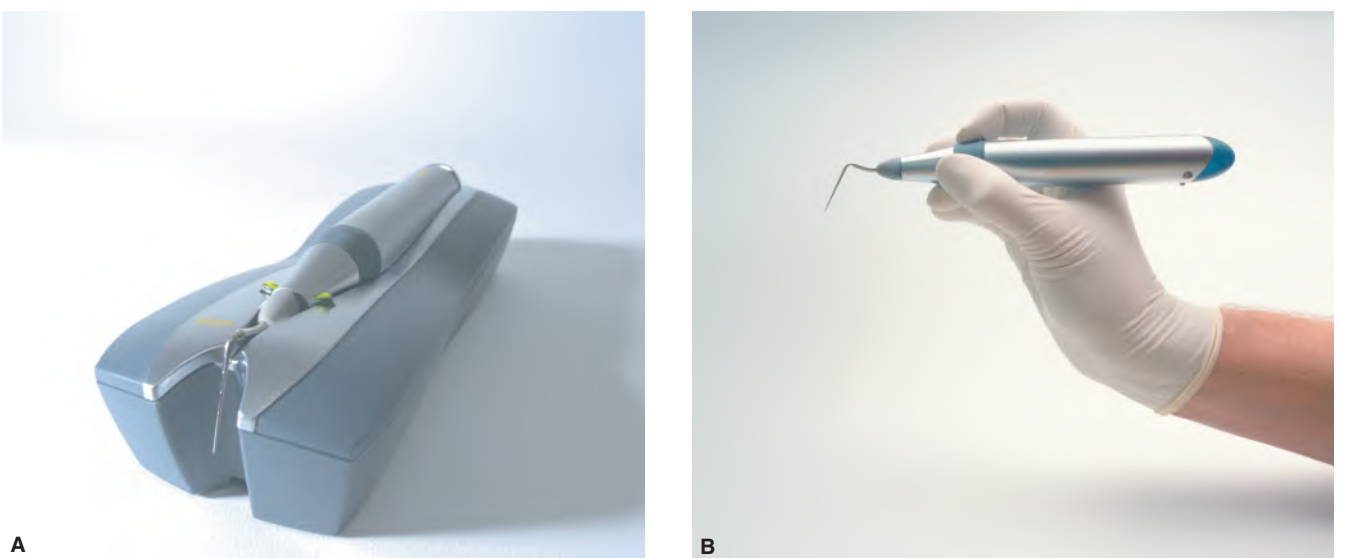

FIGURE 22-89 ***A***. The EndoTwinn system (MDCL N.V. Corporation, Amsterdam, The Netherlands). ***B***. The EndoTwinn handpiece.

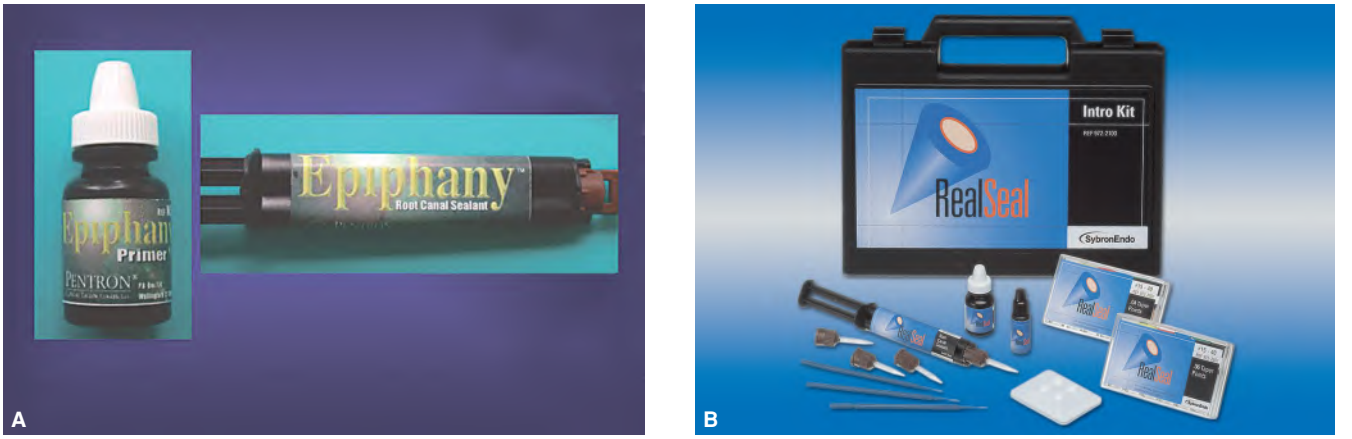

FIGURE 22-90 ***A***. Resilon™ and Epiphany Sealer™ (R/E) (Resilon Research LLC, Madison, CT, U.S.A.). ***B***. RealSeal™ (R/S) (SybronEndo, Orange, California, U.S.A.).

those used for gutta-percha techniques.[289] RealSeal is also amenable for retreatment.

Gutta-percha has been used for decades as the preferred root filling material for endodontic obturation due to its advantages of being nontoxic, biocompatible, thermoplastic, and retreatable. Gutta-percha alone, however, cannot prevent coronal microleakage and it does not bond to canal walls.[289–293] Coronal seal plays a critical role for endodontic success.[294,295] Shipper et al.[296] reported that Resilon showed minimal leakage. The sealing ability of Resilon and Epiphany sealer versus gutta-percha and AH Plus sealer were studied by Stratton et al.[297] and by Von Fraunhofer et al.,[298] who found significantly less leakage when using Resilon.

With RealSeal, after removal of the smear layer, produced during instrumentation, it may be possible to bond the obturating material to the dentinal tubules and create a "monoblock" of resin sealer and resin core filling material (Figure 22-91).[289]

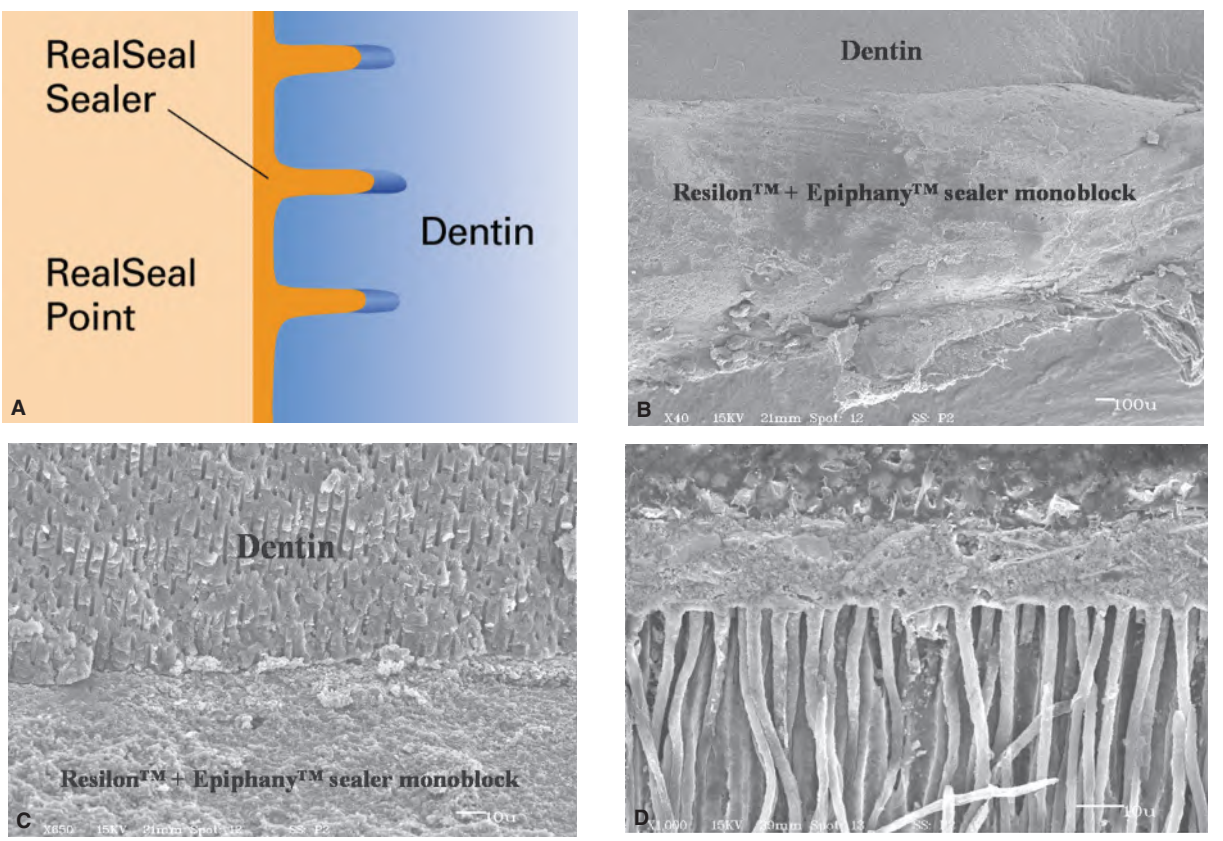

FIGURE 22-91 ***A***. Graphic illustration of Resilon sealer penetration and Resilon point creating a monoblock of resin. ***B***. Resilon "monoblock" (×40). ***C***. Resilon "monoblock" (×650). ***D***. Sealer tags and Resilon (original magnification ×1,000). (Courtesy of Drs. Rich Mounce and Gary Glassman, Toronto, Canada, U.S.A.)

RealSeal points are available in kits of various configurations and as individual components (.02, .04, .06 tapered cones with a variety of tip sizes along with accessory points ranging in size from x-fine to large). RealSeal cones are very flexible and pellets of the material are available for the Obtura gun (Spartan Obtura, Fenton, Missouri) or the B&L Beta 2.

Canal cleaning actions can remove certain amounts of dentin. If excessively removed, it may weaken the root, making it more susceptible to root fracture. Gutta-percha exerts no strengthening action to the root after obturation. It is claimed that RealSeal may have the potential to do so. Teixeira, et al.[299] found that the resistance to root fracture with Resilon was superior ($P = 0.037$) to gutta-percha/AH 26 sealer using either lateral or vertical condensation. On the other hand, another study compared the cohesive strength and stiffness of Resilon to gutta-percha, under dry conditions also after one month of water storage. It was reported that both Resilon and gutta-percha had relatively low cohesive strength and module of elasticity and did not have enough stiffness to reinforce the root after endodontic therapy.[300]

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Chapter 23

Endodontic Orthograde Retreatment and Management of Mishaps

Pierre Machtou, Clifford J. Ruddle

INTRODUCTION

Longitudinal endodontic outcome studies performed in university-based institutions or by specialists have shown a high percentage of success estimated from 90% to 95%.[1] Assuming that 90% of the treatments are successful over time, the reciprocal failure rate is 10%. In the United States alone, where the number of canals treated per year now exceeds 50 million,[2] a 10% failure rate could represent five million treatment failures per year. Extrapolating these numbers over the past three to four decades reveals that the number of failing endodontically treated teeth is very high and could approach tens of millions.[3]

Such a high number of failed root canal treated teeth has been corroborated by Figdor in Australia and Sweden.[4] Furthermore, when root canal treatment is performed in a general practice, cross-sectional studies conducted in different countries show a high and alarming prevalence of endodontic treatment failures, as evidenced by the presence of apical periodontitis in a range from 30% to 65% of cases[5] (Figure 23-1). A recent systematic review and meta-analysis, including 33 cross-sectional studies conducted in several countries in the world, show that the prevalence of apical periodontitis on 28,881 endodontically treated teeth was 36%.[6] As a result, it appears that there is a high demand for retreatment of failing cases.

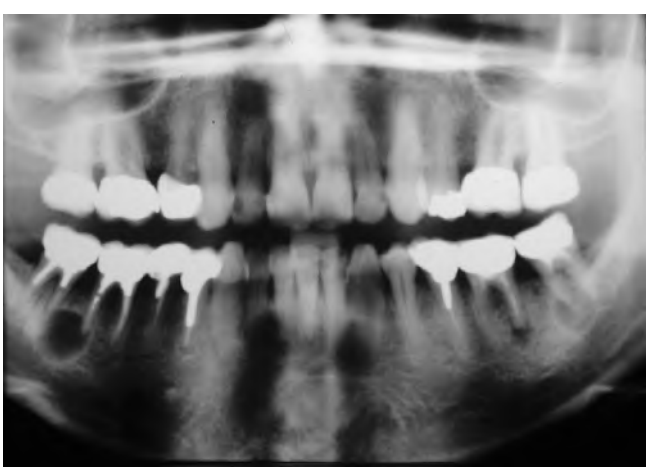


FIGURE 23-1 Treatments performed in general practice show a high prevalence of endodontic failures.

Retreatment Definition

The Glossary of Endodontic Terms published by the American Asssociation of Endodontists[7] gives the following definition: "Retreatment (revision): A procedure to remove root canal filling materials from the tooth followed by cleaning, shaping, and obturating the canals." Such a definition seems to be oversimplistic and restrictive, because in many situations, retreatment is indicated when there is no root canal filling to remove. Such is the case when a failure results from a missed canal during initial treatment. Oftentimes, the previous dentist was unaware or unable to locate one or more canals, or refers a patient during active treatment. A more precise definition involving all situations was needed.

Carr[8] defined endodontic retreatment as a procedure that is performed on a tooth that previously has had attempted defintive treatment and now requires further treatment to ensure a successful result. It is noteworthy that the term, "revision," has been substituted for retreatment in order to rule out a pejorative connotation that implies a dentist made a mistake during the initial treatment. However, "revision" is not appropriate, since retreatment is directed toward addressing deficiencies in initial treatment, as opposed to, for example, performing elective facial plastic surgery where periodic revisions may be considered as one ages.

Retreatment Vs. Initial Treatment

In terms of objectives, there is no difference between initial treatment and retreatment. In both situations, microorganisms must be eradicated from the root canal system through shaping canals and 3-D cleaning, filling, and sealing the endodontic space (Figure 23-2). It must be appreciated that, in both straightforward and complex initial treatment cases, all root canal systems are patent and anatomic complexities can, in most instances, be overcome by a skillful practitioner. In contrast, the original canal patency is oftentimes lost or severely compromised in the retreatment situation. Further, most of the teeth to be retreated have already been restored, further compounding the retreatment challenges. The radicular obstacles that obstruct canals include different types of filling materials, posts, and fractured instruments (Figure 23-3). Additionally, other formidable radicular challenges include blocks, ledges, canal transportations, and root perforations. Recognizing the scope of so many retreatment challenges, Schilder[9] introduced a new term in 1986, "Retreatodontics,"

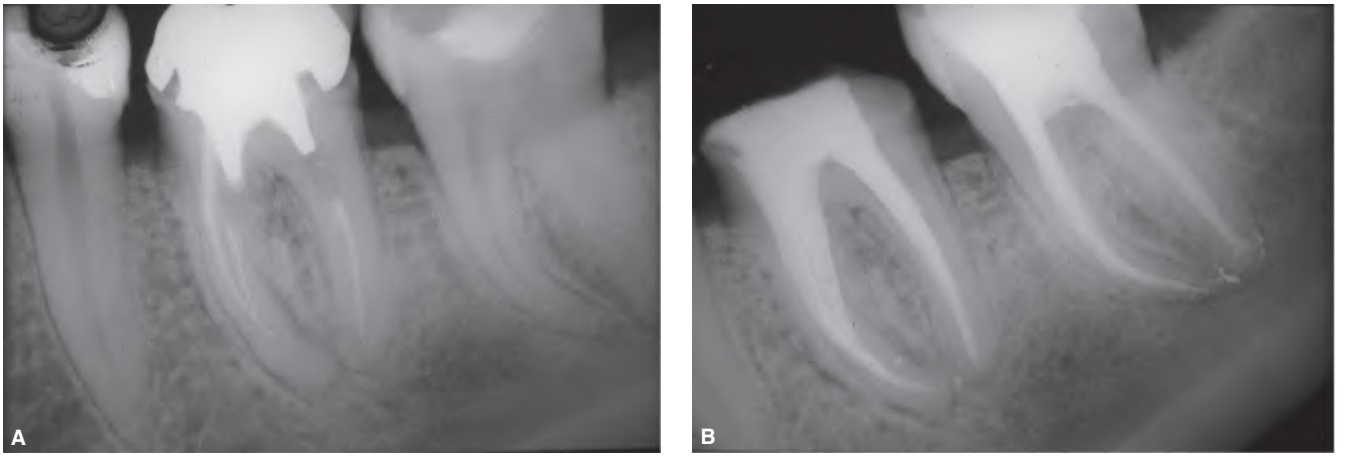


FIGURE 23-2 ***A.*** Retreatment is indicated on the mandibular first molar. In contast to the second molar, many coronal and intracanal obstacles are present. ***B.*** A post-operative radiograph demonstrates the two treatments completed.

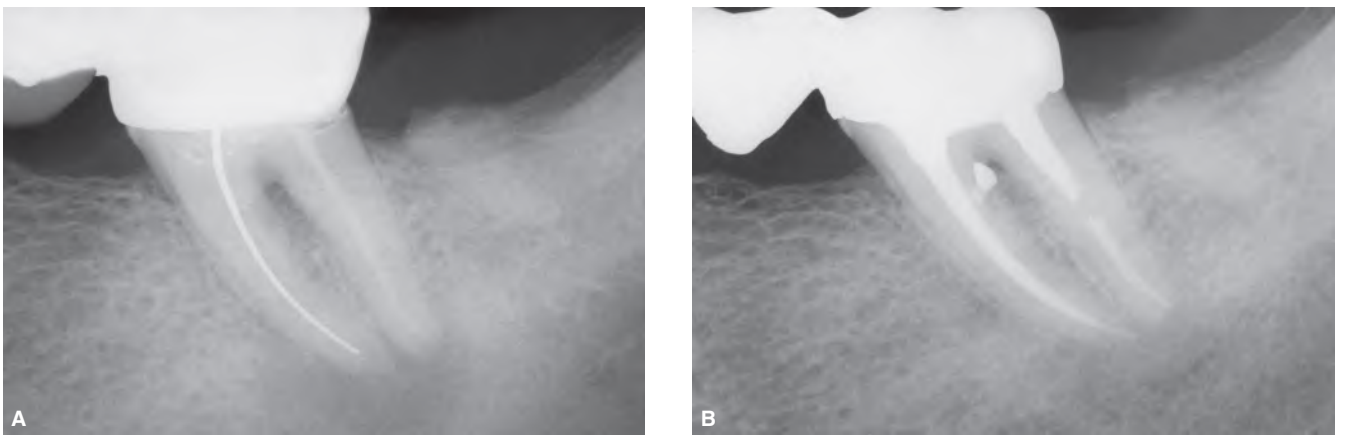


FIGURE 23-3 ***A.*** A radiograph of a mandibular left second molar reveals a poorly-fitting bridge, canals holding a silver point and paste, and evidence of furcal and apical pathoses. ***B.*** Ten-year recall radiograph shows the importance of three-dimensional endodontics and restorative excellence.

to emphasize the complexity and specificity of endodontic retreatment and he predicted that the future of endodontics would be largely focused on retreating failing cases.

Decision Making

Failure of endodontic therapy is usually based on the observation of clinical symptoms and/or radiographic findings. However, the presence of apical periodontitis in previously endodontically treated teeth is rarely associated with clinical symptoms. Therefore, lesions of endodontic origin are often discovered during routine radiographic examination. It has been found that the diagnosis of a periapical lesion was not a sufficient reason to retreat. Amazingly, a retreatment decision was only made in 39% of the cases,[10] even if painful symptoms were present.[11] Many studies have shown that radiographic interpretation of periapical changes can be subjective and large discrepancies in attitudes toward endodontic retreatment have been observed.[10–14] For example, when radiographs were shown to general dentists and specialists from different fields of dentistry to assess the periapical health status, no agreement was found.[15] Furthermore, when the same radiographs were re-examined six to eight months later by the same practitioners, about 20% of them disagreed with their former opinions.[16]

These discrepancies can be influenced by a variety of factors and variables, including the training and experience of the examiner, the conditions under which the radiograph was viewed, and the type and settings of the x-ray system used. Similar results were also obtained when digital radiography was used.[17] Paralleling the importance of utilizing the dental operating microscope is the increasing adoption of cone-beam computed tomography imaging (CBCT) in endodontics. CBCT is a diagnostic adjunct, providing three-dimensional (3-D) images, a low radiation dose, and good resolution for endodontic treatment planning. The possibility to navigate through axial and coronal slices allows the clinician to detect hidden periapical lesions, root curvatures, missed canals, and to identify the location and extent of resorptive and perforation defects and vertical root fractures (Figure 23-4).[18] For more details, see Chapters 9C and 10.

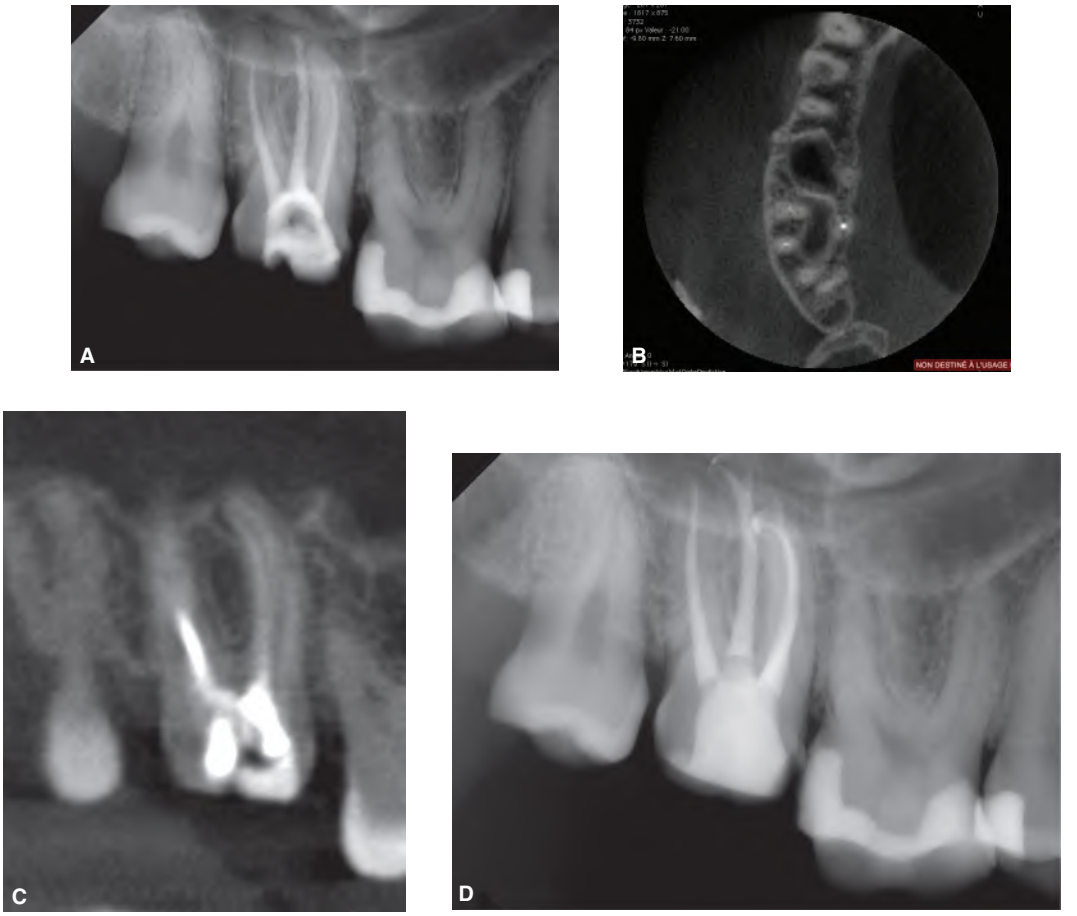


FIGURE 23-4 ***A.*** Patient was referred because of persistent pain in the right second molar after endodontic treatment. Clinical examination and radiography were non contributory. ***B.*** A missed MB-2 canal was detected on an axial slice from a CBCT examination. ***C.*** Coronal image showing the presence of a periapical lesion in the mesial root in relation to the untreated MB-2 canal. ***D.*** Postoperative radiograph showing the case after retreatment. (Courtesy of Dr. François Bronnec, Paris, France.)

The Praxis Concept

The decision-making process is a complex task for a practitioner, who may be faced with 30 different clinical situations combining different qualities of the seal, the presence of a post, and the locations and sizes of different lesions of endodontic origin (Figure 23-5). A multiplicity of factors complicate the decision-making process, including diagnostic variation,[19] risk assessment,[20] clinical context,[12,14,21] cognitive factors,[20] and treatment plans.[22]

To explain the behavior of dentists to perform endodontic retreatment, Kvist and his team developed the Praxis concept model.[23] In this model, periapical health and disease are not viewed as two different situations. The periapical status is perceived as a continuum of health ranging from no lesion (perfect health) to a large lesion (high degree of poor health). Yet, the periapical status is subjective, based on the attitudes of the dentist. Rather, the periapical status should be based on objective criteria, including clinical and radiographic findings and the risks vs. benefits of retreatment. However, the decision to initiate retreatment is, in fact, influenced by factors unrelated to the disease *per se*, including successfully managing the technical challenges, potential for pain, and costs (Figure 23-6).

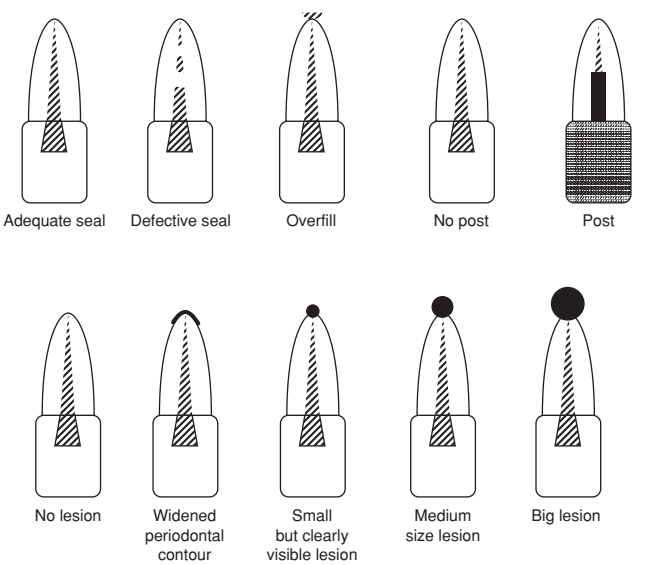


FIGURE 23-5 Different clinical situations may be encountered making the decision to retreat difficult. (Reprinted with permission from Kvist et al.[23])

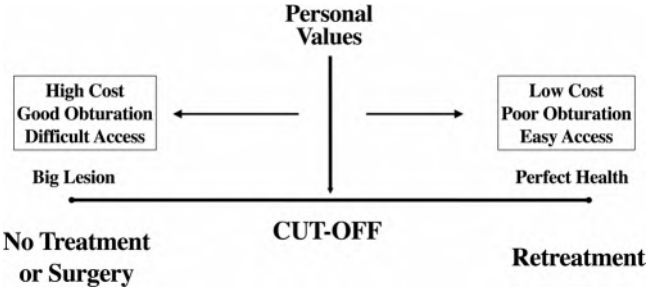


FIGURE 23-6 The Praxis model concept. (Reprinted with permission from Reit and Gröndahl.[13])

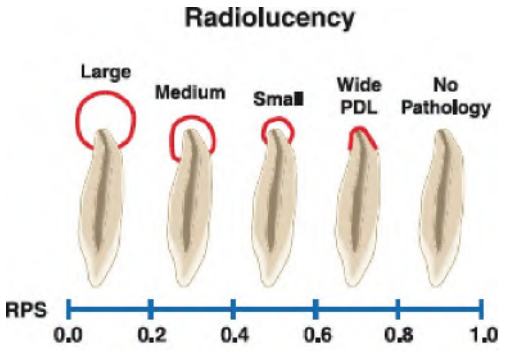


FIGURE 23-7 Calculation of the Retreatment Preference Score (RPS). Clinicians assessed five periapical conditions and chose a periapical situation as their cut-off point for retreatment. The mean score chosen was very close to medium-sized lesion (RPS = 0.38). (Adapted from Kvist and Reit[24] and reprinted from Ingle's Endodontics 6th ed., Chapter 31: Treatment of non healing endodontic therapy and management of mishaps. Hamilton, ON: BC Decker; 2008:1091.)

Two main types of tendencies have been found among dentists. The first tendency is to retreat when a periapical lesion is present, especially when the lesion is deemed large. Yet, the decision to retreat is oftentimes modified by individual values. As an example, graduate students at three universities used a "retreatment preference score" (RPS) to numerically quantify the size of a lesion. In this RPS scale, 0.0 represents a large lesion, whereas 1.0 represents no lesion. Ironically, students generally quantified the size of a lesion as 0.3 (Figure 23-7).[23] A similar study conducted with endodontic specialists showed they followed the same behavioral pattern as the "novices."[24,25]

Decision Analysis

From an analytic point of view, the RPS can be structured as a decision tree, illustrating the various clinical options and providing a rationale basis for thinking and making the right decision.[13] Moreover, this method serves as a way to present the different treatment options in order for the patient to be invoved in the decision-making process.

The retreatment decision-making process should be seen as a 3 "**D**" process.[21,26,27]

Decide if the Endodontically Treated Tooth is a Failure Two situations are encountered regarding the presence or absence of a lesion.

A. The first situation corresponds to a tooth with technical deficiencies in primary treatment, yet there are no clinical signs or symptoms or radiographic evidence of failure. The absence of a lesion is not always synonymous with an absence of microorganisms in the root canal system.[28] When microorganisms are in a dormant stage, a balance may exist between the host defenses and the intracanal irritants. This delicate equilibrium can be upset by a change in the microbial flora or in the host resistance, or by microbial recontamination through coronal leakage when there is a deficient coronal restoration. If there is a coronal seal, and/or when the coronal restoration does not require replacement, radiographic and clinical monitoring may be considered. A study by Van Nieuwenhuisen, Aouar, and D'Hoore[29] showed that, in this situation, a wait-and-see approach over a six-year period led to complications in as little as only 2.8% of cases. On the other hand, if a new restoration is scheduled, these cases should be considered as potential failures[30] and retreatment should be considered prudent (Figure 23-8).[31] However, retreatment of these cases has been shown to lead to failure in 6% of the cases,[32] mainly due to over-instrumentation and overfilling (Figure 23-9).
B. In the second situation, the patient is also asymptomatic, yet a periapical lesion exists. In these instances, the question is whether these lesions will heal without additional intervention. The quality of the root canal filling must be assessed and, if practical, information should be gathered on how and when the initial treatment was performed. Usually, when the quality of treatment appears adequate, the healing process will take place after a minimum period of one year. However, it has been shown that some lesions required 10 years or more to heal.[33,34] Nevertheless, according to the current consensus, most periradicular lesions will heal in four years.[30] It is important to note that, when contamination occurs during intracanal procedures, apical periodontitis oftentimes develops quickly, within one year of the original treatment.[35] In these instances, healing should not be expected and retreatment is indicated. Infrequently, a small radiolucency can be the result of an inflammatory reaction due to the toxic effects of some root canal filling materials. Importantly, the diagnostician should make the distinction between a root canal that radiographically appears well filled with surplus vs. a root canal that is laterally underfilled and vertically overextended. In the instance of a well-filled root canal system, toxicity generally decreases with time and the lesion resolves. Such a hypothesis should also be considered when the lesion is associated with recent treatment that was carried out months earlier.[36]

Decide to Treat A differential diagnosis must be made when a radiographic radiolucency has been detected to rule in or out a lesion of endodontic origin. Histologic studies have shown the presence of granulomatous tissue or cysts in 95% of cases.[37] These inflammatory reactions are frequently the result of a persistent root canal infection[28,38,39] or, less frequently, attributable to extra canal infection.[40,41] On the other hand, some patients will present asymptomatic, yet may exhibit a

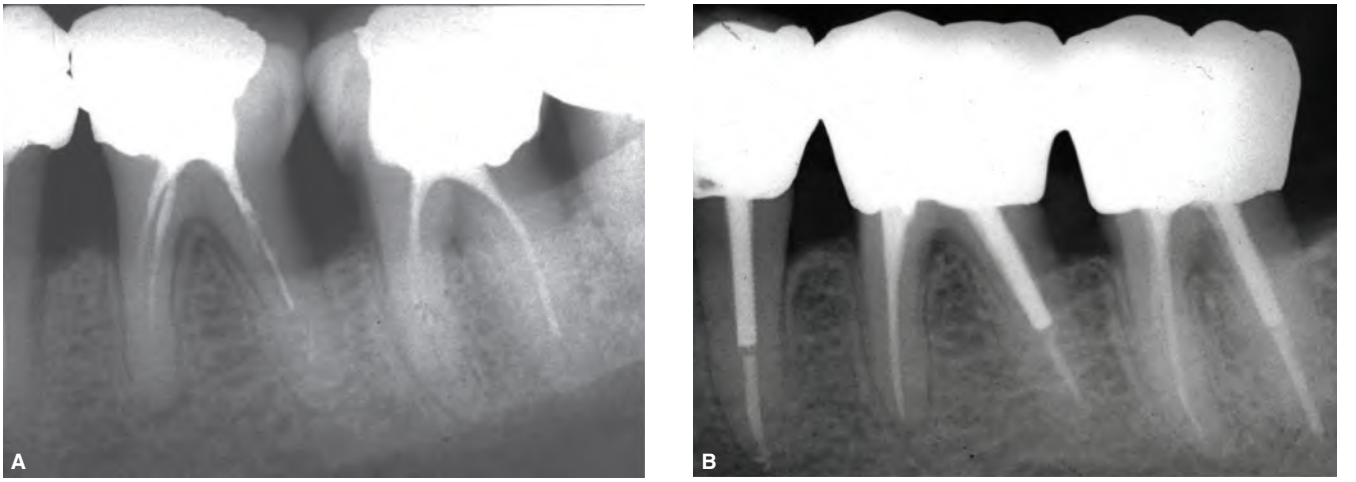


FIGURE 23-8 ***A***. Retreatments are indicated in teeth with poorly sealed canals when a complex treatment planning involving periodontics and prosthodontics is scheduled. ***B***. 12-year follow-up radiograph after retreatments and final restoration.

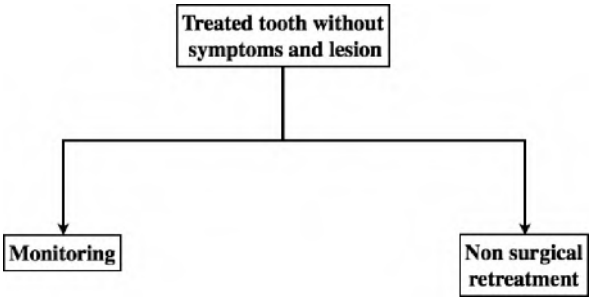


FIGURE 23-9 Decision tree: Tooth without symptoms and periapical lesion.

periapical radiolucency that may, in fact, be healing by scar tissue formation.[37,42]

The benefits vs. risks of endodontic retreatment must be assessed and weighed. Currently, little evidence exists connecting the presence of a periapical lesion with the systemic health of the patient,[43] except for coronary heart disease.[44] Similarly, scarce information is available about the risk of flare-ups when retreating quiescent periapical lesions. According to Eriksen,[45] the estimated incidence of quiescent lesions spontaneously exacerbating appears to be less than 5% per year. In contrast, asymptomatic patients with quiescent lesions have a flare-up incidence of 20% to 40% when retreatment is initiated.[46,47] One explanation may be related to the difference in the composition of microbial flora present during initial treatment of a necrotic tooth vs. retreatment.[39]

With that said, it is appreciated there are some risks associated with the retreatment procedure, itself. For example, when a post and crown restoration has to be removed, the residual tooth structure can be weakened, resulting in a tooth fracture. Further, procedural errors, like root thinning or perforations, may result from excessive re-instrumentation.[30] When a patient reports signs or symptoms associated with an endodontically treated tooth and the radiographic examination is nondiagnostic, CBCT examination is indicated. CBCT provides three-dimensional images and more diagnostic information than a well-angulated periapical radiograph, alone (Figure 23-10).[18]

Decide What Type of Treatment is to be Undertaken

Depending on the situation, three therapeutic options can be considered: extraction, orthograde, or surgical retreatment (Figure 23-11).

As mentioned earlier, many teeth that are candidates for endodontic retreatment have been already restored. If orthograde retreatment is selected, the presence of remaining tooth structure must be anticipated before disassembly. Sufficient cervical tooth structure must be present in order to achieve a new and reliable restoration.[48] Where there is insufficient tooth structure and periodontal crown lengthening is contraindicated, tooth extraction is indicated and a new treatment plan is required. Orthograde retreatment is the treatment of choice in cases that radiographically exhibit poorly sealed canals. Therefore, the goal is to improve the quality of the previous treatment (Figure 23-12). In these instances, the rate of success is comparable to initial treatment when access to the root canal terminus is possible, the canal(s) can be well shaped, and their related root canal system can be disinfected and filled.[49] Indications for a surgical approach arise from the limitations to achieve these goals.

Surgical correction may be considered when there are iatrogenic errors radiographically noted in the apical region, when access to the root canal system is too dangerous, when the tooth to be retreated is involved in a complex or long-span splint restoration, or when what appears to be adequate treatment is associated with signs or symptoms. Over years, follow-up studies comparing orthograde retreatment and endodontic surgery have shown a poorer rate of success for a surgical approach.[30] On the other hand, surgical success increases when nonsurgical retreatment is first undertaken to more fully address the root canal system. It is appreciated that today's surgeon has many advantages due to advancements in armamentarium. These advancements include the microscope, ultrasonic tips, and biocompatible retrofilling materials.[50–52] It is

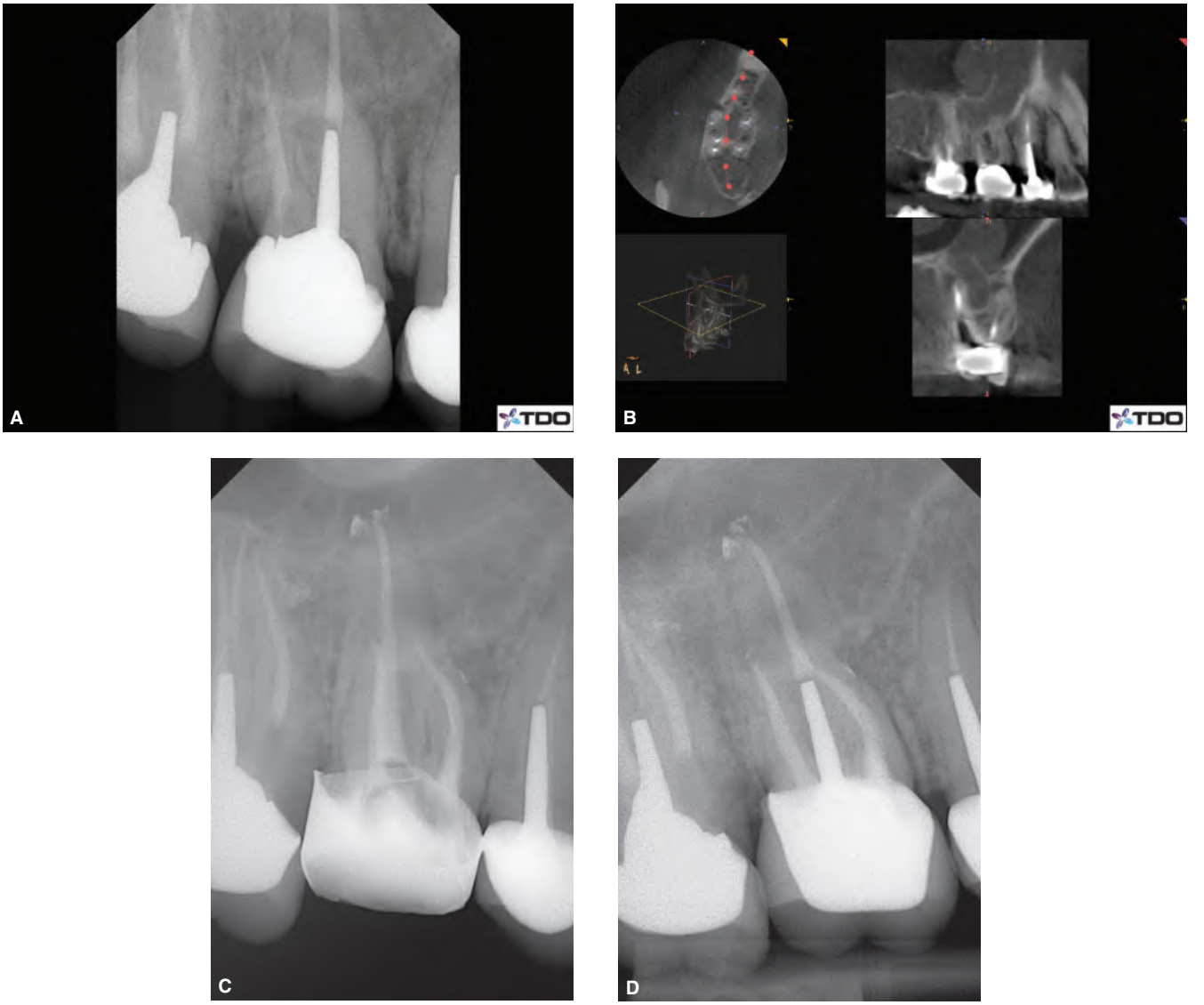


FIGURE 23-10 ***A.*** The patient complained of pain on the right first molar. Clinical examination and radiographs were non contributory. ***B.*** Coronal images from a CBCT showed two periapical lesions on the MB and palatal roots. ***C.*** Postoperative radiograph after retreatment and 3-D filling. ***D.*** Pain was relieved. A new restoration was placed two weeks later.

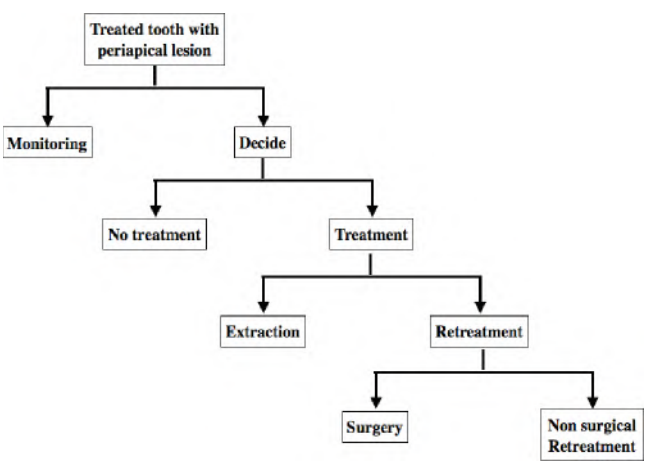


FIGURE 23-11 Decision tree: Tooth with periapical lesion-treatment options.

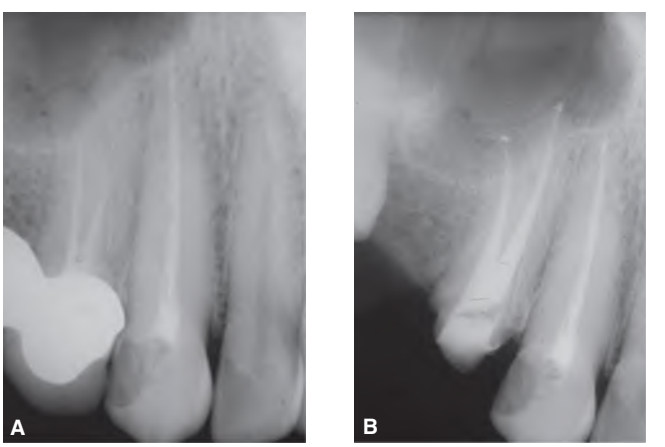


FIGURE 23-12 ***A.*** Poorly sealed canals are an indication for orthograde retreatment. ***B.*** Tooth retreated.

noteworthy that healing after surgical retreatment is faster than after nonsurgical retreatment and can be observed at one year. However, after four years, these two procedural results are balanced.[53] Nevertheless, surgical access and techniques are restrictive in their ability to remove the harmful content from the root canal system.

Patient Preferences

It is essential to disclose the different therapeutic options in order for the patient to participate in the decision-making process. Full information should be given in simple and understandable language so the patient is more involved in the therapeutic act and becomes an actor in the decision. Simple principles to follow have been given by Reit[26] in order to make the best decision:

- 1st principle: Any persistant lesion after treatment must be retreated.
- 2nd principle: A persistant lesion *may not* be retreated if:
  - ◊ Retreatment is not the patient's choice.
  - ◊ The risks outweigh the benefits.
  - ◊ Global cost exceeds predictable alternatives.

Attitudes toward periapical disease vary among individual patients. Some patients worry about living with an infection and readily accept a recommendation for a retreatment procedure. Asymptomatic patients are less concerned about a chronic lesion, weigh the pros and cons, and may finally decide to do nothing. Even when the decision is contrary to the dentist's opinion, the patient's right to choose must prevail.

Clinical Procedures and Sequencing Dissassembly

Endodontics is a matter of strategy and tactics, and this is particularly true in the case of retreatment. First, the clinician has to understand the cause of the failure and then ask the question if he/she has the judgment, experience, and training to give the patient the best chance for success. Importantly, the clinician must have the technology to help guide each retreatment procedure toward longterm success. This approach can be summarized by the triad: ***analyze***, ***plan***, and ***execute***.

General and local objectives must be well integrated before attempting to retreat. General objectives include technical and pathological indications. In the first circumstance, the goal is to improve the quality of the treatment and avoid any contamination. The second objective is to control, then eliminate, infection. Local objectives involve coronal disassembly, complete access, ruling in or out hopeless fractures, identifying all orifices/canals, removing previous obturation materials, eliminating post and retrieving broken/separated instruments, and repairing iatrogenic perforations and apical transportations. When properly performed, all of these activities promote shaping canals, when indicated that in turn encourages 3-D disinfection and filling root canal systems. This is the essence of nonsurgical retreatment.

The authors recognize that the way endodontics is being taught and practiced can vary from country to country. For example, in some parts of the world, there is more emphasis on placing posts in virtually all endodontically treated and restored teeth; whereas, in other parts of the world, a core build-up is performed without a post. Therefore, retreatment sequencing will vary according to the clinical examination and diagnostic radiographs associated with each presenting patient. When there is no post, for example, finding missed canals or removing endodontic root canal fillers may commence upon regaining access. Therefore, sequencing endodontic treatment and initiating the appropriate treatment approach varies according to each patient's specific condition, radiographic examination, and clinical discovery following coronal disassembly.

GAINING ACCESS TO THE ROOT CANAL/CORONAL DISASSEMBLY

Apical periodontitis may be the result of persistent microorganisms following intracanal instrumentation and disinfection. But a secondary infection may occur through coronal leakage due to defective restorations; a cause for late failures.[54,55] Therefore, prior to retreatment of a failing case, amalgam and defective restorations should be systematically removed. This enables the clinician to clean out potential recurrent decay, assess the value of the remaining crown structure, and possibly discover the presence of cracks. The next step to facilitate retreatment is to place a new temporary restoration, usually a glass-ionomer cement, either a copper or orthodontic band, or a prefabricated crown to rebuild the missing tooth structure (Figure 23-13). Pretreatment is a a mandatory step in orthograde retreatment and a prerequisite for future work in the best aseptic conditions. For more details, see Chapter 19, "Preparation for endodontic treatment."

Most of the teeth to be retreated have been restored with crowns and sometimes posts. It is prudent to disassemble all restoratives from the pulp chamber to promote identifying canal orifices that may be hidden by cements, and composite or metal materials.[56] Occasionally, access to the pulp chamber can be created through an existing crown if it is judged to be functionally designed, well-fitting, and esthetically pleasing (Figure 23-14), or if the tooth is part of long-span fixed bridges. However, when the axial alignment of the crown and root are different, caution must be exercised regaining access so as to not needlessly destroy tooth structure or perforate. When access procedures could potentially damage the crown, or if the restorative is deemed inadequeate, then the restoration should be sacrificed. When an access cavity is created through a metal restoration, the axial walls should be extended laterally to eliminate interferences and provide freedom for the subsequent use of shaping instruments. Access cavities should be designed to prevent debris, such as metal filings, from falling into the canal that could serve to create an irreversible blockage. Owing to their own weight, metal shavings will not stay in suspension within the irrigating solution.[56]

Factors Influencing Restorative Removal

The removal of a restorative is dramatically enhanced when there is knowledge, respect, and appreciation for the concepts, materials, and techniques utilized in restorative and

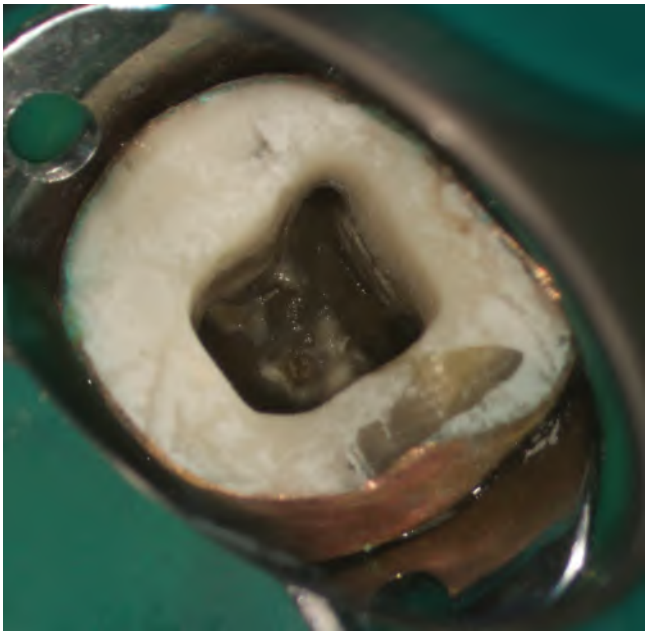
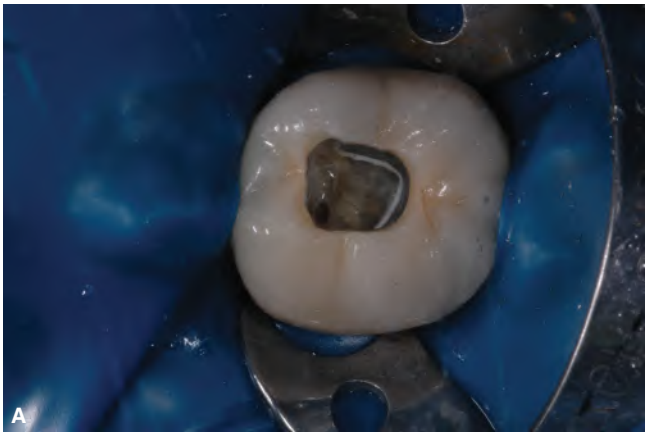


FIGURE 23-13 A copper band was placed to rebuild the missing tooth structure and allowed the creation of a four-wall access cavity.



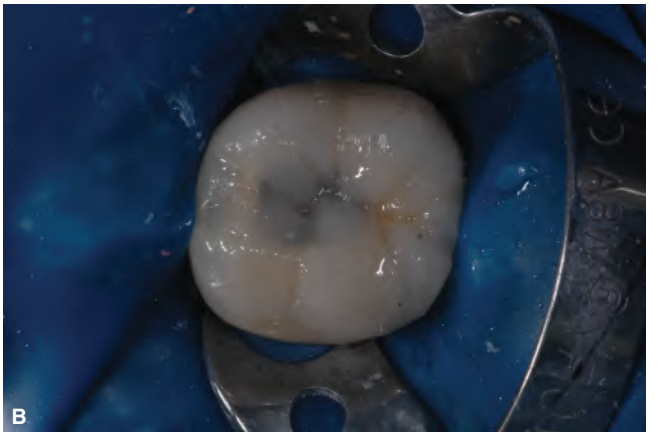


FIGURE 23-14 ***A.*** Access is made through a well fitting and esthetically pleasing crown. ***B.*** After retreatment. A composite resin was used to restore the crown.

reconstructive dentistry. The safe dislodgement of a restorative is dependent on five factors that must be considered.

1. *Preparation Type*: Preparations vary in retention depending on the total surface area of the tooth covered and the height, diameter, and degree of taper of the axial walls.
2. *Restoration Design and Strength*: The design and ultimate strength of a restorative is dependent on its physical properties, thickness of material(s), and the quality and techniques of the laboratory technician.
3. *Restorative Material(s)*: The composition of a restoration ranges from different metals to tooth-colored restoratives, such as porcelain. How these materials behave related to the stresses and strains required during removal must be appreciated.
4. *Cementing Agent*: The retention of cements ranges from weak to strong, generally progressing from zinc oxide-eugenol, polycarboxylate, silico phosphate, zinc phosphate, glass ionomers, resin modified glass ionomers to bonded resins. Clearly, the new generation bonding materials, in conjunction with well designed and retentive preparations, have made restorative removal more difficult[57] and, at times, unwise.
5. *Removal Devices*: The safe and successful dislodgment of prosthetic dentistry requires knowledge regarding the selection and use of a variety of devices. Clinicians need to identify and become familiar with each device, its safe application, effectiveness, limitations, and costs.

The clinician must obtain a good history, confer with the original treating dentist if possible, and then consult with the patient to clearly define the risk versus benefit when entertaining the intact removal of an existing restorative.

Coronal Disassembly

Coronal disassembly can be carried out in three distinct ways: destructive, minimally destructive, and conservative.

Destructive Disassembly

Destructive disassembly involves cutting through the crown with a carbide bur (transmetal bur, *Dentsply Maillefer; Ballaigues, Switzerland* or H4MC bur, *Komet; Rock Hill, South Carolina, USA)*. On the facial surface of a restorative crown, a slot is made from the cervical margin and extends toward the cavo surface, then to the middle of the occlusal surface. Ultrasonics is applied inside this slot to disintegrate the luting cement. Subsequently, an elevator (Figure 23-15) or the Christensen Crown Remover (*Hu-Friedy, Chicago, Illinois, USA*) may be used to flex apart the crown and loosen it. This procedure is predictable and safe, and if needed, the crown can be relined to serve as an interim restorative (Figure 23-16). This is also an excellent method for removing long span bridges, where each abutment is individually managed. In single-rooted teeth, the slot should involve both buccal and palatal faces (Figure 23-16).

Minimally Destructive Disassembly

In this approach, a minimally invasive procedure is performed and results in a small amount of force required to dislodge

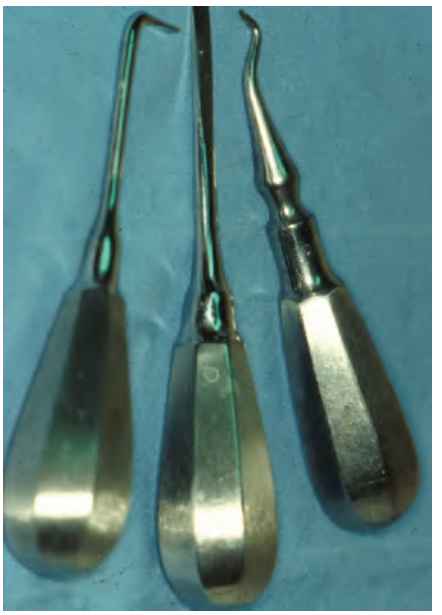


FIGURE 23-15 Different types of elevators used for crown removal.

the restorative. Some examples of minimally destructive disassembly devices are as follows:

- WAMKeys (*WAM; Aix en Provence, France*, or *WAM; Montréal, QC, Canada*): This system is easy-to-use and convenient to safely remove the majority of metallic and/or porcelain crowns and short bridges. The intro kit includes three narrow-handled instruments with distal ends that each provide a different sized cam (Figure 23-17). Three steps should be followed. First, a horizontal slot is created with a transmetal bur in the middle of the facial aspect of the crown or about one millimiter below the central groove of the occlusal restorative surface. In the instance of a procelain-fused-to-metal crown, a 016 diamond round bur (*Komet*) is used with a water coolant to protect the porcelain and cut a class V tapered cavity through the porcelain and to the underlying metal substructure (Figure 23-18A). Second, a transmetal bur is used to cut through the metal and create a trench to the middle of the occlusal surface (Figure 23-18B). Third, an appropriately-sized WAMKey is selected, inserted inside this trench, and gently rotated to elevate the restorative crown along its own axis (Figure 23-18C and D).[56]

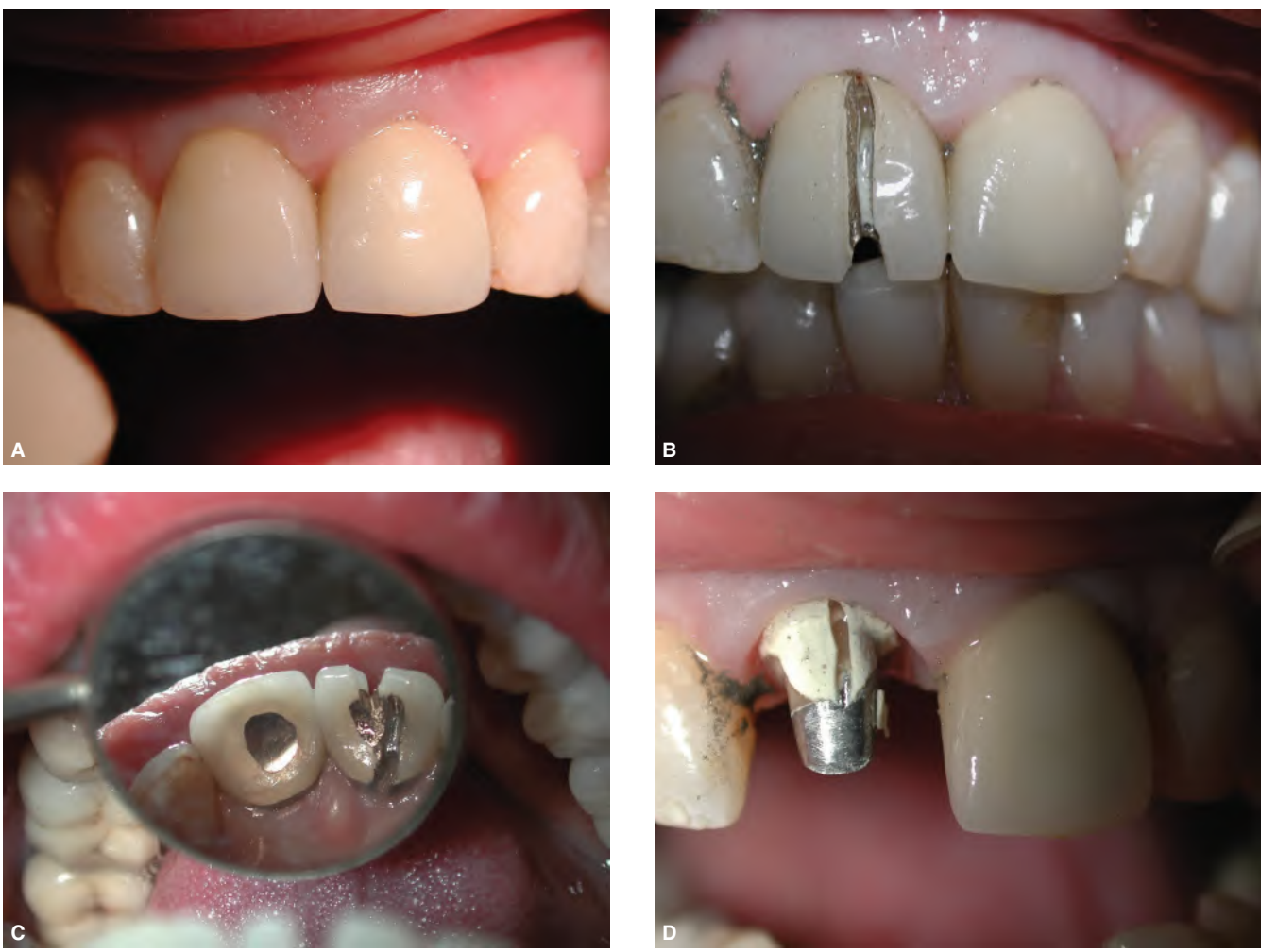


FIGURE 23-16 ***A.*** Orthograde retreatment is indicated in the crowned maxillary right central incisor. ***B.*** Starting from the cervical area and following the tooth structure, a slot is made on the buccal surface. ***C.*** The slot should involve both the buccal and palatal faces. ***D.*** Crown removed.

- Metalift™ *(Classic Practice Resources; Baton Rouge, Louisiana, USA)*: The Metalift (Figure 23-19A) is designed to safely remove inlays, onlays, three-quarter crowns, full crowns and fixed bridges in a minimally-invasive way so these restoratives can be reused, if desired. The kit includes all items needed to perform the procedure, as follows: One, in the instance of a procelain crown or bridge, the cylindrical diamond bur is utilized to precisely remove the porcelain and expose the underlying metal (Figure 23-19B). Two, with the help of magnification, create a pilot hole with a small carbide round bur through the metal and to solid structure (Figure 23-19C). Three, a twist drill is used to create a precision channel in the metal that exactly matches the size of the Metalift instrument to be used later in step five (Figure 23-19D). Four, substructure material is carefully removed on the bottom side of the metal so as to not damage the precision channel and to prevent threading the Metalift instrument into tooth structure. Five, the Metalift instrument is threaded through the metal until its distal end contacts tooth structure and rotation is continued to lift off the crown (Figure 23-19E and F).
- The Kline Crown Remover *(Brasseler; Savanna, Georgia, USA)*, and the Higa Bridge Remover *(Higa Manufacturing; West Vancouver, BC, Canada)* operate with the same concept.

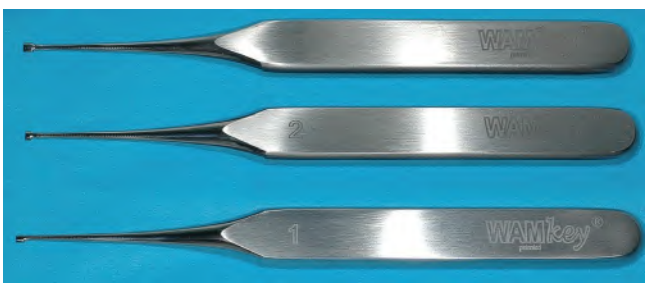


FIGURE 23-17 WamKeys (*WAM, Aix en Provence France, WAM, Montréal, QC, Canada*).

Conservative Disassembly

Conservative disassembly involves utilizing percussive devices and techniques.[57] In this approach, safe and successful removal of a prosthesis is accomplished by disintegrating the luting cement. This removal method utilizes manual sliding hammers or pneumatic crown removers. The tip of these devices grasps the cervical crown margin on the buccal or lingual aspect and a controlled series of impact strokes are delivered to loosen the restoration. However, these impact shocks may induce misdirected forces that can be extremely harmful and predispose to coronal or radicular fractures. Therefore, the authors caution against their routine use. Examples of this type of crown remover device include the manual sliding hammer *(Miltex; Plainsboro, New Jersey, USA or Anthogyr; Atlanta, Georgia, USA)* and Crown-A-Matic *(Peerless International, Inc; North Easton, Masssachusetts, USA)*.

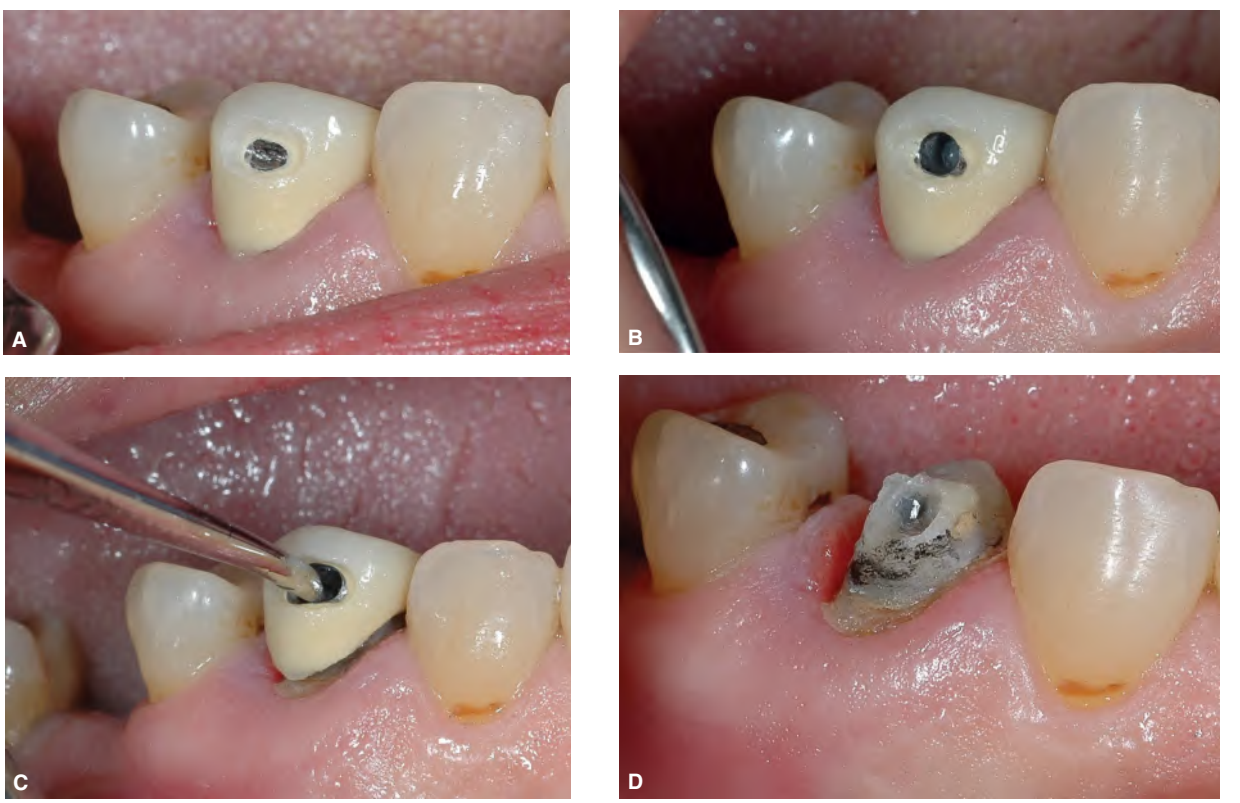


FIGURE 23-18 ***A.*** A diamond round bur is used with a water coolant to protect the porcelain and cut a class V tapered cavity through the porcelain to the underlying metal substructure. ***B.*** A trench is made with a transmetal bur through the underlying metal substructure to reach the middle of the occlusal surface. ***C.*** The WamKey is inserted inside the trench and gently rotated to elevate the crown. ***D.*** It's not necessary for the WamKey to encroach the core.

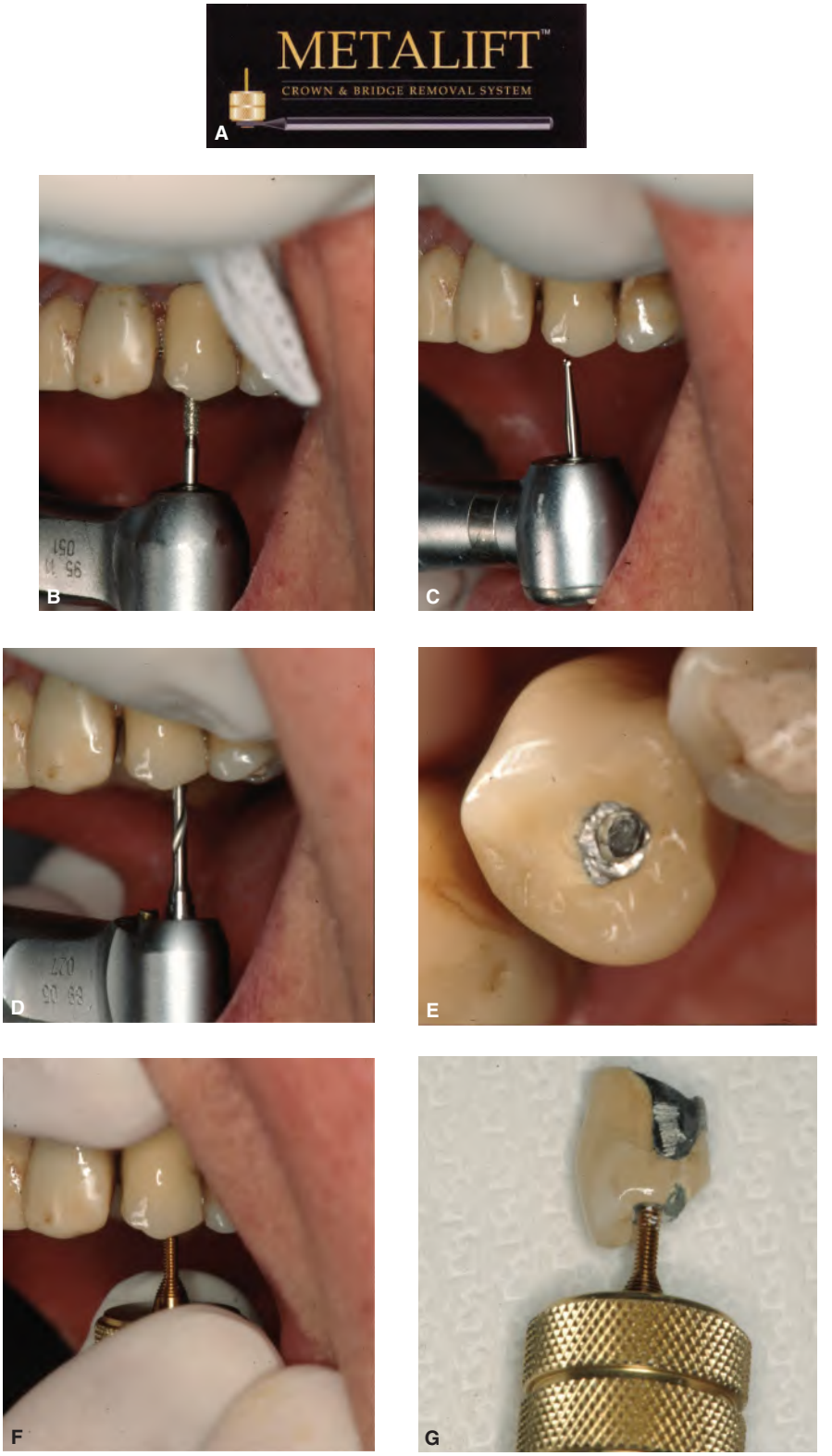


FIGURE 23-19 ***A.*** Metalift. ***B.*** The cylindrical diamond bur removes the porcelain and exposes the underlying metal. ***C.*** The tiny round bur creates a pilot hole to reach the tooth structure. ***D.*** The twist drill creates a precision channel to match the Metalift instrument. ***E.*** A close-up image of the prepared access before the use of the Metalift instrument. ***F.*** The Metalift instrument threads through the metal and contacts the tooth structure. ***G.*** Crown removed.

On the other hand, a special consideration must be given to the Coronaflex (*Kavo Dental; Charlotte, North Carolina, USA*). The Coronaflex (Figure 23-20) is an elegant and very useful technique to perform bridge removal. This removal method encourages an axial pulling direction that serves to mitigate damaging a prosthesis. The procedure is easy, quick, and safe, as well as comfortable for the patient. The Coronaflex utilizes compressed air to produce ultrashort impulses at the tip of

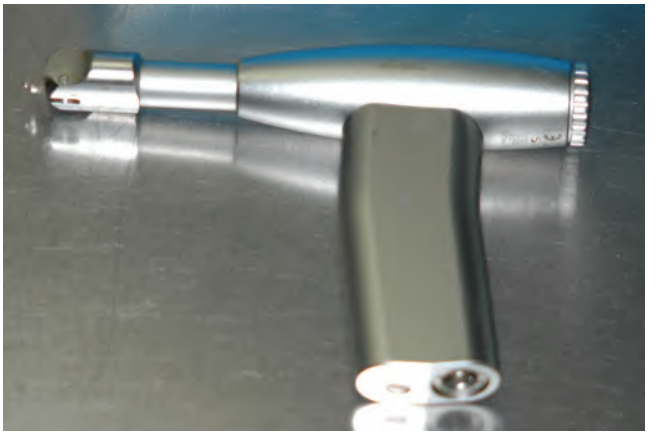


FIGURE 23-20 Coronaflex.

the instrument to break the luting cement. The recommended method of use is the so-called "parachute technique." Metallic wires are looped through two or more embrasures of the bridge, that, in turn, are connected to a metal rod. The tip of the pneumatic handpiece is then placed against the rod and, upon activation, delivers a series of micro-pulses that break the cement bond, allowing for the removal of the bridge along its own axis of insertion. Similarly, when a tooth is soundly embedded in the periodontium, the parachute technique is an expedient way to remove a cast post and core, in one piece, from any given tooth.[56]

Grasping devices are inefficient to remove fixed cemented prosthesis. They are best utilized in removing provisionalized dentistry[57] and include K.Y. Pliers (G.C. America; Alsip, Illinois, USA), the Wynman Crown Gripper (Miltex Instrument Company; Lake Success, New York, USA), and the Trident Crown Placer-Remover (Trident Dental Inc; Hendersonville, North Carolina, USA).

REMOVAL OF POSTS AND CORES

It is common for clinicians to encounter endodontically treated teeth that contain posts. Frequently, when endodontic treatment is failing, the need arises to remove a post to facilitate successful nonsurgical retreatment. In other instances, the endodontic treatment may be judged successful, but the restorative needs require the removal of an existing post to improve the design, mechanics, or esthetics of a new restoration. Over time, many techniques have been advocated for the removal of posts and other large intracanal obstructions.[58–60]

Factors Influencing Post Removal

There are many factors that influence successful post removal, such as operator judgment, training, experience, and utilizing the best technologies and techniques.[3] Further, clinicians should have knowledge and respect for the anatomy of teeth and be familiar with the typical range of variation associated with each tooth type.[61] As an example, it is important to know tooth morphology, including the length, circumferential dimensions, and curvature of any given root, as well as the depth of an external concavity, if present. This information is best appreciated by obtaining three well-angulated preoperative radiographs. Radiographs also assist the clinician in visualizing the length, diameter, and direction of the post, and aid in determining if it extends coronally into the pulp chamber.[62]

Other factors influencing post removal are the post type and cementing agent.[63,64] Posts can be catalogued into parallel versus tapered, active versus nonactive, and metallic versus non-metallic compositions (Figure 23-21).[65,66] Posts retained with the classic cements like zinc phosphate can generally be removed; however, posts bonded into the root canal space with materials like composite resins or glass ionomers are oftentimes more difficult to remove.[67] In addition, other important factors that impact post removal are the available interocclusal space, existing restoration, and whether the coronal most aspect of the post is supra or subcrestal (Figure 23-22). In general, post removal becomes more challenging moving from anterior to posterior teeth. The difficulty in removing a post substantially increases in furcated teeth containing multiple posts joined coronally with single or multiple interlocking key-ways (Figure 23-23).

When evaluating a tooth for post removal, the clinician must weigh risk versus benefit before proceeding with this procedure.[68,69] As an example, the relative radiodensity between a titanium or a titanium alloy post can appear very similar, or even identical, to gutta-percha when viewed radiographically. As such, when considering nonsurgical retreatment, clinicians need to be familiar with the radiographic characteristics of these nonmetallic posts.[70] A root can be structurally weakened, perforated, or fractured during any phase of retreatment ranging from radicular disassembly to subsequent shaping and filling procedures. In some instances, it may be wise to consider a surgical approach to resolve an endodontic failure. However, surgery should not be performed promiscuously because of lack of training in the best, presently developed techniques utilized for post removal.

Ultrasonic Removal of Prefabricated Post Associated with a Composite or Amalgam Core

The goal is to carefully remove the restorative material with a high-speed carbide long shank bur, paying attention not to damage the protruding head of the post. In the case of a composite core, the difference in color between the metallic post and the filling material makes this procedure easy. After freeing the head of the post, residual fragments of the restorative material and the luting cement must be carefully removed by using a thin ultrasonic tip (Start-X #3, Dentsply, Maillefer; Ballaigues, Switzerland), ProUltra (#3 Dentsply Tulsa Dental Specialties; Tulsa, Oklahoma, USA) (Figure 23-24), or ET20 (Acteon USA; Mt. Laurel, New Jersey, USA). The post is then vibrated with a dedicated ultrasonic tip (Start-X #4, ProUltra #1, or ET PR) with a water coolant until it can be removed (Figure 23-25). The ultrasonic tip should be kept in intimate contact with the post to maximize energy transfer

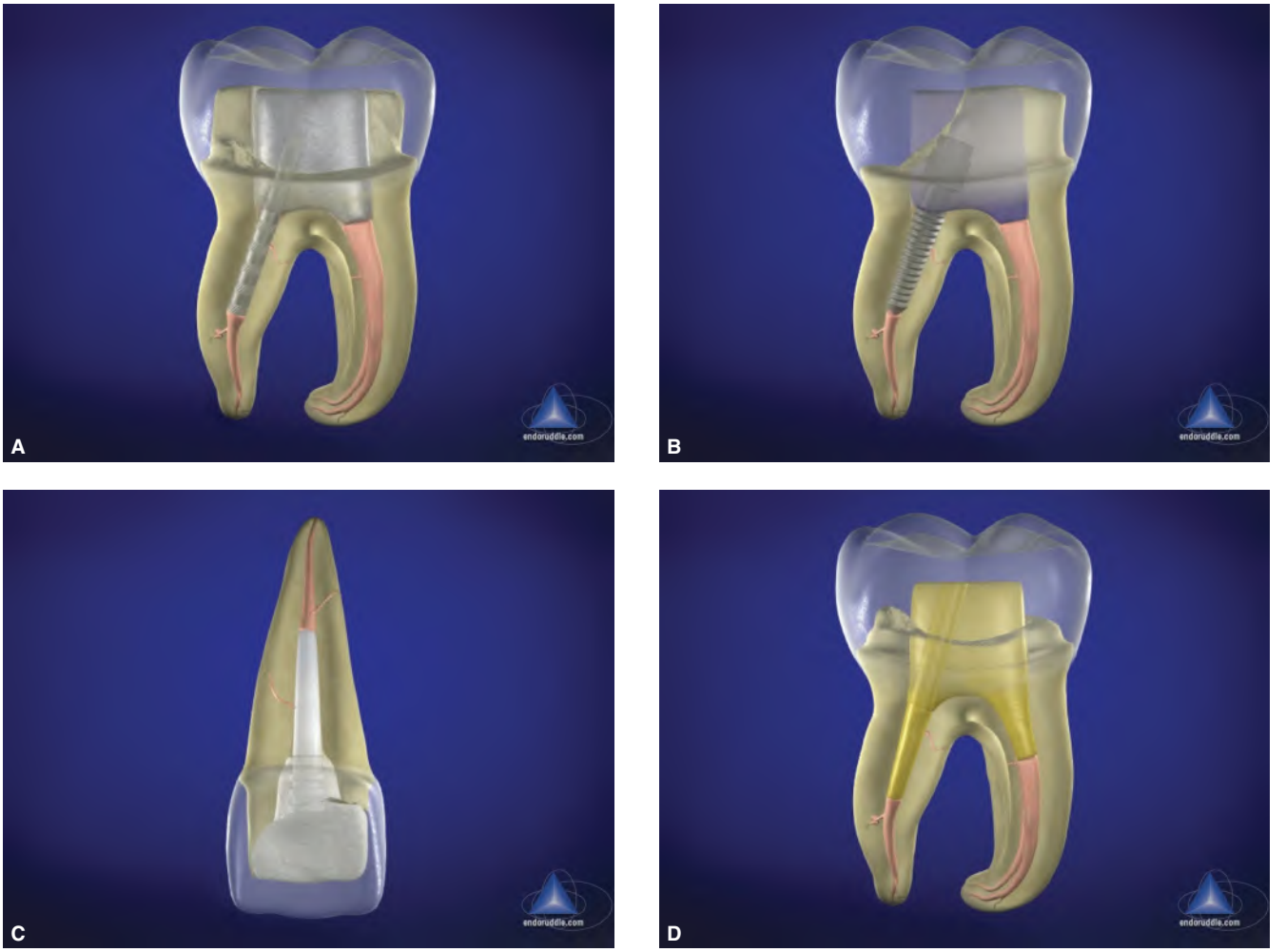


FIGURE 23-21 ***A.*** Parallel post. ***B.*** Screw post. ***C.*** Composite post. ***D.*** Cast gold post/core.

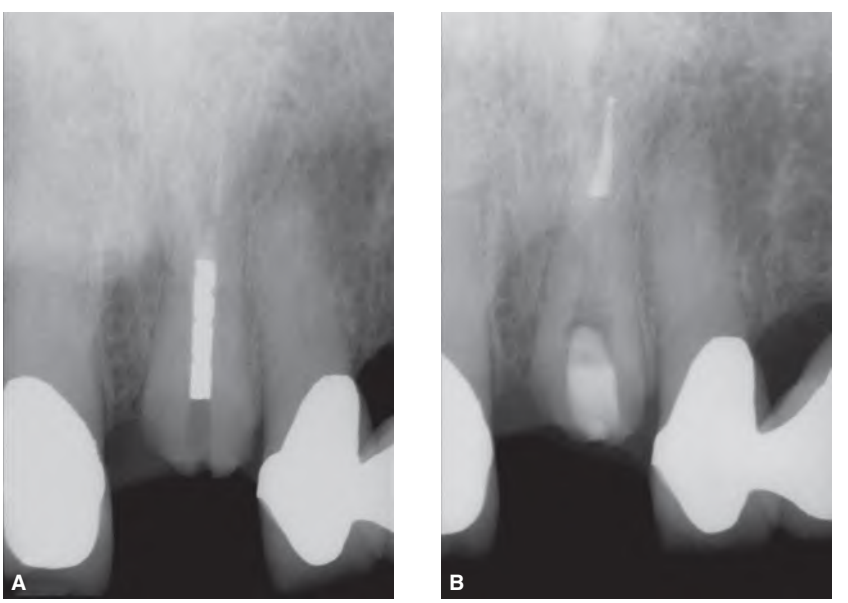
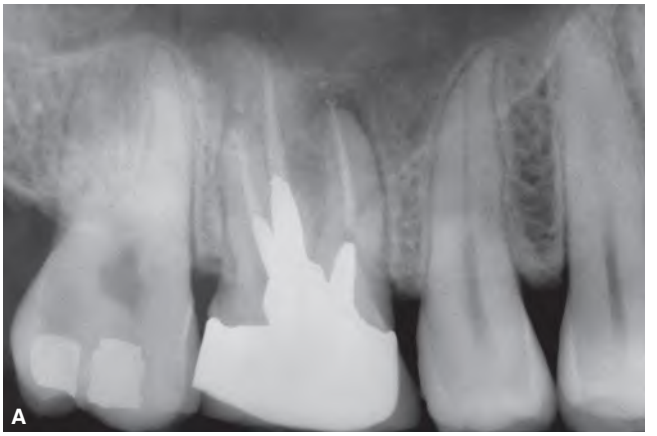
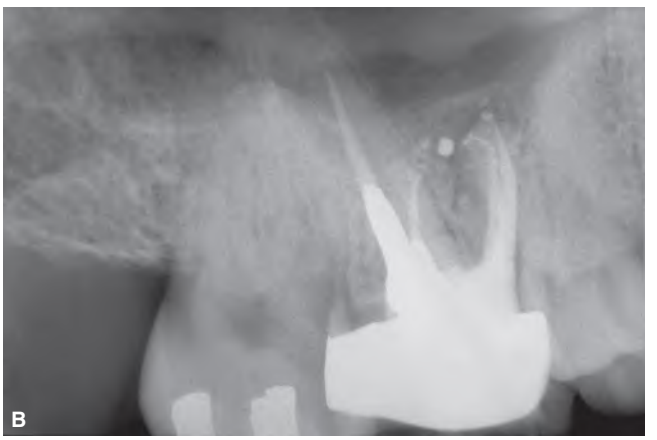


FIGURE 23-22 ***A.*** Preoperative radiograph of a maxillary central incisor showing a post fractured subcrestally; this example serves to emphasize the importance of treatment planning. ***B.*** Post-treatment radiograph following post removal, retreatment, and post space done prior to crown lengthening and restorative treatment.





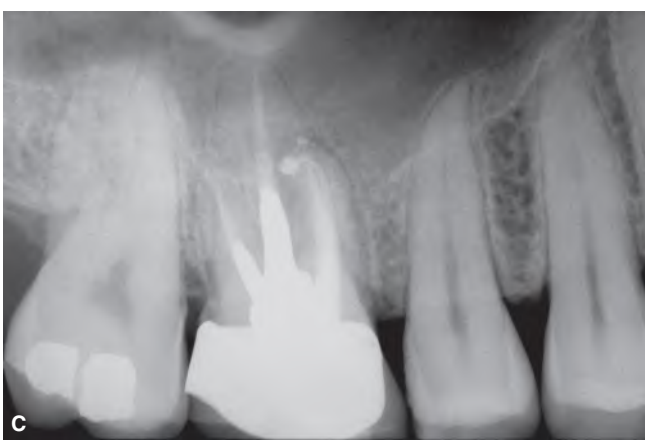


FIGURE 23-23 ***A.*** Preoperative radiograph reveals the MB root of the maxillary first molar is failing endodontically. Note, 3 cast posts interlocking within the pulp chamber. ***B.*** A distal angulated radiograph demonstrating the retreatment effort of the MB root. Note the identification and treatment of the MB-2 system. ***C.*** A two-year recall radiograph demonstrates excellent bony healing associated with the MB root.

and promote cement/bond failure. In the case of an amalgam filling, it is important to work the high-speed bur from the periphery toward the location of the post so as to not damage it. In the presence of a screwpost, the ultrasonic tip is rotated counterclockwise (CCW) around the post to dislodge it.

It must be recognized that the byproduct of ultrasonic energy is heat.[71–73] When performing ultrasonic procedures

FIGURE 23-24 The ProUltra ENDO 1-5 ultrasonic instruments have an abrasive zirconium nitride coating to improve efficiency, precision and clinical performance. The ProUltra ENDO-6, 7, and 8 titanium ultrasonic instruments provide longer lengths and smaller diameters, and are utilized when space is restrictive.

for longer periods of time and against larger conductive metal posts, the field should be frequently flushed with water to decrease heat buildup and the potential for dangerous heat transfer to the attachment apparatus. Experience suggests that, after removing all circumferential restorative materials, the majority of posts can be safely and successfully removed within approximately 10 minutes.[3,73] However, certain posts resist removal even after ultrasonic efforts using this "10-Minute Rule." As such, clinicians need a safe and efficient fallback position to liberate these posts.[3,74,75]

For safety, the following precautions should be followed:

- Carefully examine the preoperative radiograph to assess the thickness of the radicular walls between the post and the periodontal tissue. If the thickness is less than 1 mm, the risk of overheating the attachment apparatus quickly increases.
- One or two minute breaks are recommended between ultrasonic sessions.
- Intermittent use of an ice spray (Endo Ice) on the head of the post is recommended. Indeed, a permanent water spray is insufficient to reach and cool down the apical tip of the post. If this ultrasonic attempt is unsuccessful, dedicated mechanical post removal devices should be used.

Another vibrating post removal method employs Rotosonics. This method provides a straightforward technique to potentially loosen and remove a fully exposed post or screw-post. The Regular Tip Roto-Pro bur (Ellman International; Hewlett, New York, USA) is a high-speed, friction grip bur whose six faces are joined by six edges and, when rotated one revolution, its edges produce six vibrations per revolution. When the instrument is rotated at 200,000 rpm, it produces 1.2 million vibrations per minute, or 20,000 vibrations per second. This instrument provides an inexpensive method to remove posts and screw-pots. The bur is kept in intimate contact with

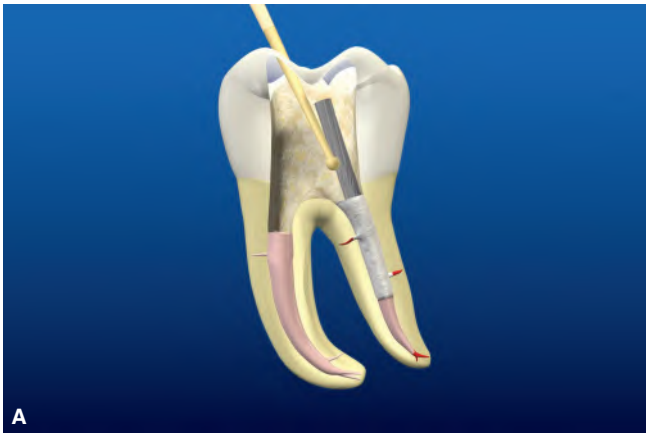


FIGURE 23-25 ***A.*** The ProUltra ENDO-1 is activated and used to powerfully vibrate against all aspects of the exposed post. ***B.*** Illustration demonstrating the importance of using an external water source to reduce temperature and eliminate heat transfer. ***C.*** Following post removal, an ultrasonic instrument breaks up intracanal cement and a White Mac Tip collimates air and blows out debris.

the obstruction and is generally carried CCW around the post. Clinically, rotosonic vibration provides a low-tech method to potentially remove a screw-post or post retained with a more traditional cement, such as zinc phosphate.

Removal of a Fiber Post

A fiber post is usually associated with a composite restorative material. The composite and the protruding head of the post are first removed with a carbide bur mounted on a high-speed handpiece. It is noteworthy that the post should be ground down to the canal orifice. When approaching the pulpal floor, an ultrasonic tip replaces the bur to carefully help eliminate any residual material. For fiber post removal, the goal is to break the bond between the fibers by heating. After creation of a pilot hole in the center of the post with a thin ultrasonic tip (Start-X #3, Dentsply Maillefer; Ballaigues, Switzerland or ET25, Acteon USA; Mt. Laurel, New Jersey, USA) or the pointer drill included in the new Gonon kit (FFDM Pneumat; Bourges, France), the same ultrasonic tip is used with direct vision and without a water coolant to bore through the middle of the post and disintegrate it. The orientation of the fibers helps keep the activated tip in the proper alignment. The last step is to eliminate adhesive fibers from the canal walls with the ultrasonic tip that is used at a low intensity setting with a light, brush-cutting motion. Additionally, fiber posts may be removed with a Unicore drill (Ultradent; South Jordan, Utah, USA), rotated at 15,000 rpm. Start with the small-sized drill (0.6 mm), identified with a white stripe on its handle, then proceed to the yellow-striped drill (0.8 mm), and continue with the red, blue, and green sizes, as necessary, until the fiber post is eliminated. Ceramic and zirconium posts should be avoided since their retrieval is very difficult, thermally dangerous, and virtually impossible.[65]

Removal of Metallic Cast Cores

Metallic cast cores are usually associated with cast posts. In the case of a single-rooted tooth with a cast post and core, two techniques may be used:

1. Utilization of the Coronaflex, as described earlier, when the periodontal environment is sound.
2. Reduction of the core with a transmetal bur without damaging radicular tooth structure. Reduction commences circumferentially around the periphery to the estimated location of the post. The goal is to keep only a cylindrical portion of the core as an extension of the post. Then, ultrasonics and/or a post removal system is used to retrieve the post (see section on mechanical post removal).

In the case of an inlay core within the pulp chamber of a multi-rooted tooth, there may be a single or multiple interlocking key-ways. In these instances, the core must be separated into as many pieces as there are posts present. In the case of a very hard core, such as one fabricated of NiCr, special care is exercised so the bur follows tooth structure, yet does not damage the floor of the pulp chamber. Due to the large amount of vibration delivered during the drilling process, portions of the core devoid of the posts are usually quickly eliminated (Figure 23-26).[56] The head of the post is then cleared from the remaining core portion and post removal is achieved with ultrasonics and/or a post removal system.

Post Removal: Mechanical Options

A number of different devices have been designed to mechanically remove a post.[58,60] However, many of these devices, such as the Masserann kit (Micromega; Besançon, France) and the

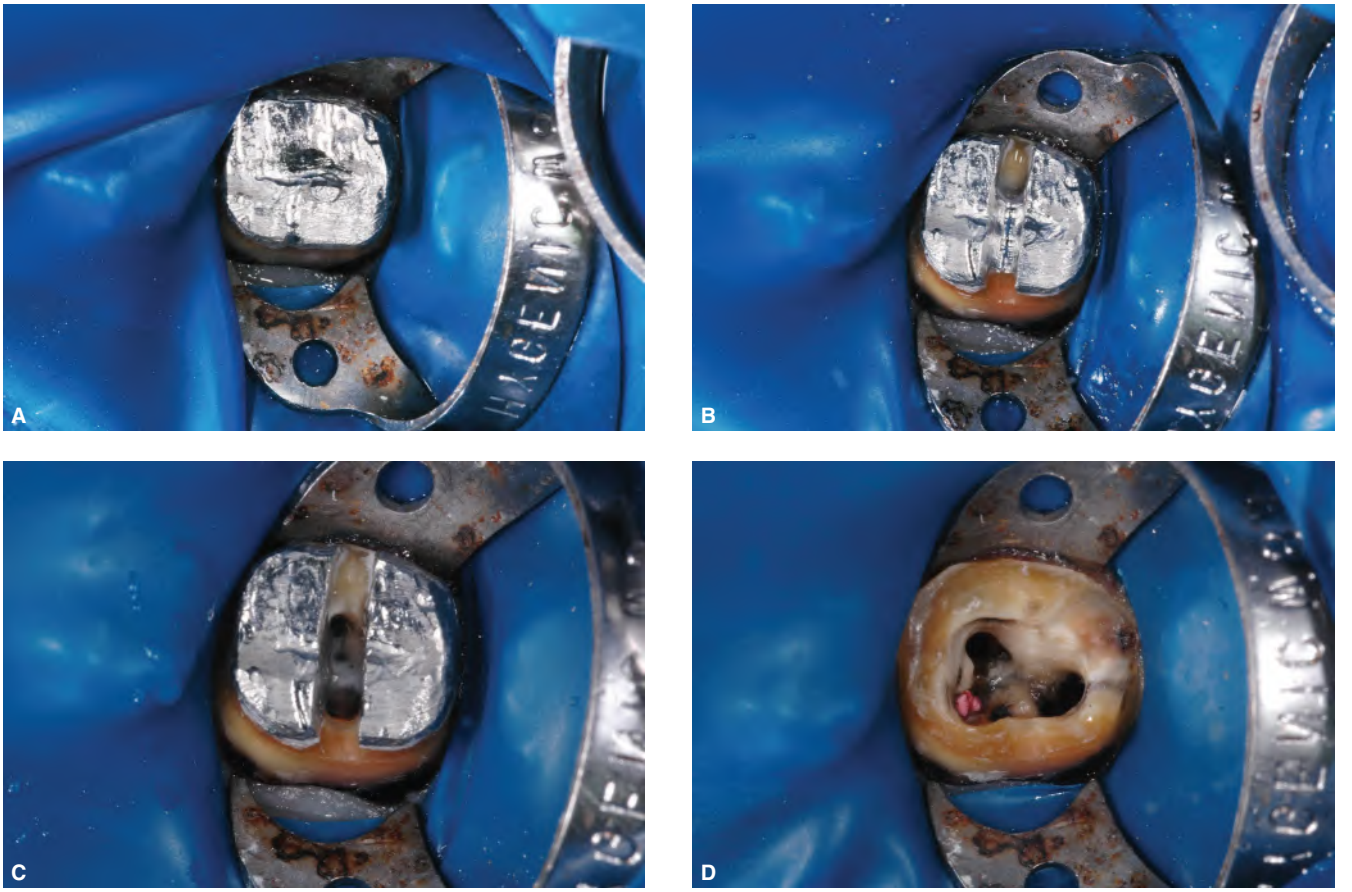


FIGURE 23-26 ***A.*** The mandibular first molar was restored with a NiCr cast core and two posts. ***B.*** Following the tooth structure, a bucco-lingual trench is initiated to separate the core in two pieces. ***C.*** Special care is exercised not to damage the pulp chamber floor. ***D.*** Posts are easily removed due to the combined action of the vibrations delivered during the drilling process and ultrasonics energy.

Post Puller (Brasseler USA; Savannah, Georgia, USA) have had limited success because they frequently require the excessive removal of tooth structure that predisposes to ledges, perforations, or root fractures. The Gonon Post Remover System (EFDM-Pneumat; Bourges, France) represents a definite improvement over the Masserann and the Post Puller devices in that it is less invasive, safe,[68] and has enjoyed good success,[75] but regrettably, by a limited number of clinicians in North America. The Post Removal System (PRS) (Sybron Endo; Orange, California, USA) is a similar device and operates on the corkscrew principle: one force is applied against tooth structure, providing the fulcrum, while the pulling force is placed on the post, creating enough leverage to overcome the bond. Both systems may be predictably used to remove passive conical or parallel posts, cast posts and screw posts. Their respective techniques will be described.

Gonon Post Remover System

Recently, the former Gonon kit has been revisited providing several advantages. It offers fewer instruments with better sizing and can be sterilized. Two trephines are available with matching color-coded and correspondingly sized tubular taps. The tubular taps feature a reverse threading to unscrew serrated and screw posts. The beaks of the extracting pliers are thinner and shorter to facilitate their placement when there is narrow occlusal space. Three sizes of silicone washers and a fiber post kit are included in the kit (Figure 23-27).

The Gonon post removal technique is as follows.

1. After crown removal, circumferential reduction of the core is performed to free the head of the post.
2. To facilitate the centering of the trephine, the head of the post is tapered with a dedicated bur.
3. The largest high-strength trephine is used to bore, grind down, and gauge the protruding head of the post to the exact correspondingly-sized tubular tap. This tubular tap serves to thread down the head of the post and provide a good grip. The more cylindrical the head of the post is bored, the better the trephine efficiency. A high-torque handpiece is used at 15,000 to 20,000 rpm, with short pecking motions, to machine down the head of the post 2 to 3 mm.
4. Before manually screwing the tap onto the post, three washers are placed in the correct sequence onto its shank. They serve to distribute the forces onto the root surface as the post is being extracted. The flat metallic washer is inserted onto the tubular tap first, the concave washer is next inserted onto the top toward the root, and the silicone washer is positioned last and acts as a cushion.

FIGURE 23-27 The Gonon Universal Post remover. Note the color-coding matching trephines and taps. Arrows on tubular taps indicate reverse threading.

5. The extracting pliers are connected to the tubular tap and the beaks of the pliers are expanded by turning the knurled knob. This procedure and resultant force will generally separate the post from the root (Figure 23-28). If too much resistance is felt when rotating the knob, it is useful to reduce resistance by turning the knob CCW a millimeter or two before continuing the clockwise (CW) rotation. In addition, indirect ultrasonics may be applied on the tap to help break the bond.[73] In a study conducted on the incidence of root fractures and methods used for post removal, it was shown that post removal is a predictable procedure with a extremely low rate of root fracture: 0.06% on a total of 1,600 posts removed.[68] This figure is slightly higher than the 0.002% reported in a survey of Australian and New Zealand endodontists.[60]

After post removal, some residual luting cement may be present in the canal beyond the apical tip of the post. With good vision below the orifice, this material can be easily removed with an ultrasonic tip.

Post Removal System

The Post Removal System (PRS) is designed to mechanically engage and remove different kinds of post types or other intracanal obstructions with cross-sectional diameters of 0.60 mm or greater.[3,77] The PRS kit contains extracting pliers, a transmetal bur, five trephines of varying internal diameters, five corresponding tubular taps with internal diameters ranging from 0.60 to 1.60 mm, a torque bar, tube spacers, and a selection of rubber bumpers (Figure 23-29). The preparatory procedures before utilizing the PRS require straightline access and complete circumferential visualization of the post within the pulp chamber.

A transmetal bur is used to round off or taper the coronal most aspect of the post (Figure 23-30A). "Doming" the head of the post will serve to effectively guide the subsequent instruments over the post. A drop or two of chelator, such as RC Prep, Glyde or ProLube, is then placed on the head of the post to act as a lubricant to facilitate the machining process. In order to ensure circumferential milling, the largest trephine that will just engage the post is selected. The latch-type trephines should rotate at approximately 15,000 rpm in a CW direction, in a slow-speed, high-torque handpiece. The trephine is used with a "peck" drilling motion to maintain rpm and to keep the head of the post cooler so it does not work-harden and become more difficult to machine. The trephine is utilized to machine down a 2 to 3 mm length of the most coronal aspect of the exposed post (Figure 23-30B). If the chosen trephine fits passively, then a sequentially smaller size trephine is selected to ensure proper circumferential milling. In some instances, the configuration of the coronal-most aspect of the post, such as a cast post/core, dictates the use of a transmetal bur or diamond to grind down the head of the post to create a relatively round cylinder. The trephine can then machine a precisely round cross-sectional diameter on the post.

In general, the trephine used for machining the post dictates the subsequent selection of a correspondingly-sized tubular tap. An appropriately-sized protective rubber bumper is selected and inserted over the distal end of the tap. The bumper serves to cushion, evenly distribute the loads, and protect the tooth during the removal procedure. The tubular tap is pushed against the head of the milled-down post and is manually turned CCW to form threads (Figure 23-30C). Firm apical pressure and small quarter-turn CCW motions will generally draw down and securely engage the tap to the post. The tap can be screwed over the post as little as 1 mm or, more optimally, up to a maximum of 3 mm. Caution should be exercised so that the tap is not drawn down too far over the post because its maximum internal depth is 4 mm. If the tap bottoms out against the post head, it can predispose to stripping the threads, breaking the wall of the tap, or shearing off the obstruction inside the lumen of the tap. When the tubular tap has snugly engaged the post, the protective rubber bumper is pushed down onto the biting surface of the tooth (Figure 23-30D).

The post removal pliers are then selected and the extracting jaws are mounted onto the tubular tap. The instrument is held securely with one hand, while the fingers of the other hand begin opening the jaws by turning the screw knob CW. As the jaws slowly begin to open, increasing pressure will be noted on the screw knob. The clinician should repeatedly verify that the compressing rubber cushion is properly protecting the tooth. Further, when utilizing this removal method, the clinician should visually confirm the post is being safely withdrawn along the long axis of the root canal. If turning the screw knob becomes increasingly difficult, the clinician should either hesitate a few seconds before continuing and/or use the indirect ultrasonic technique to vibrate on the post-engaged tubular tap (Figure 23-30E). In combination, the PRS and indirect ultrasonic techniques enhance post retention failure, encourage the screw knob to turn further, and are potent adjuncts to successful post removal (Figure 23-30F).[3]

Ultimately, the PRS provides clinicians an important post removal method that can be safely employed when ultrasonic techniques are unsuccessful. Clinicians also encounter

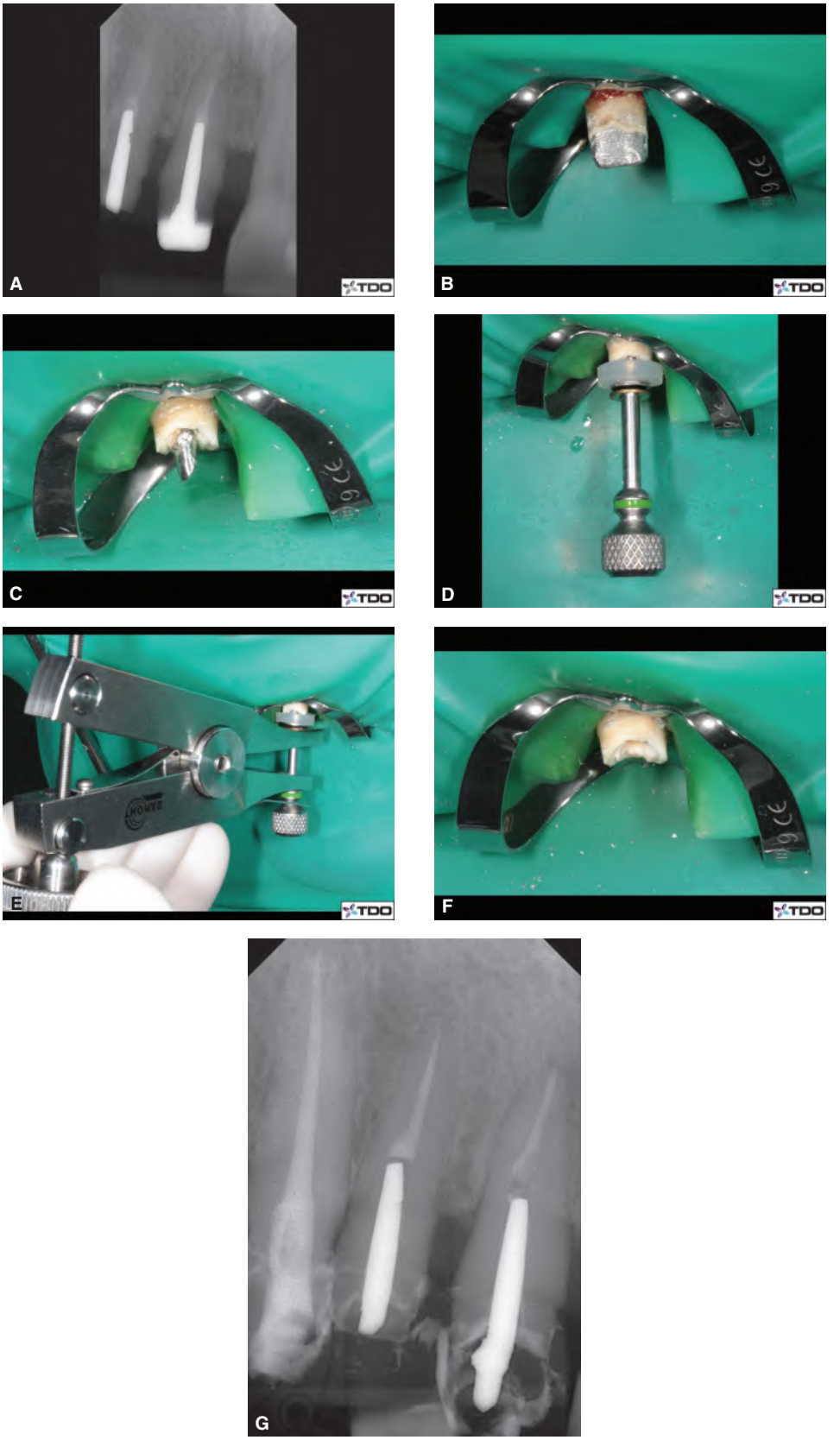


FIGURE 23-28 ***A.*** Two metallic posts must be removed before retreatment. ***B.*** Clinical view of the metallic core. ***C.*** Circumferential pre-reduction of the core before tapering the head of the post to facilitate the centering of the trephine. ***D.*** The tubular tap with the three washers creates a threading on the post. ***E.*** The pliers are connected to the tubular tap. ***F.*** Post removed from the root. ***G.*** Postoperative radiograph of the teeth after retreatment and placement of provisional restorations.

FIGURE 23-29 The Post Removal System is a kit designed to mechanically engage and potentially remove different types of posts.

actively engaged threaded posts that require removal. The PRS is specifically designed to address this scenario because each tubular tap turns in a CCW rotation. The post head is milled down as previously described and a tubular tap threaded until snug. In instances where threaded posts are encountered, the use of the extracting pliers is contraindicated. Typically, the clinician backs the post out of the canal using a CCW rotation with finger pressure. If the post is strongly anchored, an ultrasonic instrument may be used to vibrate on the tap, and if necessary, the torque bar may be inserted into the handle port to increase leverage.

Masserann Kit

When a post is broken deep inside the root, the Masserann kit may be considered to conserve root structure (Figure 23-31).[56] A wide range of trephines manufactured with soft steel are available and the one matching the post space is

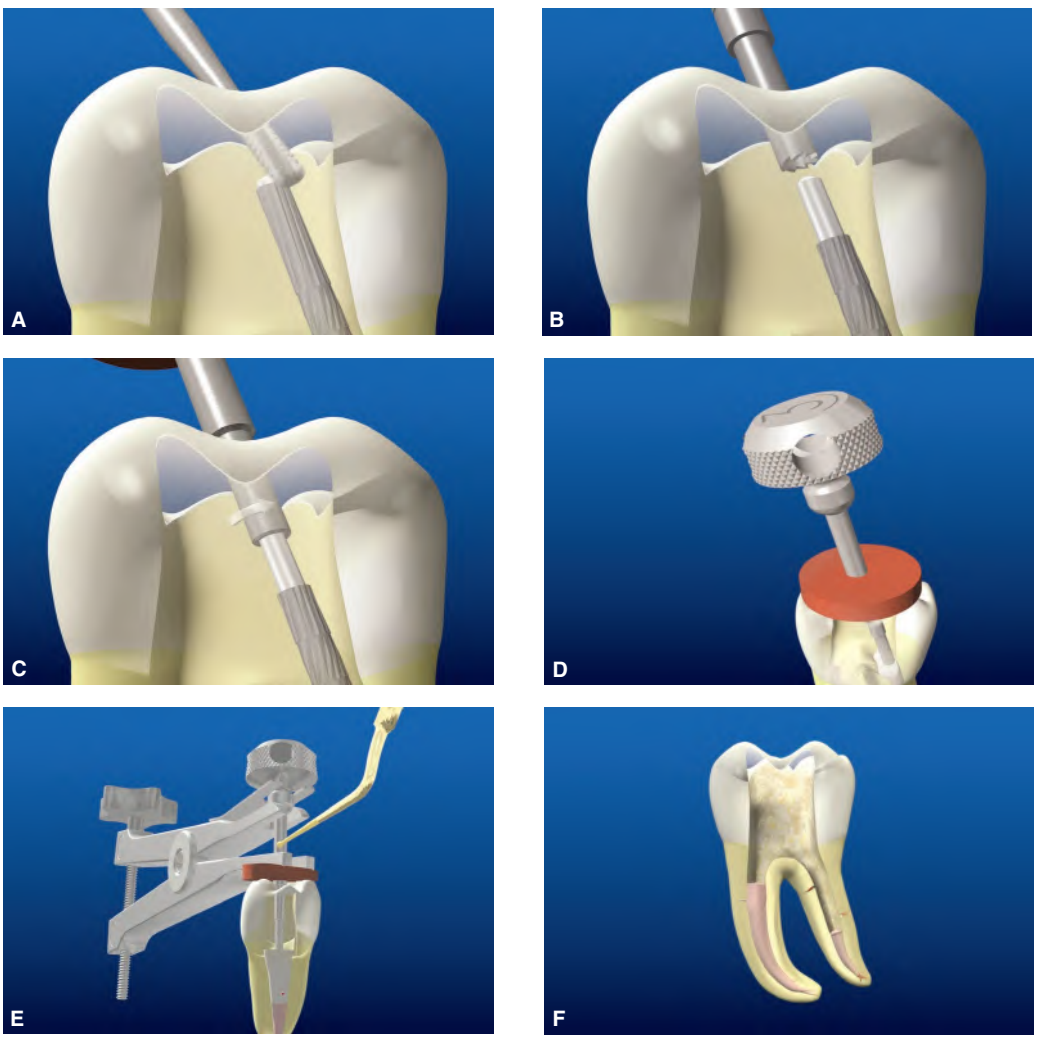


FIGURE 23-30 ***A.*** Illustration of a transmetal bur efficiently doming the post head. ***B.*** No. 3 trephine has precisely machined down the coronal 3 mm of the post. ***C.*** Tubular tap is turned counterclockwise to form threads, draw down, and strongly engage the post. ***D.*** Once the tap is securely engaged to the post, the rubber bumper is seated against the occlusal surface to protect the tooth. ***E.*** Mounted and activated PRS extracting pliers. Note, the energized ProUltra ENDO-1 can be advantageously placed against the post-engaged tap to synergistically facilitate the removal effort. ***F.*** Post removal. Attention can now be directed towards selecting the best gutta-percha removal scheme.

FIGURE 23-31 The Masserann kit.

selected. The trephine is mounted onto a mandrel and rotated CCW to create a thin trench around half the length of the post. During the procedure, the trephine must be regularly cleaned of dentine dust. Next, the smaller trephine is placed and used in a CCW motion around the broken post trying to reach the depth of the trench. The soft steel trephine deforms during apical progression and, at any given moment, the friction between the trephine and the post becomes greater than the retention of the post, allowing it to be removed. Such a procedure is time-consuming and the clinician must weigh risk vs. benefit when using this method.

MISSED CANALS

The etiology of endodontic failure is multi-faceted, but a statistically significant percentage of failures are related to missed root canal systems. Missed canals hold tissue, and at times microorganisms and related irritants that inevitably contribute to clinical symptoms and lesions of endodontic origin (Figure 23-32).[76–81]. Historically, and still too often, surgical treatment has been directed toward "corking" the end of the canal with the hopes that the retrograde material will incarcerate biological irritants within the root canal system over the life of the patient (Figure 23-33).[82,83] Although this clinical scenario occurs anecdotally, it is not nearly as predictable as nonsurgical retreatment. Endodontic prognosis is maximized in teeth whose root canal systems are cleaned, shaped, and filled in *all* their dimensions (Figure 23-34).[84–85]

Canal Anatomy

There are several tooth groups whose roots notoriously hold additional systems.[86,87]

- *Maxillary Central Incisors* on occasion may contain one or more extra canals.[88,89]
- *Maxillary 1st Premolars* are at times three-rooted and have mesiobuccal (MB), distobuccal (DB), and palatal root canal systems.[90]
- *Maxillary 2nd Premolars* contain broad roots buccal to lingual and, although the orifices are commonly ribbon-shaped, clinicians need to expect deep canal divisions or apical bifidities.[91]
- *Maxillary 1st Molars* have two systems in the MB root more than 90% of the time and these two systems oftentimes communicate anatomically via an isthmus.[92] According to the literature, about 40% of the time, the two root canal systems merge and exit as one apical portal of exit; about 60% of the time, the two canals are separate and have their own individual portals of exit. These systems can be identified and treated in over 75% of the cases *without* a microscope and in approximately 90% of the cases *with* a microscope.[93,94]
- *Maxillary 2nd Molars* should be suspected of having a second canal in the MB root until proven otherwise.[93] Some maxillary molars exhibit bizarre anatomy within the MB root; the grooves should be explored for the possibility of a third system.
- *Mandibular Incisors* have broad roots facial to lingual and have a second more lingual canal approximately 45% of the time.[95] Access cavities should be carried more lingual at the expense of the cingulum to address this potential system.
- *Mandibular Premolars* have roots that frequently hold complex root canal systems. The anatomical variations include displaced orifices, deep divisions, loops and branches, and multiple portals of exit apically.[96]
- *Mandibular 1st and 2nd Molars* routinely have significant variations within what has become known as "normal anatomy."[59,86] Clinicians need to check the mesial root for a third system that may be displaced or located within the groove between the MB and mesiolingual (ML) orifices.[97,98] The broad distal root commonly contains an extra canal that may be separated along its length or become contiguous, following cleaning and shaping procedures. However, even when distobuccal (DB) and distolingual (DL) systems become common, deep branching with multiple apical portals of exit are normal.
- *C-Shaped Molars* pose challenges in endodontic treatment; they tend to have fused roots, deep pulp chambers, and the cul-de-sac furcation is toward the lingual aspect. Clinicians need to be familiar with this aberrant canal form and be well aware of its radiographic features and its incidence within various population groups.[99]

Armamentarium and Techniques

There are multiple concepts, armamentarium, and instruments that are useful to find missed canals. The following represents the most important.

- *Anatomical familiarity*: Any dentist can become a serious student of endodontic anatomy by reviewing the historic work of Walter Hess or visualizing state-of-the-art μCT images (www.ehumanlearning.com). Knowledge, understanding, and appreciation for root canal system anatomy influence predictably successful endodontic treatment outcomes. For more details, see Chapter 1, "Anatomy and morphology of teeth and their root canal systems."
- *Radiographic analysis* is critical when evaluating an endodontic failure.[100,101] Well-angulated periapical films should be taken with the cone directed straight-on,

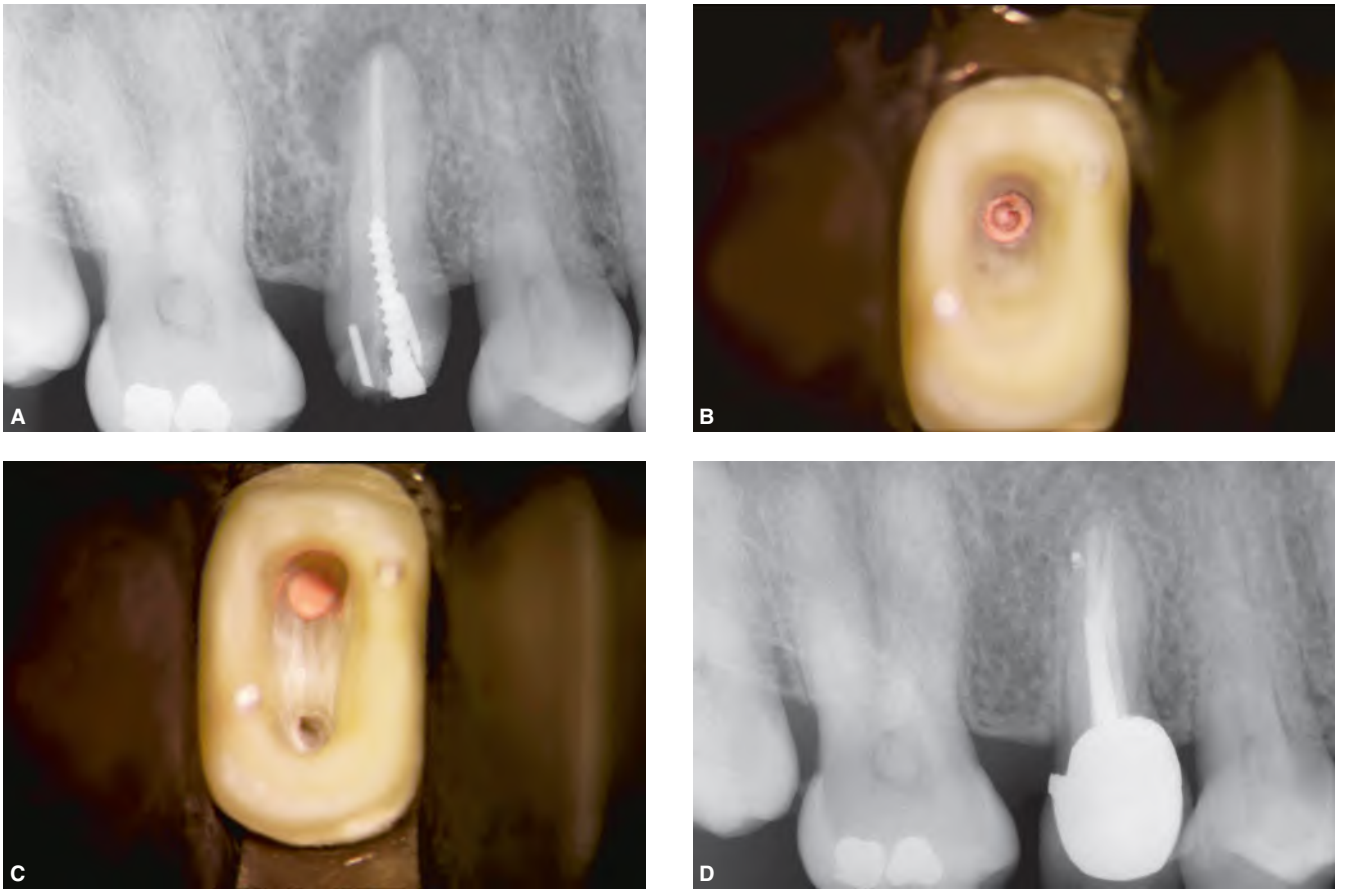


FIGURE 23-32 ***A.*** Radiograph of a maxillary right second premolar reveals pins, a post, incomplete endodontic treatments and an asymmetrical lesion. ***B.*** Photograph at 12× magnification shows the post is out of the buccal canal, thread marks in the gutta-percha from the screw post, and evidence of a missed palatal canal system. ***C.*** Photograph at 12× magnification demonstrates complete access and identification of the palatal orifice/system. ***D.*** Ten-year recall radiograph shows excellent osseous repair. This case demonstrates the importance of 3-D endodontics, and a well-designed and sealed restoration.

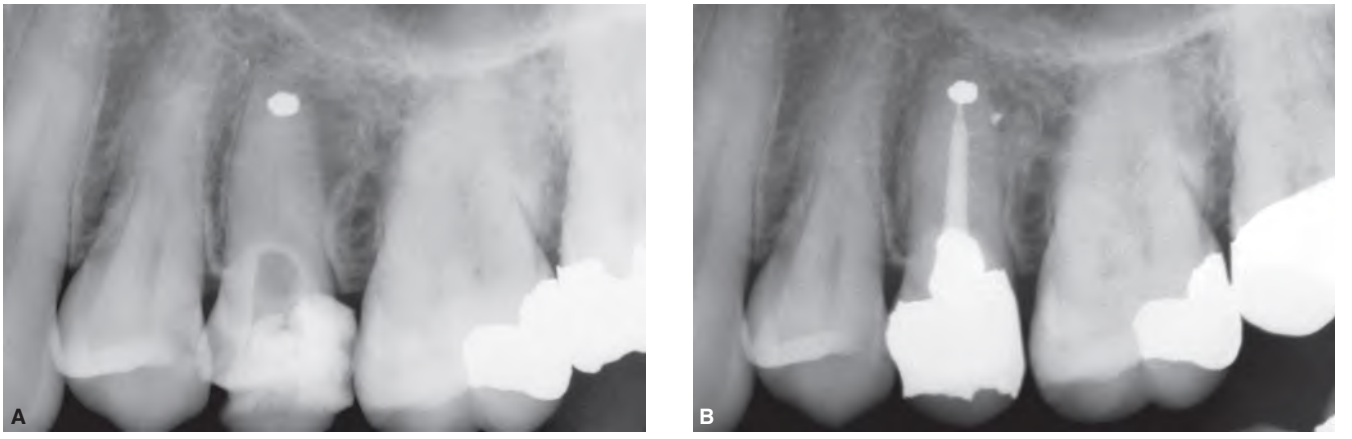


FIGURE 23-33 ***A.*** Preoperative radiograph reveals a failing treatment in a maxillary premolar with a laterally positioned lesion of endodontic origin. Note the "calcified" canal and history of surgery. ***B.*** One-year recall radiograph demonstrates evidence of bony healing and the importance of nonsurgical retreatment.

mesioblique, and distoblique. This technique oftentimes reveals and clarifies the three-dimensional morphology of the tooth. In teeth where complete endodontic treatment has been performed, obturation materials are seen radiographically as centered within the root, regardless of the selected angle of the radiograph. Conversely, if the obturation materials appear positioned asymmetrically within the long axis of the root, a missed canal should be suspected (Figure 23-32).[102] Digital Radiography (Kodak; Atlanta, Georgia) affords a variety of software features significantly

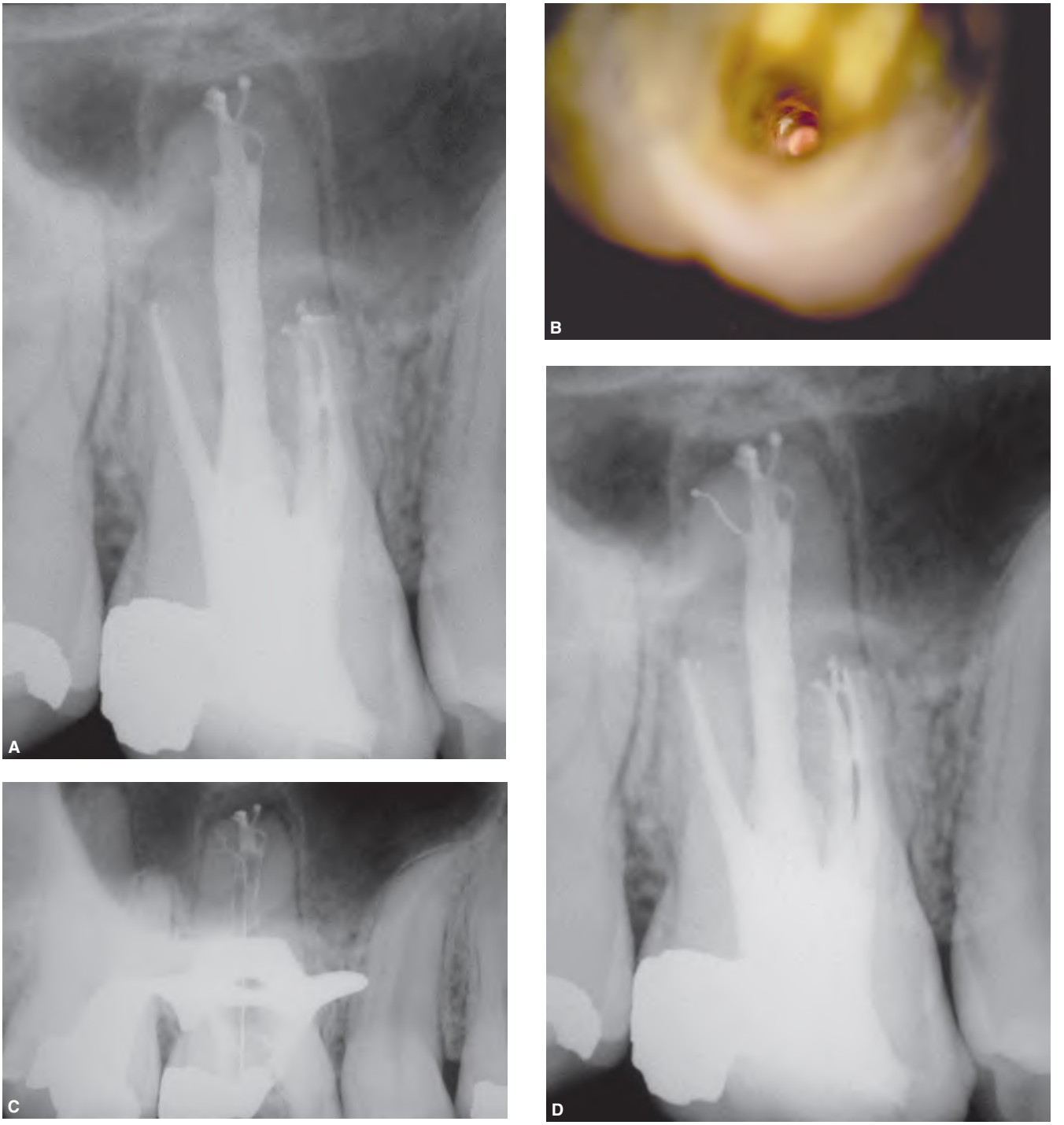


FIGURE 23-34 ***A.*** Radiograph of a maxillary right first molar with a failing treatment in the palatal root. ***B.*** A deep look into the apical one-third of the palatal canal reveals debris in the entrance to a lateral canal. ***C.*** Working radiograph demonstrates a no. 10 file moving through this ramification to its terminus. ***D.*** Post-treatment radiograph depicting the retreatment effort.

enhancing radiographic diagnostics in identifying hidden, calcified, or untreated canals. Certainly, CBCT technology represents a major advancement in radiographic diagnostics and facilitates identifying aberrant, mineralized, or previously missed canals.[18] For more details, see Chapter 9, "Imaging devices and techniques."

- *Vision* is enhanced with magnification glasses, headlamps, and transilluminating devices. The dental operating microscope (Carl Zeiss Surgical; Thornwood, New York, USA) affords extraordinary light and magnification, and gives the clinician unsurpassed vision, control, and confidence in identifying or chasing extra canals (Figure 23-34).[102] Operating microscopes typically have a range of magnification from about 2.5× to around 20×. Magnification plus lighting equals vision. Surgical length burs enhance direct vision by

importantly moving the head of the handpiece further away from the occlusal table and improving the line of sight along the shaft of the bur.

- *Access cavities* should be prepared and expanded so their smallest dimensions are dictated by the separation of the orifices on the pulpal floor and their widest dimensions are at the occlusal table. The isthmus areas and/or developmental grooves are firmly probed with an explorer in an effort to find a "catch" (Figure 23-35). In general, the access preparation should be prepared, so the operator can look in the mouth-mirror of a furcated tooth and visualize all of the orifices without moving the mirror. Importantly, axial walls should be flared, flattened, and finished to provide straightline access to the orifice(s).
- *Piezoelectric ultrasonics* provides a breakthrough for exploring and identifying missed canals (Figure 23-36). It should be fully appreciated that ultrasonic instruments perform optimally when they are designed, manufactured, and tuned for a specific generator. Synergistically, a piezoelectric generator (Newtron P5, Acteon USA; Mt. Laurel, New Jersey, USA) in conjunction with ultrasonic instruments (ProUltra ENDO Instruments, Dentsply Maillefer; Ballaigues, Switzerland) are utilized to transfer energy and perform a variety of clinical procedures.[103] Ultrasonic systems importantly eliminate the bulky head of the conventional handpiece that notoriously obstructs vision. The working ends of specific ultrasonic instruments are 10 times smaller than the smallest manufactured round burs and their abrasive coatings allow them to precisely sand away dentin when exploring for missed canals (Figure 23-24).
- *Micro-Openers* (Dentsply Maillefer; Ballaigues, Switzerland) are flexible, stainless steel hand instruments that feature ergonomically designed off-set handles. Micro-Openers have limited length cutting blades that, in conjunction with their .04 and .06 tapers, enhance tensile strength, making it easier to locate, penetrate, and perform initial canal enlargement procedures. These instruments provide unobstructed vision when operating in difficult teeth with limited access (Figure 23-37).
- *Various dyes*, like methylene blue, can be irrigated into the pulp chambers of teeth to aid in diagnosis. The chamber is subsequently rinsed thoroughly with water, dried, and visualized. Frequently, the dye will be absorbed into orifices, fins, and isthmus areas that serves to "roadmap" the anatomy. This technique can aid in the identification and treatment of missed canals and enhance diagnostics, including the visualization of fractures.
- *Sodium hypochlorite (NaOCl)* can aid in the diagnosis of missed or hidden canals. This has been called the *"champagne test."*[8] Following cleaning and shaping procedures, the access cavity is flooded with NaOCl and the solution is observed to see if oxygen bubbles are emanating toward the occlusal table. A positive *"bubble"* reaction signifies that sodium hypochlorite is either reacting with residual tissue within a missed canal or with residual chelator that is still present within a canal being prepared.[56]
- *Transillumination* is accomplished by positioning a fiber optic wand either above or below the dental dam and directing light buccal to lingual. Diagnostics are, at times, improved by turning off the overhead or microscope light source to achieve a different perspective.
- *Explorer Pressure*: The endodontic hand-held explorer should be strong, thin, and have a durable pointed tip. Firm explorer pressure provides a safe way to punch through a thin layer of secondary dentin and expose a hidden, receded, and more mineralized canal.
- *White Line Test:* Oftentimes, a shelf of dentin meets the pulpal floor and forms a groove. It is important to recognize that extra canals can be more readily identified by progressively sanding away this shelf of dentin with abrasively coated or micro-milled ultrasonic instruments. During ultrasonic procedures in necrotic teeth, dentinal

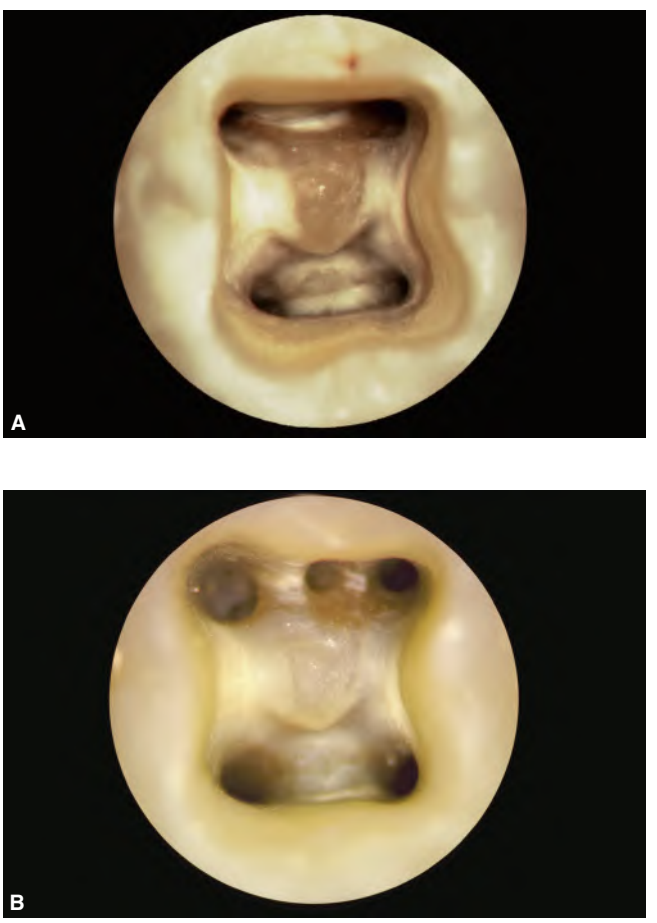


FIGURE 23-35 ***A.*** A view into an access cavity suggests that there are apparently four orifices/systems in this mandibular first molar. ***B.*** Photograph demonstrating a third mesial orifice and the importance of exploring the isthmus connecting the mesiobuccal and mesiolingual systems.

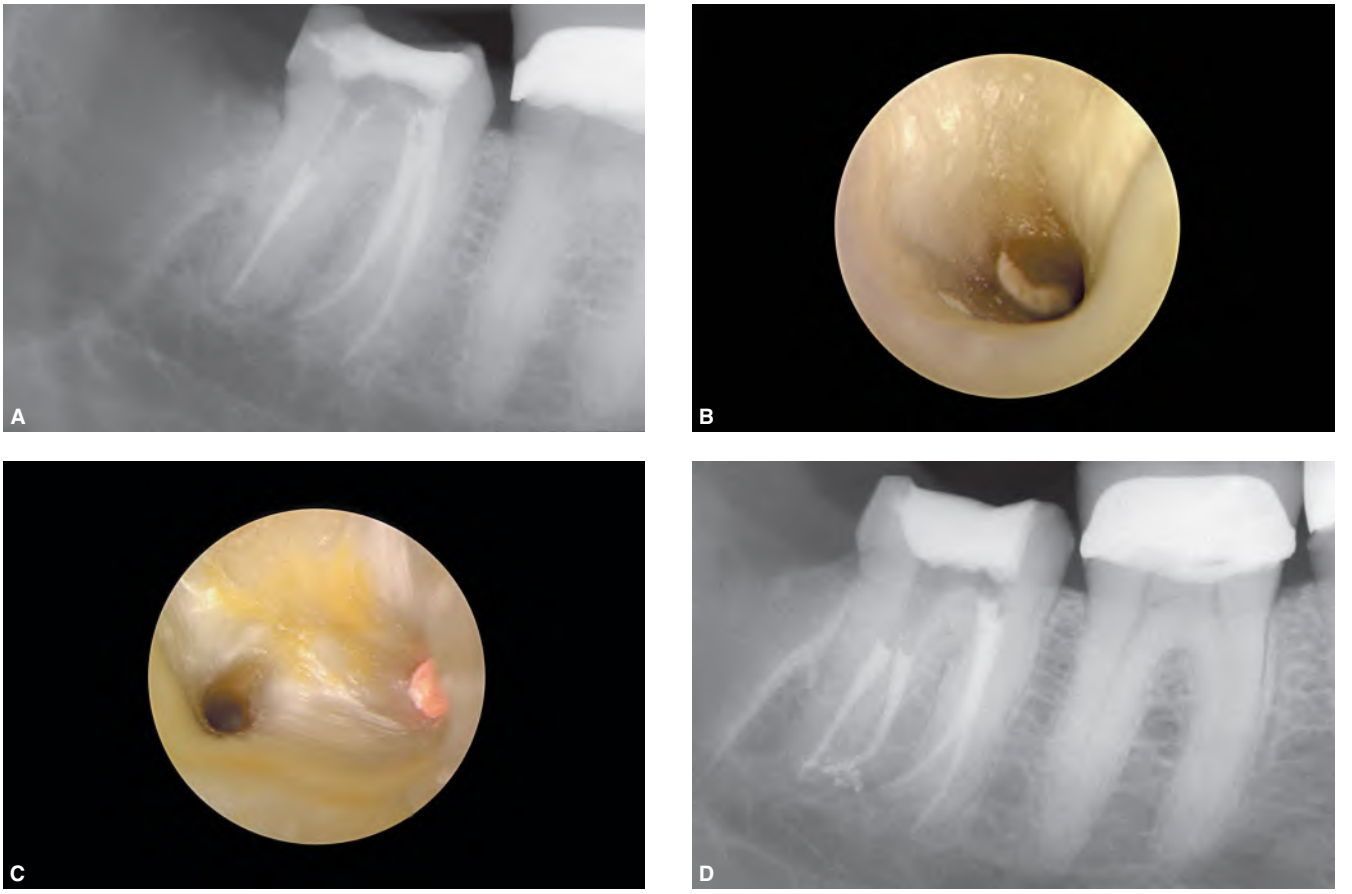


FIGURE 23-36 ***A.*** Radiograph showing a mandibular right second molar with three canals treated endodontically. The obturation material is not centered within the distal root. ***B.*** A view into the centered distal orifice reveals obturation material positioned subcrestally in a fin of this canal. ***C.*** Photograph showing improved straightline access to the DL orifice and gutta-percha deep within the previously treated DB system. ***D.*** Post-treatment radiograph demonstrates the packed DL root canal system.

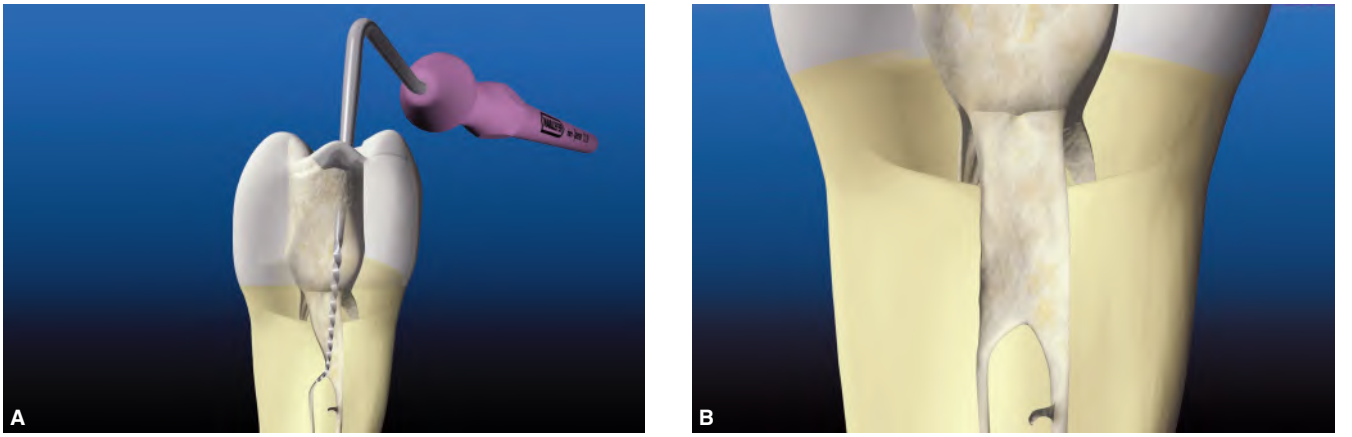


FIGURE 23-37 ***A.*** A distal view of a mandibular molar animation. The Micro-Opener's offset handle affords unobstructed vision and is utilized to initiate treatment in areas where access is difficult and restricted. ***B.*** Micro-Opener has carved away the triangle of dentin creating straightline access to the second distal system.

dust moves into available anatomical space, such as the isthmus, and forms a visible white line. The white line test is a visible road map that can be followed and diagnostically aid in identifying, as an example, an MB2 orifice/canal.

- *Red Line Test:* In vital cases, blood frequently moves into an isthmus area. Like a dye, blood absorbs into orifices, fins, and isthmuses, and serves to roadmap and aid in the identification of the underlying anatomy. This is called the red line test.

- *Restorative Disassembly*: Removing a full-coverage dental restoration provides direct visualization of the underlying tooth preparation. Coronal disassembly improves the predictability of safely entering the pulp chamber and identifying any given orifice.
- *Perio-Probing* is another important idea for locating canals. Probing the sulcus can provide important information as to the relationship between the long axis of the clinical crown and the underlying root.
- *Symmetry* is an important visual test that can encourage looking for another orifice/canal or confirm that all canals have been properly identified. The rules of symmetry suggest that if any given root contains only one canal, then regardless of its anatomical configuration, the orifice should be positioned an equal distance from the external cavo surface of the root.[104]
- *Color* roadmaps the anatomy. Oftentimes, a dark groove on the pulpal floor of a multi-canal tooth can be followed and will lead to another canal orifice. Additionally, orifices frequently appear a darker color than the surrounding dentin in teeth exhibiting calcification.

Persistent apical periodontitis is often the result of a missed MB-2 canal in maxilllary molars. If discovered, missed canals can usually be three-dimensionally cleaned, shaped, and packed. However, if a missed canal is suspected but cannot be readily identified, then an endodontic referral may be prudent to avoid further complications. Caution should be exercised when contemplating surgery due to the aforementioned concerns; but at times, surgery may be necessary in the hopes of salvaging the tooth.

GAINING ACCESS TO THE CANAL TERMINUS

Removal of Filling Materials

Once the coronal obstacles have been removed, intracanal obstacles, mainly radicular filling materials, should be eliminated before attempting to negotiate the unfilled portion of the root canal system (Figure 23-38). Apical to the previously placed obturation material, it is normal to encounter a variety of mishaps, such as blocks, ledges, fractured instruments, transportations, and perforations. Utilizing illumination and magnification with the operating microscope, dedicated ultrasonic tips, MTA, and bioceramic cements have served to push the limits of therapeutic possibilities, enabling successful retreatment and salvaging many cases that would not have been treatable in the past.

The obturation materials most frequently found in the root canal space generally reflect a past era of knowledge, a current school of thought, or a personal philosophy of treatment. Experience reveals there are four commonly encountered materials found in obturated root canal systems. These materials are *gutta-percha*, *carrier-based obturators*, *silver points* and *paste fillers*. In addition, new obturation materials have recently been recommended for filling root canal systems; as an example, resin-based materials can generally be removed using the same technique as will be outlined for the removal of gutta-percha. Frequently it is necessary to remove

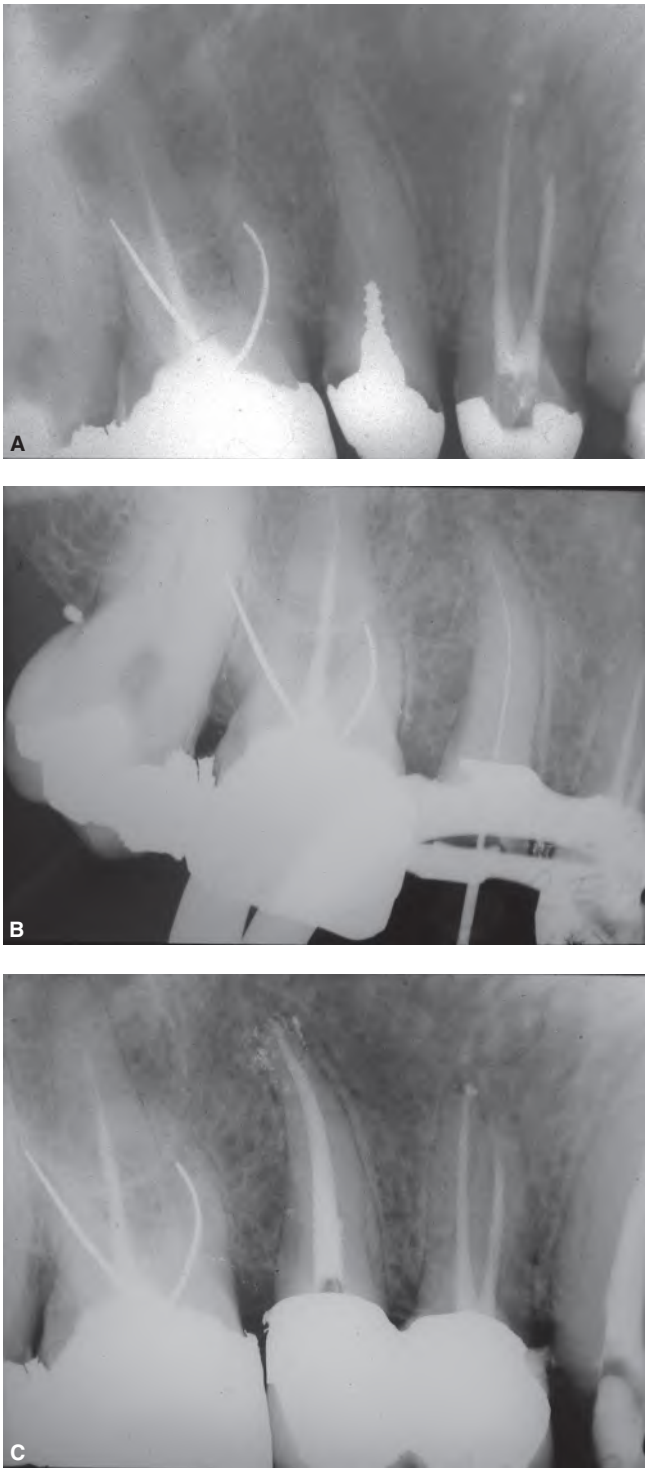


FIGURE 23-38 ***A.*** The maxillary second premolar is scheduled to be retreated before placement of a new restoration. Coronal and intracanal obstacles must be removed. ***B.*** The root canal is negotiated. ***C.*** Postoperative radiograph demonstrating obturating material in a lateral canal and apical ramifications.

an obturation material to achieve endodontic retreatment success or to facilitate placing a post for restorative reasons. The effective and efficient removal of an obturation material requires utilizing the most predictable methods from the past, in conjunction with the timely integration of the best presently developed techniques.

Gutta-Percha Removal

The relative difficulty in removing gutta-percha varies according to the canal's length, cross-sectional diameter, and curvature. Regardless of technique, gutta-percha is best removed from a root canal in a *progressive* manner to avoid the risk of definitive canal blockage and prevent inadvertent displacement of irritants periapically. Dividing the root into thirds, gutta-percha is initially removed from the canal in the coronal one-third, then the middle one-third, and finally eliminated from the apical one-third. In canals that are relatively large and straight, single cones can, at times, be removed with one instrument in one motion (Figure 23-39). For other canals, there are a number of possible gutta-percha removal schemes. The techniques include rotary files, ultrasonic instruments, heat, hand files with heat or chemicals, and paper points with chemicals.[105] Of these options, the best technique(s) for a specific case is selected based on preoperative radiographs and clinically assessing the diameter of the orifices after re-entering the pulp chamber. Certainly, a combination of methods is generally required, and, in concert, provide safe, efficient, and potentially complete elimination of gutta-percha and sealer from the internal anatomy of the root canal system.

Rotary Instruments

Rotary instrumentation is the most efficient method for removing gutta-percha from a previously treated root canal (Figure 23-40). The first step is to assess precisely the level of the previous obturation. The ProTaper Retreatment Kit (Dentsply Maillefer; Ballaigues, Switzerland) is an innovative system comprised of 3 nickel titanium (NiTi) instruments that vary in length (16 mm, 18 mm, and 22 mm respectively for D1, D2 and D3), diameter, and taper (30/09, 25/08, 20/07) (Figure 23-41). Rotary instruments should be used with caution in underprepared canals and are generally not selected for removing gutta-percha in canals that do not accept them *passively*. When attempting gutta-percha removal, it is useful to mentally divide the root into thirds and then select two or three appropriately-sized rotary instruments that will fit passively within these progressively smaller regions.

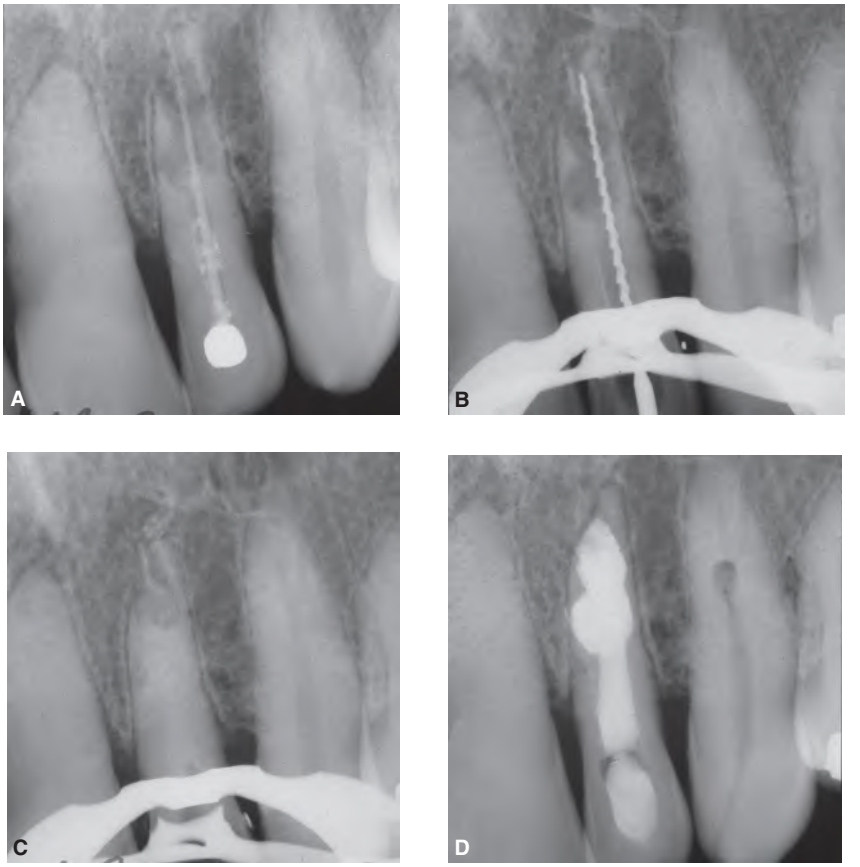


FIGURE 23-39 ***A.*** Radiograph showing a poorly sealed lateral incisor with a gutta- percha cone floating in the canal. An extensive internal resortion perforates the root in apical and mesial areas. ***B.*** In relatively large and straight canals, single gutta-percha cones can be removed with one H-file in one motion. ***C.*** The root canal was medicated with calcium hydoxyde for three months. ***D.*** Postoperative radiograph after root canal filling demonstrating excellent repair and formation of a new periodontal ligament in the perforated areas.

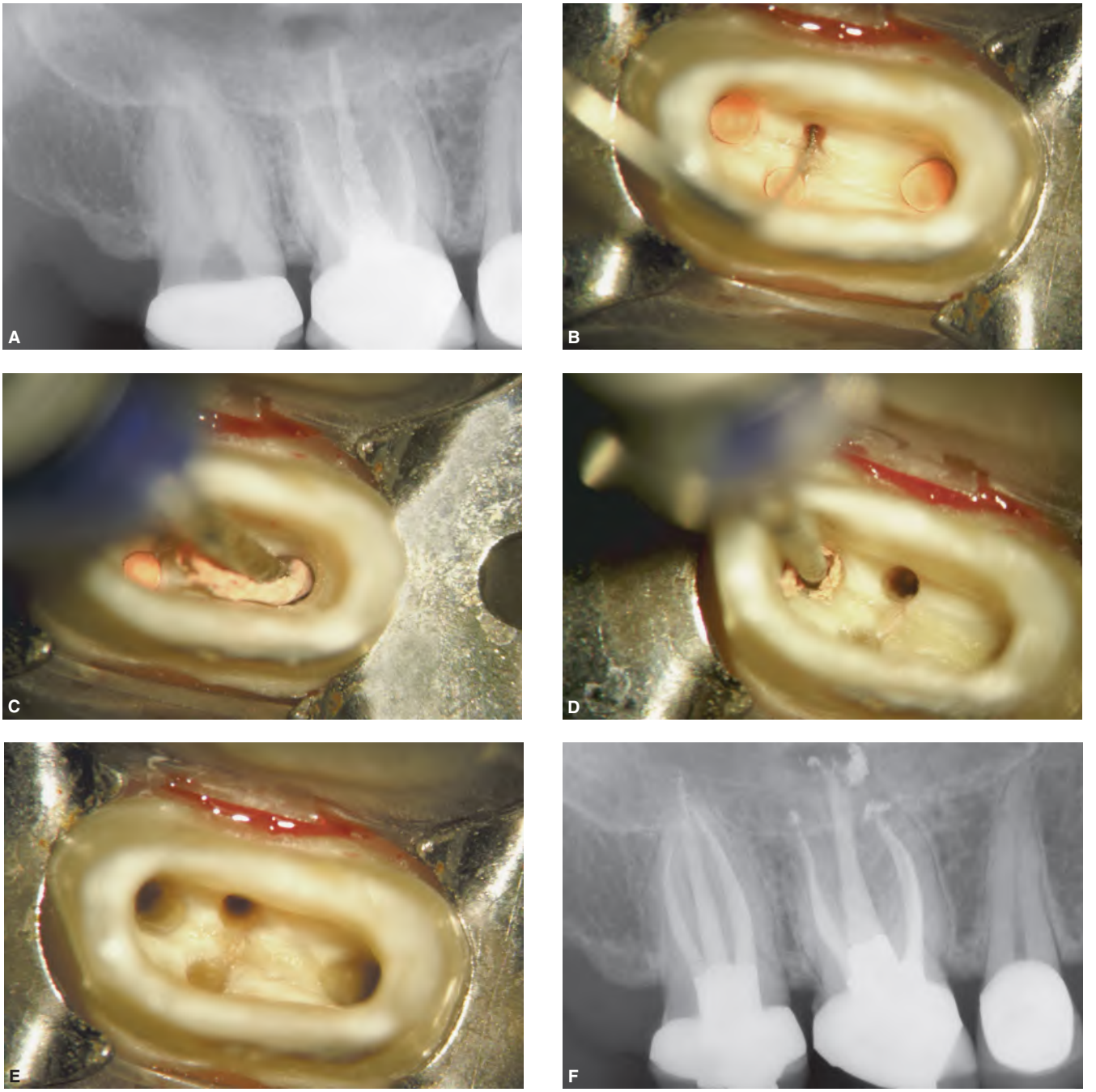


FIGURE 23-40 ***A***. Radiograph of a failing endodontic treatment in a maxillary right first molar showing that the previously treated canals are underfilled and underextended. ***B***. Photograph at 15× magnification demonstrates crown removal, straightline access, and the identification of an MB-2 system. ***C***. Photograph at 15× magnification demonstrates removal of gutta-percha from the palatal canal with a .06 tapered NiTi rotary profile. ***D***. Removal of gutta-percha from the mesiobuccal canal with a smaller sized .06 tapered NiTi rotary profile. ***E***. Pulpal floor and orifices following canal preparation procedures. ***F***. Postoperative radiograph demonstrating the value of nonsurgical retreatment and its potential to address root canal systems and improve quality of treatment.

To mechanically soften and engage gutta-percha, rotary instruments must turn at speeds ranging from 800 to 1200 rpm. Ultimately, the rotational speed selected is based on the friction required to mechanically soften and effectively remove gutta-percha coronally, owing to the fluted design of the instrument. Using a well-angulated radiograph to precisely assess the level of the previous obturation, care should be taken to use short pecking motions to engage the gutta-percha. The file should be removed and frequently inspected to assure filling material can be visualized on the apical flutes. Following initial engagement of the gutta-percha, a brushing motion on the outstroke helps improve its removal. If resistance to progression is felt, a radiograph should be taken to assess if all material has been removed or not. If there is no

FIGURE 23-41 The ProTaper Retreatment Kit *(Dentsply Maillefer; Ballaigues, Switzerland)*.

radiographic evidence of residual filling material, it is wise to continue the procedure with stainless steel hand K-files.

Heat

A power source, in conjunction with specific electric heat carrier instruments (Touch'n Heat 5004, SybronEndo; Orange, California, USA or Calamus, Dentsply Maillefer; Ballaigues, Switzerland), may be used to thermosoften and remove "bites" of gutta-percha from root canal systems. The cross-sectional diameter of the heat carrier limits its ability to plunge into underprepared canals and around pathways of curvature; however, in larger canals, this method works quite well. The technique is to activate the instrument so that it is red-hot and then plunge it into the coronal-most aspect of the gutta-percha. The heat carrier is then deactivated and, as it cools, will typically freeze a bite of gutta-percha on its working end. Instrument withdrawal generally results in the removal of an attached bite of gutta-percha. This process is repeated as long as it continues to be productive.

File and Chemical

The file and chemical removal option is best utilized to remove gutta-percha from small, curved, and densely filled canals. A pilot hole is first created in the coronal portion of the canal with either a hot heat carrier or by using the Protaper Retreatment D1 instrument. Two or three drops of chloroform are then introduced into the newly created reservoir inside the root canal. Chloroform is still the reagent of choice and plays an important role in chemically softening gutta-percha.[106–109] A sequential technique is advocated to remove the filling material. It involves the use of H-type files, 21 mm in descending order to gently "pick" into the chemically softened gutta-percha. Shorter instruments are preferred since they provide more stiffness and have less tendency to flex. Without trying to engage the more apical material, a lateral motion is used with the H-file to remove gutta-percha remnants at this level. This method is repeated to progress apically until gutta-percha is no longer evident on the cutting flutes of the file when it is withdrawn from a solvent-filled canal. Such a progressive removal technique helps prevent the needless extrusion of chemically softened gutta-percha periapically. Once the "file and chemical" gutta-percha removal technique has been completed, clinicians need to appreciate that they still need to address the residual sealer and gutta-percha that remain in the irregularities of the root canal system.[105] The wicking technique and canal re-instrumentation will overcome this problem.

Paper Point and Chemical

Gutta-percha and most sealers are miscible in chloroform and solvents and, once in solution, can be absorbed and removed with appropriately-sized paper points. Drying solvent-filled canals with paper points is known as "wicking" and is *always* the final step of gutta-percha removal.[3] The wicking action is essential in removing residual gutta-percha and sealer out of fins, cul-de-sacs, and aberrations of the root canal systems. In this technique, the canal is first flushed with chloroform and the solution is then absorbed and removed with appropriately-sized paper points. Paper points "wick" by pulling dissolved materials laterally into the shaped canal. Chemical flushing and wicking procedures more effectively liberate residual gutta-percha and sealer from the root canal system (Figure 23-42D). This process is repeated as long as it continues to be visibly productive. Alternatively, the EndoActivator is a powerful adjunct to help eliminate residual sealer from the canal walls. Solvent is flooded into the pulp chamber before using the EndoActivator. A preselected polymer tip is activated and serves to vigorously agitate the solvent, create turbulence, and enhance the cleaning efficiency. Paper points are then used to absorb the dissolved materials.

Even when paper points come out of the canal clean, white, and dry, the clinician should assume residual gutta-percha and sealer are still present. At this point, the chamber is again flooded with chloroform, but it is now introduced with more of an irrigation and vacuum "flushing action." The irrigating canula is placed below the orifice and the solvent is passively and repeatedly irrigated, then aspirated. This alternating method of irrigating-then-aspirating creates a vigorous back-and-forth turbulence that powerfully promotes the elimination of the root canal filling materials. Following chloroform wicking procedures, the canal is liberally flushed with 70% isopropyl alcohol and wicked to further encourage the elimination of chemically softened gutta-percha residues (Figure 23-42D). Removal of all residual material, in this manner, will enhance the efficacy of sodium hypochlorite when it is utilized during subsequent cleaning and shaping procedures.

Carrier-Based Gutta-percha Removal

Carrier-based obturators were originally metal and file-like, yet over the past several years, they have been manufactured from easier-to-remove plastic materials. Recently, a cross-linked gutta-percha core has replaced the plastic carrier, making retreatment easier.[110] Metal carriers, although no longer distributed, are occasionally encountered clinically and are more difficult to remove

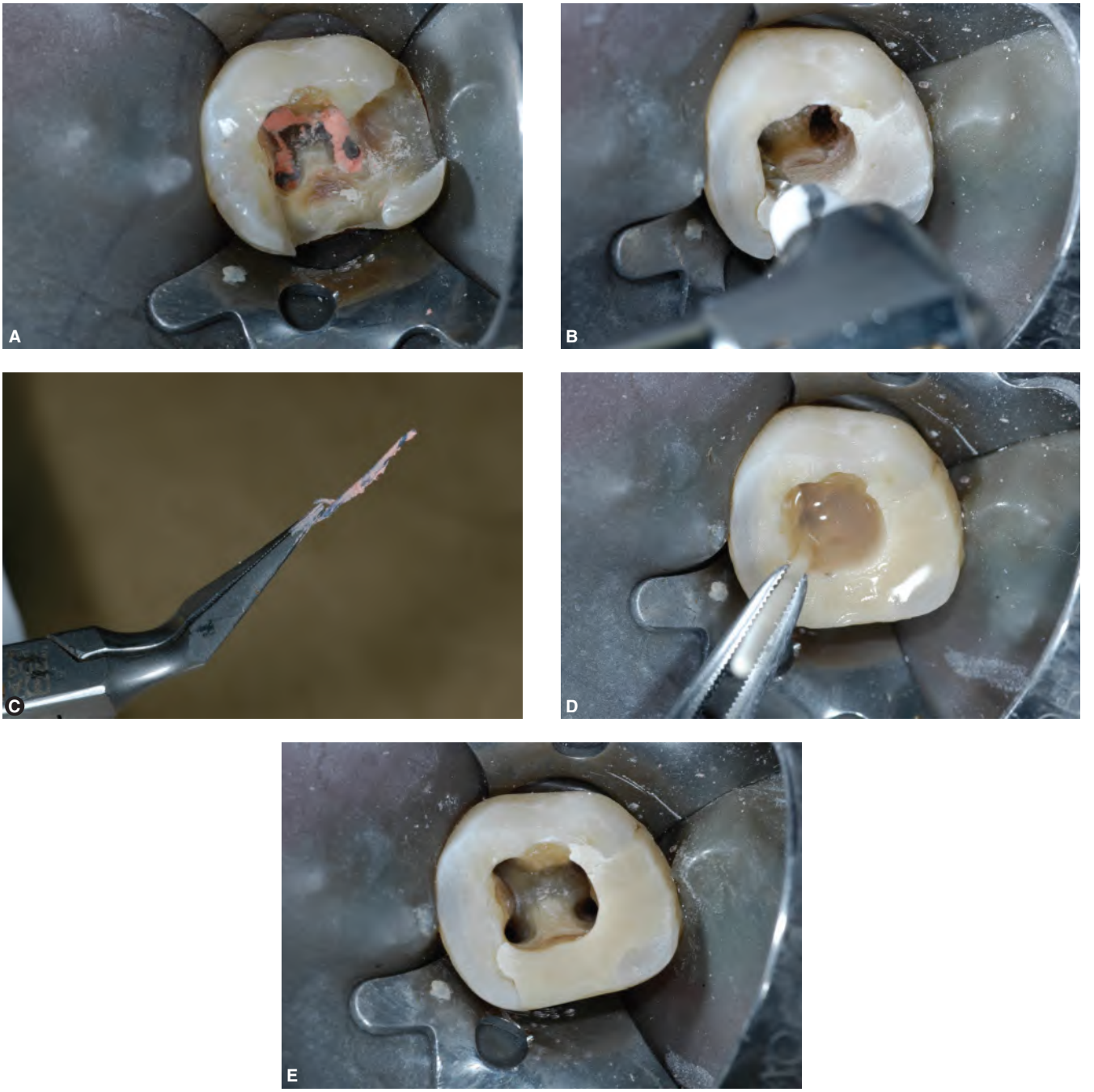


FIGURE 23-42 ***A.*** After removal of the coronal restoration, plastic carriers are clearly seen in the canals of this mandibular molar. ***B.*** Following pre-treatment and access cavity, a Protaper Retreatment instrument is used to remove the plastic carrier in the ML canal. ***C.*** Plastic carrier was removed and grasped with Stieglitz pliers. ***D.*** The wicking procedure. The pulp chamber is filled with solvent and a paper point absorbs the residual sealer. ***E.*** Cleanliness of the pulp chamber and canals before obturation.

than silver points because their cutting flutes, at times, engage lateral dentin.

Following careful access and complete circumferential exposure of the carrier, suitable grasping pliers are selected and a purchase is obtained on the carrier. When possible, the relative tightness of the carrier within the canal can then be tested utilizing pliers. Successful removal in these cases is enhanced by recognizing that the carrier is frozen in a sea of hardened gutta-percha. With this in mind, the following techniques are used to remove carriers:

- Rotary instrumentation can be used to effectively and efficiently auger a plastic carrier out of a canal. This should only be attempted if there is sufficient space between the carrier and the canal walls. A ProTaper Retreatment D2 instrument is placed at the carrier groove location and rotated at 800 rpm with light pressure. The frictional heat melts the gutta-percha that allows the instrument to advance apically. Owing to the greater taper, the instrument will bind between the plastic carrier and the dentin, allowing the carrier to be extricated coronally (Figure 23-42).

- Another method involves the use of a heat carrier to be placed alongside the carrier to produce heat and thermosoften the gutta-percha in the coronal aspect. This step should be immediately followed by a forceful insertion of an H-file into the softened gutta-percha to grasp the carrier, and then a pulling action to retrieve it.
- Using solvents to remove plastic carriers is also effective and quite predictable. Chloroform will chemically soften the gutta-percha and allow H-files to work deeper, progressively undermining and loosening a carrier for removal.[111]
- The File Removal System (FRS) may be considered, in certain cases, to remove a carrier. This method of removal is especially appropriate if the core of the carrier is metal and has cutting flutes that are engaging lateral dentin.

If any of these efforts are successful, the carrier will be extricated from the canal. The canal can then be retreated, as if it were a gutta-percha case, using any of the gutta-percha removal strategies followed by chemical flushing and paper point wicking procedures.

Paste Removal

A great variety of paste types exists with the difference being chemical formulation. Originally, paste use was intended for those patients who could not afford conventional endodontics and this modality of treatment was considered a benevolent alternative to extraction. Regretfully, countless cases were unsuccessful and too often the "magic" paste was used with hopes of overcoming deficiencies in the removal of tissue and related irritants during canal preparation (Figure 23-43).

When evaluating a paste case for retreatment, it is useful to clinically understand that pastes can generally be divided into soft, penetrable, and removable versus hard, impenetrable, and at times, unremovable.[112,113] Typically, the paste used in the United States remains soft within the canal and is readily removed. Notoriously, the whitish-colored paste oftentimes used in Russia and the reddish-brown resin paste commonly utilized in Eastern Europe and the Pacific Rim pose formidable challenges in removal because they set-up brick-hard. However, it is important to understand that, due to the method of placement, the densest portion of the paste in the canal is coronally and the material is progressively less dense moving apically (Figure 23-44). In addition, retreatment of paste-filled cases is often wrought with surprises, as clinicians frequently encounter calcifications, resorptions, and flare-ups, which should be anticipated and communicated.

Solvents and Hand Files

Reagents like Endosolv "E" or DMS IV (Dentsply Maillefer; Ballaigues, Switzerland) and Endosolv "R" (Endoco; Memphis, TN, USA) can be helpful in chemically softening soluble and hard pastes.[113] The "R" designates the solution of choice for the removal of resin-based pastes and the "E" and DMS IV are the solution for the elimination of eugenate-based pastes (Figure 23-45). For soluble pastes, the floor of the access cavity is first cleared of paste residues with an ultrasonic tip and then flooded with the solvent. A DG16 explorer firmly probes the canal orifice and brings the solvent in contact with the paste. If a good catch is felt, the efficiency of the solvent is confirmed. The technique to be implemented is similar to that described for gutta-percha removal (see *File and Chemical*

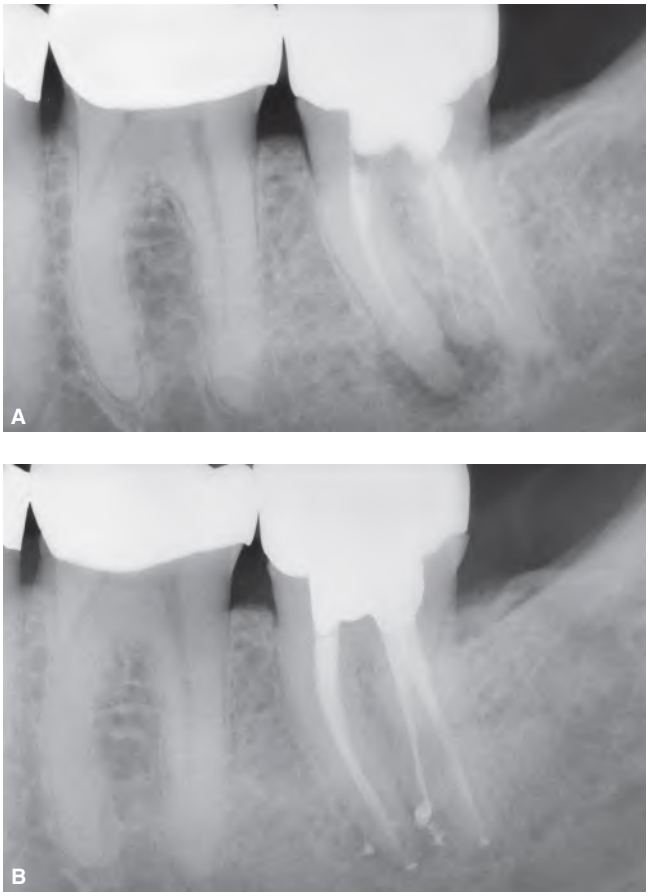


FIGURE 23-43 ***A.*** Preoperative radiograph of a failing endodontic treatment with a paste canal filling in a mandibular left second molar. Note the extra DL root. ***B.*** Five-year recall radiograph showing the treatment of multiple apical portals of exit and excellent osseous healing.

FIGURE 23-44 An illustration demonstrating that the decrease in density of paste-fillers is usually directly proportional to the length of the filling.

FIGURE 23-45 Endosolv "E" is a solvent for eugenate-based pastes whereas Endosolv "R" is the solvent for resin-based pastes.

Removal). Depending on the canal volume, 21 mm H-files are used in descending order to progressively eliminate the material. Regular renewal of the solvent is needed.

For hard pastes like epoxy resins, bakelites, glass ionomers, and zinc phosphate, no efficient solvents are available.[112,113] Alternatively, reagents can be placed interappointment against a paste-type material via paper points or cotton pellets to promote shrinkage and potentially facilitate subsequent removal.[113] The best method to remove them is with ultrasonics.

Ultrasonic Energy

Ultrasonic instruments, in conjunction with the microscope, afford excellent control in removing paste from the straight-away portions of a canal.[114,115] Specifically, the ProUltra ENDO-3, 4, and 5 zirconium nitride-coated ultrasonic instruments may be used below the orifice to remove brick-hard resin-type paste (Figure 23-46). To remove paste apical to a curvature, small-sized hand files must be used first to negotiate this region of the canal. Once a pilot hole has been created, then an appropriately-sized precurved file may be attached to a specially designed adapter (File Adapter 11, SybronEndo; Orange, California, USA) that mounts onto and is activated by the ultrasonic handpiece.

Heat

Certain resin pastes soften with heat. Heat carriers can be selected if this modality of removal is chosen.

Rotary Instruments

Stainless steel .02 tapered hand files may be used to negotiate through paste fillers. These files can potentially create a pilot hole for safe-ended NiTi rotary instruments to follow and effectively auger the toxic material coronally. The procedure is as follows.

1. On a well-angulated pre-op radiograph, precisely asses the endpoint of the previous obturation.
2. Use solvent and hand files, or rotate the Protaper Retreatment D1 file to create a pilot hole.

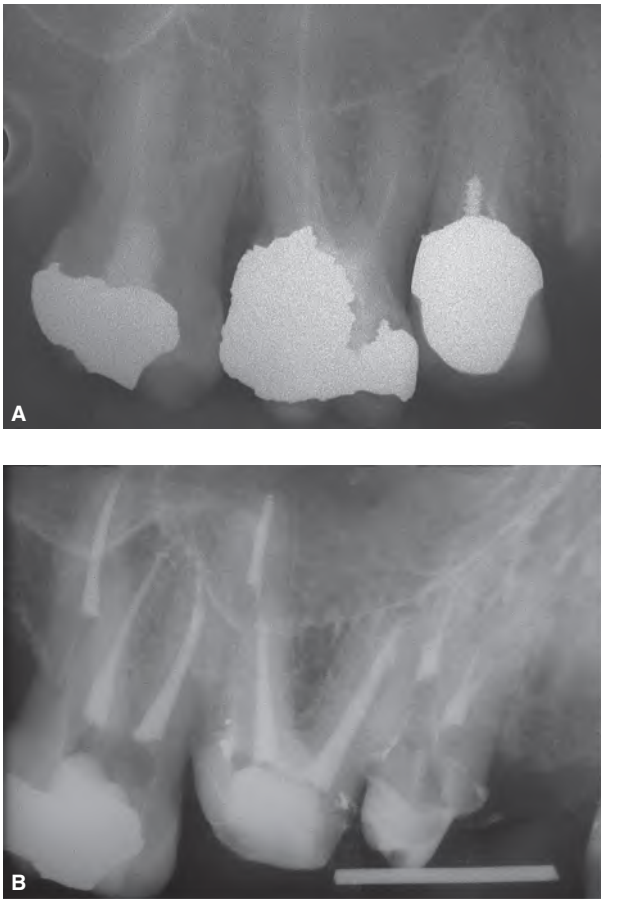


FIGURE 23-46 ***A.*** The maxillary premolar and molars teeth have been filled with a resin-type paste and must be retreated. ***B.*** Ultrasonic tips have been used in the straight portion of the canals to remove the brick-hard resin type paste. Deviation occured in the buccal canal of the premolar. At this stage, the procedure was stopped and the canal filled.

3. Use the D2 and D3 files with a short pecking motion at 500 rpm and check for the presence of material along the active portion of each file's more apical extent.
4. If resistance is met, go to stainless steel hand K files.

Dangerous, but at times helpful, is the use of end-cutting NiTi rotary instruments (Quantec NiTi Rotary Instruments, SybronEndo; Orange, California, USA) to penetrate paste.

Micro-Debriders

After removing the paste filler and shaping the canal, it is axiomatic that residual paste will still be noted within the irregularities of the root canal preparation. Micro-Debriders (Dentsply Maillefer; Ballaigues, Switzerland) are specially designed instruments to precisely remove residual paste materials from a root canal system. These stainless steel instruments enhance vision due to their ergonomically designed offset handles, have D_0 diameters of 0.20 and 0.30, and are available in .02 tapers with 16 mm of efficient Hedstrom-type cutting blades (Figure 23-47).

FIGURE 23-47 Micro-Debriders have offset handles to enhance vision, 16 mm of efficient Hedstrom-type cutting blades, and are used to remove residual obturation materials from the irregularities of canal systems.

Solvents and Paper Points

Following paste removal, chemical flushing with solvents, in conjunction with paper point wicking, is important to further remove and liberate material from the irregularities of the root canal system.

Silver Point Removal

The relative ease of removing failing silver points is based on the fact that chronic leakage, breakdown, and bacterial infection greatly reduce the seal, and hence, the lateral retention. Before a given silver point retrieval technique is selected, it is useful to recall the canal preparation prescribed for this method of obturation. Typically, the apical 2 to 3 mm of the canal was prepared relatively parallel and then flared coronal to this apical zone. When clinicians evaluate silver point failures, they should recognize that the silver point is parallel over length, hope for a coronally shaped canal, and take advantage of this space discrepancy when approaching retreatment.[116] Many techniques have been developed for removing silver points, primarily due to their varying lengths, diameters, and positions they occupy within the root canal space.[3,117–119] Certain removal techniques evolved to address silver points that bind in unshaped canals over distance. Other techniques arose to remove silver points with large cross-sectional diameters, approaching the size of smaller posts. Finally, other techniques are necessary to remove the split cone or intentionally sectioned silver points lying deep within the root canal space.

Access

Typically, the coronal heads of silver points are within pulp chambers and generally entombed in cements, composites, or amalgam cores. Access preparations must be thoughtfully planned and carefully performed so as to minimize the risk of inadvertently foreshortening the silver points (Figure 23-48). Initial access is accomplished with high-speed, surgical-length cutting tools. Subsequently, ultrasonic

FIGURE 23-48 ***A.*** Illustration of a mandibular molar reminding the clinician how silver points are commonly entombed within various restorative materials. ***B.*** When caution is not exercised upon re-entering the pulp chamber, silver points may be inadvertently foreshortened.

instruments may be carefully utilized within the pulp chamber to brush-cut away restorative materials and progressively expose the silver point.

Pliers Removal

After completing access and fully exposing the part of the silver point confined to the pulp chamber, a suitable grasping instrument (Stieglitz Forceps, Hu-Friedy; Chicago, Illinois, USA) is selected. To identify the best removal strategy, obtain a strong purchase on the silver point and gently pull to confirm its relative tightness. Oftentimes, dentists make the mistake of over-manipulating the head of the silver point, which needlessly foreshortens it, making subsequent retrieval efforts difficult, if not impossible. When grasping a silver point, rather than trying to pull it straight out of the canal, the pliers are rotated and levered, using fulcrum mechanics, against the restoration or tooth structure to enhance removal (Figure 23-49).[3]

Indirect Ultrasonics

When a segment of a silver point is encountered below the orifice and space is restricted, the appropriate

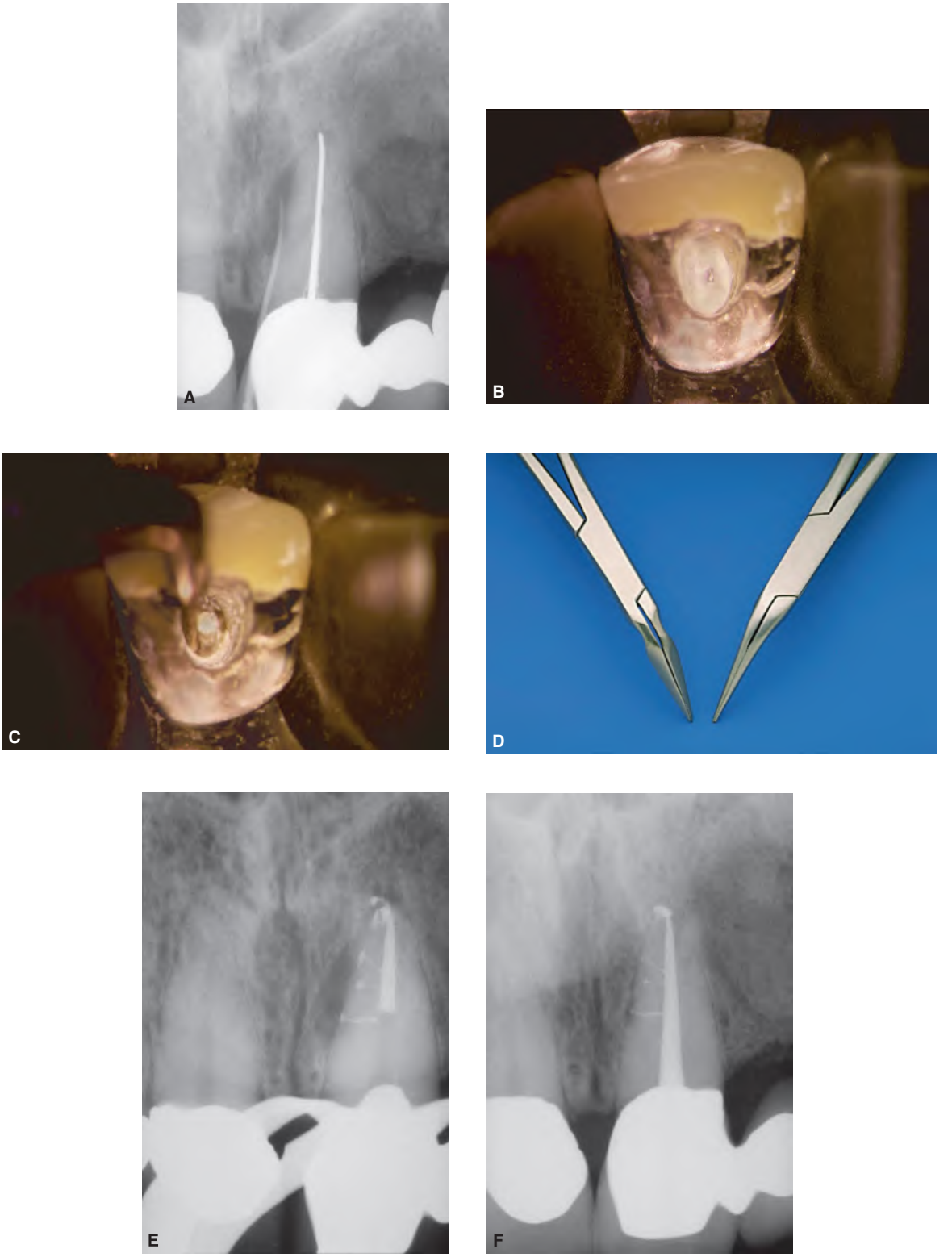


FIGURE 23-49 ***A***. Preoperative radiograph depicting a failing endodontic treatment in the maxillary central incisor serving as a bridge abutment. A gutta-percha point tracing a fistulous tract to a large lateral lesion, and a canal underfilled and slightly overextended. ***B***. Magnification at 15× reveals lingual access and restorative build-up around the coronal-most aspect of the exposed silver point. ***C***. Magnification at 15× demonstrates the ProUltra ENDO-3 ultrasonic instrument removing material laterally to the silver point. ***D***. The left image shows the Stieglitz pliers utilized for grasping intracanal obstructions. The right image shows the "modified" Stieglitz pliers whose working end has been machined down to enhance access. ***E***. A working radiograph, taken during the downpack, reveals apical and lateral corkage. Note the thermosoftened and compacted gutta-percha is apical to the most coronally positioned lateral canal. ***F***. Five-year follow-up radiograph showing the three-dimensional endodontic treatment promoted healing.

ultrasonic instruments should be selected (ProUltra ENDO-3, 4, and 5, Dentsply Maillefer; Ballaigues, Switzerland). These ultrasonic instruments have parallel walls and provide progressively longer lengths and smaller diameters. The appropriate instrument is selected based on its anticipated depth of use and the available canal diameter. The ultrasonic instruments are used to trephine circumferentially around the obstruction, break up cement, and safely expose as much of the silver point as possible. Caution should be exercised so that ultrasonic instruments are *not used directly* on silver points because elemental silver is soft and rapidly erodes during mechanical manipulation. Once the surrounding material is removed, ultrasonic energy may then be transmitted directly onto grasping pliers to synergistically enhance the retrieval efforts. This form of *indirect* ultrasonics advantageously transfers energy along the silver point, breaks up material deep within the canal, and enhances removal efforts.

Files, Solvents, and Chelators

If grasping techniques and/or indirect ultrasonics are unsuccessful, then the clinician should immediately abort this approach and appreciate that silver points are perfectly round and root canal systems are typically irregular in their cross-sectional shapes. This discrepancy between the round silver wire and an irregularly shaped canal provides the clinician with an opportunity to use solvents and a size 10 or 15 stainless steel file. In a solvent-filled chamber, files are used lateral to the silver point to break up cements, and to undermine and loosen the silver point for removal. In underprepared canals, chelators are at times better than solvents, as they allow the instrument to "slip and slide" and work laterally to the silver point. At this stage, a size 15 K file is inserted into an ultrasonic handpiece and placed alongside the silver cone. Once accomplished, this file is activated and used in a short amplitude, in-and-out movement with copious water irrigation to float out the cone (Figure 23-50).[56] If sufficient space exists or can be created between the silver point and the canal wall, a size 20, 25, or 30 hedstrom file can be inserted into this space. The "Hedstrom displacement technique" powerfully promotes the removal process, as the instrument's positive rake angle bites, engages, and establishes a strong purchase on any metallurgically soft silver point (Figure 23-51).

Microtube Removal Options

There are several microtube removal methods, both old and new, that are designed to mechanically engage an intracanal obstruction, like a silver point. However, it must be understood and fully appreciated that many, if not most, of these microtube removal methods frequently require the excessive removal of dentin and oftentimes prove ineffective. For clinicians, the critical distinction when considering microtube removal methods is not the inside diameter of a device, but importantly, its outside diameter, that dictates how deep it can be safely introduced into a canal. The following represents the various microtube removal methods and techniques:

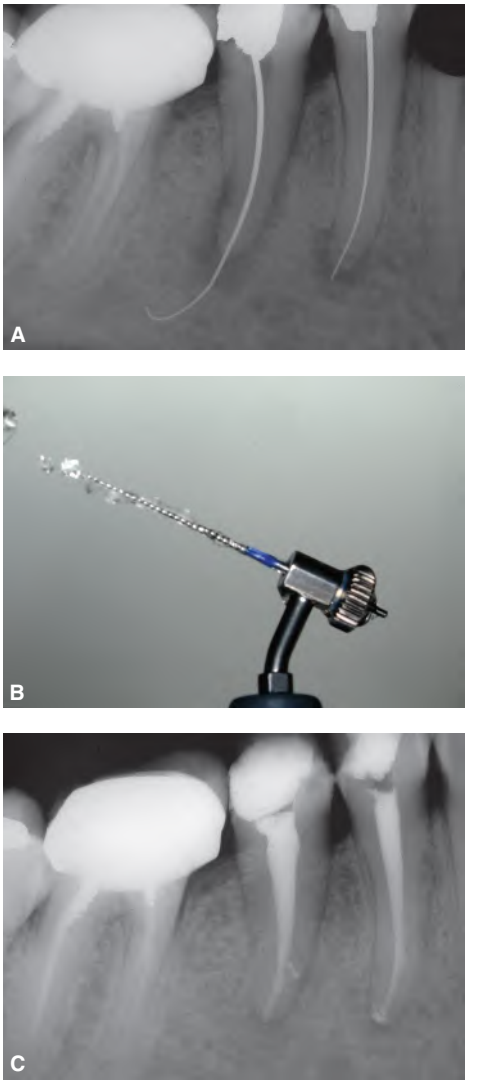


FIGURE 23-50 ***A.*** Preoperative radiograph depicting failing endodontic treatments in mandibular premolars obturated with silver cones. ***B.*** Ultrasonic files or Stainless K files can be mounted on the ENAC handpiece. ***C.*** One-year recall radiograph shows complete healing. Desappointingly, the teeth had not been restored.

- Lasso and Anchor: In this removal method, an appropriately-sized microtube is selected, a wire passed through the tube, then looped at one end and passed back through the tube. This loop can potentially lasso a coronally exposed obstruction and, when successful, form a purchase by pushing the tube apically, while simultaneously pulling the wire ends coronally.[119] Although reported in the literature, this removal method has been essentially replaced with more practical techniques.
- Tube and Glue: The Cancellier Extractor Kit (SybronEndo; Orange, California, USA) contains four different sized microtubes with outside diameters of approximately 0.50,

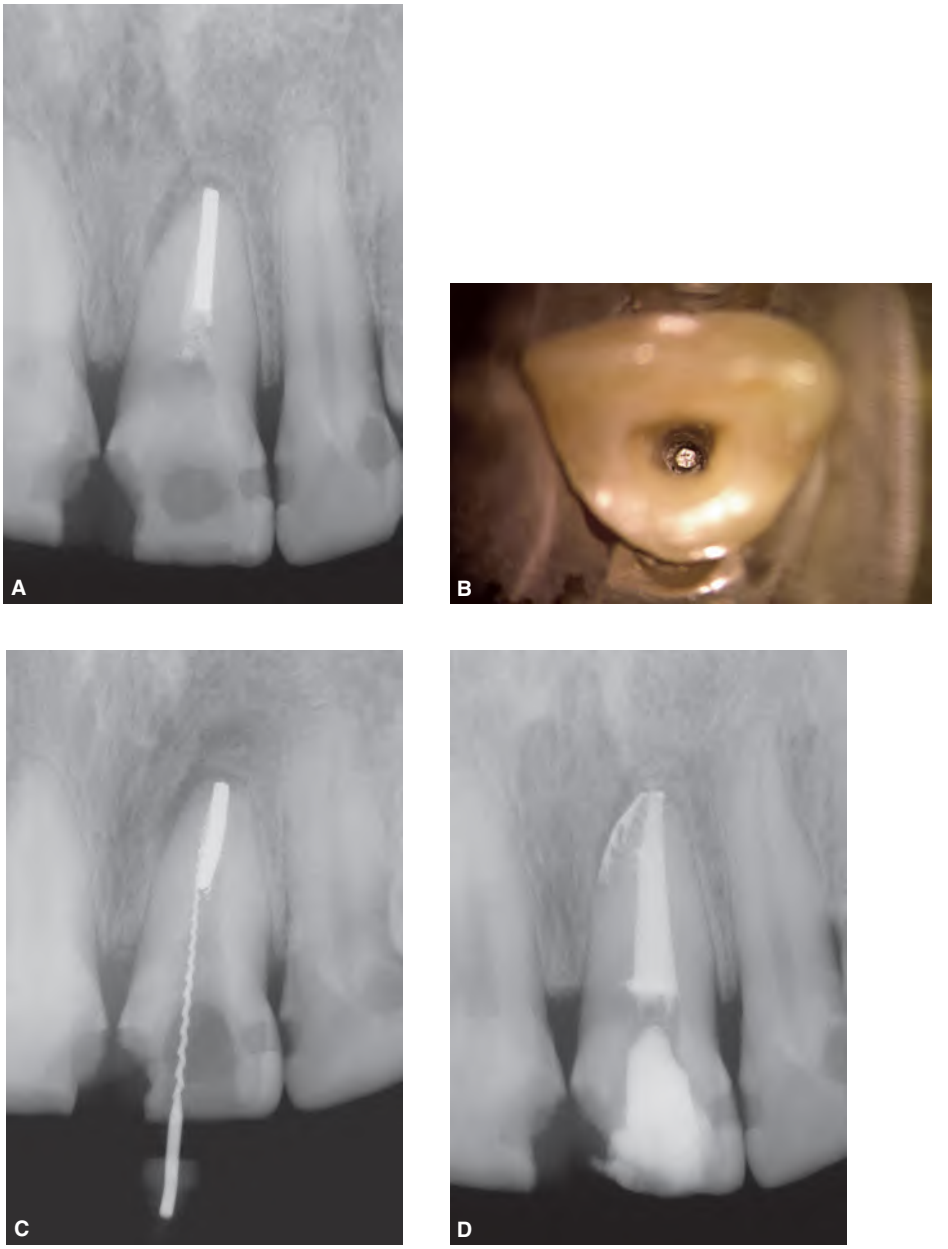


FIGURE 23-51 ***A.*** Preoperative radiograph of failing endodontic treatment in left central incisor. Note the sectioned silver point and asymmetrical lesion apically. ***B.*** Photograph showing the deeply positioned and exposed silver point. ***C.*** Working length radiograph showing a 35 Hedstrom file bypassing the silver point to length. ***D.*** A Postoperative radiograph showing obturation control, apical corkage and filling material in many lateral canals. (Courtesy of Dr. Michael J. Scianamblo, San Rafael, California, U.S.A.)

0.60, 0.70, and 0.80 mm. An abrasively-coated ultrasonic instrument is typically used to trephine around and ideally expose the coronal 3 mm of an obstruction. A microtube is prefit to ensure its internal diameter can just fit over the coronally exposed obstruction. The prefit microtube may now be bonded onto the obstruction with a self-curing composite core build-up, such as Core Paste[3,8] (DenMat; Lompoc, California, USA). The Cancellier microtubes are safely scaled for progressively deeper placement into canals of posterior roots. This removal method is quite effective for retrieving a silver point, a non-fluted broken instrument, or when there is difficulty retrieving a separated file that is already loose. Caution should be exercised to not use too much composite, which could inadvertently block a canal.

- Tap and Thread: The Post Removal System (PRS) kit (SybronEndo; Orange, California, USA) contains five microtubular taps. The smallest PRS tap can be used to internally form threads and mechanically engage the most coronal aspect of any obstruction whose diameter is 0.60 mm or greater.[3] These microtubular taps contain a reverse thread and engage an obstruction by turning in a CCW motion. Because intracanal space is often restrictive, this system is generally used to engage obstructions that extend into the pulp chamber (Figure 23-29).

- Masserann: The Masserann kit (Micromega; Besançon, France) represents a time-honored method to purchase and remove a broken instrument.[120] Although this device has been around for over 40 years, it is superbly made and can form a strong purchase. However, its smallest tubular extractors have outside diameters of about 1.20 mm and 1.50 mm, which limit their safe use to generally larger canals in anterior teeth.
- Spinal Tap Needle: A spinal tap needle *(Ranfac; Avon, Massachusetts*, USA*)*, in conjunction with its metal insert plunger or a Hedstrom file, is another technique advocated to remove broken instruments.[3] With limitations, this method of removal involves sizing the correct microtube so it can be placed over an ultrasonically exposed obstruction. Microtube sizes that are clinically relevant are 19, 21, and 23 gauge needles, corresponding to outside tube diameters of approximately 1.00, 0.80, and 0.60 mm, respectively. Because of their unique ability to engage, smaller sized hedstroem files may be selected and inserted into the coronal-most aspect of the microtube.[3,77] The hedstroem is passed down the length of the tube until it wedges tightly between the obstruction and the internal lumen of the microtube (Figure 23-52). However, because ISO files taper 0.32 mm over 16 mm of cutting blades, the taper of the file oftentimes restricts its placement through a smaller sized parallel microtube. In this instance, the spinal tap needle's metal insert plunger must be used to potentially form a purchase on the obstruction. This method is quite effective when removing obstructions from larger canals.
- Endo Extractor / Meisinger Meitrac: The Endo Extractor System (Roydent; Rochester Hills, Michigan, USA) and the recently released Meisinger Meitrac Instrument System (Hager & Meisinger GmbH; Neuss, Germany) are able to gain a strong mechanical purchase on a silver point or a broken instrument. The Endo Extractor System is a "Jacob Chuck-like" device featuring three short-sized instruments. After selecting an appropriately-sized extractor, the ring is rotated CCW to open the jaws. The extractor is carefully placed over the head of the silver point and the ring is rotated CW, enabling the jaws to circumferentially grasp it (Figure 23-53) The indirect ultrasonic method may then be used against the handle to encourage the removal procedure.[56] The Meitrac Instrument System has been reported in a trade journal to be able to remove broken files from otherwise inaccessible locations. However, the smallest Meitrac I trephine and extractor have outside diameters of approximately 1.50 mm. This diameter limits the practical use of this instrument to the coronal aspects of larger canals.
- File Removal System: The File Removal System (FRS) *(Dentsply Maillefer; Ballaigues, Switzerland)* provides another mechanical method for the removal of intracanal obstructions such as silver points, carrier-based obturators, or broken file segments (Figure 23-54). The FRS is indicated when ultrasonic efforts prove to be unsuccessful and may be used to remove broken instruments lodged in the straightaway portions of the root or partially around the canal curvature.[3,77] The method of use of this device will be discussed in the next section.

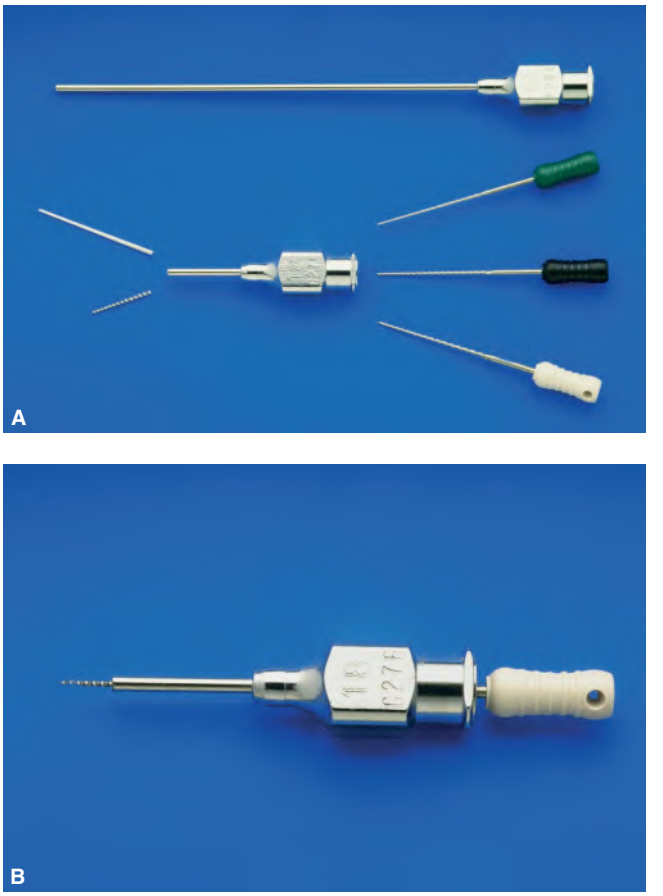


FIGURE 23-52 ***A.*** Photograph showing a full length spinal tap needle (STN). The STN is shortened and placed over a coronally-exposed object. Hedstrom files are passed through the proximal end of the STN to engage an obstruction. ***B.*** The assembled STN. A 45 Hedstrom file is engaging a separated file segment.

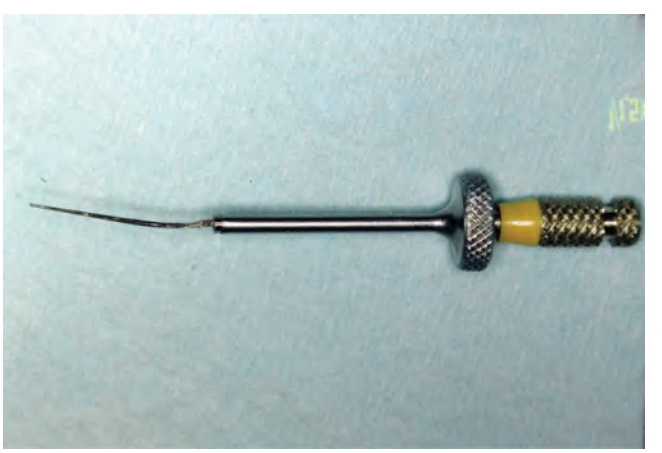


FIGURE 23-53 The Roydent Endo Extractor system.

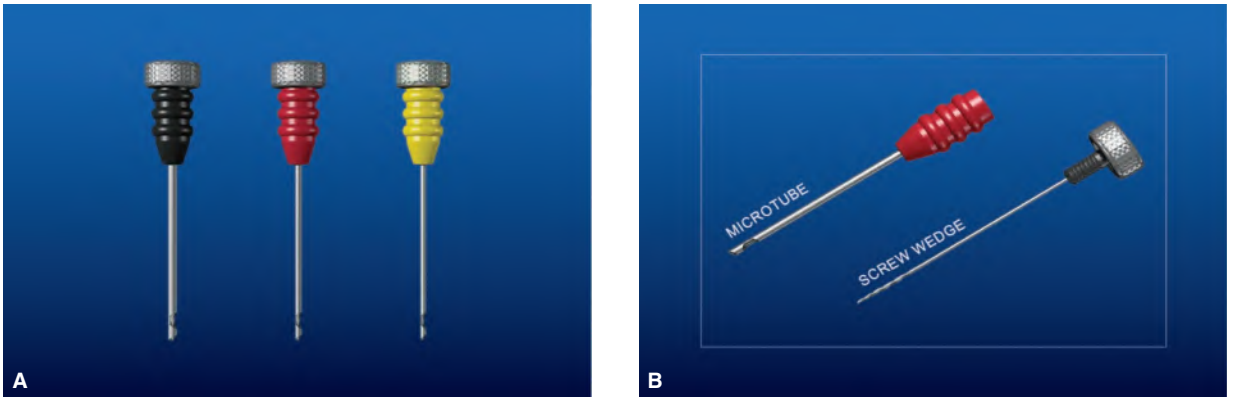


FIGURE 23-54 ***A.*** The File Removal System (FRS) is a set of devices utilized for removing broken instruments or other intracanal obstructions. ***B.*** Each iRS instrument is comprised of a microtube and an internal screw wedge designed to mechanically engage and remove intracanal obstructions.

BROKEN INSTRUMENT REMOVAL

Every clinician who has performed endodontics has experienced a variety of emotions ranging from the thrill-of-the-fill to an upset like the procedural accident of breaking an instrument. During root canal preparation procedures, the potential for instrument breakage is always present. When instrument breakage occurs, it immediately provokes frustration, despair, and anxiety.[121] In fact, the broken instrument dilemma has caused such emotional distress that this event is frequently referred to as a "separated" or "disarticulated" file.

Many clinicians associate a "broken instrument" with a separated file, but the term could also apply to a sectioned silver point, a segment of a lentulo, a gates glidden drill, a portion of a carrier-based obturator, or any other device obstructing the canal.[122,123] With the advent of rotary NiTi files, there has been an unfortunate increase in the occurrence of broken instruments and the factors contributing to breakage have been identified.[124,125] The prognosis of leaving versus removing broken instruments from the canal have been discussed in the literature.[83,126,127] Over the years a variety of approaches for managing broken instruments have been presented.[128–130]

Today, separated instruments can usually be removed due to technological advancements in vision, ultrasonic instrumentation, and microtube delivery methods.[77,131] Specifically, the increasing integration of the dental operating microscope into clinical practice is allowing clinicians to visualize the coronal-most aspect of most broken instruments.[132] The microscope fulfills the age-old adage, "If you can see it, you can probably do it." In combination, the microscope and ultrasonic instrumentation have driven *microsonic* techniques, that have dramatically improved the potential and safety of removing broken instruments.[3,133,134]

Factors Influencing Broken Instrument Removal

The factors influencing broken instrument removal should be identified and fully appreciated.[135,136] The ability to nonsurgically access and remove a broken instrument will be influenced by the diameter, length, and position of the obstruction within a canal. Additionally, the potential to safely remove a broken instrument is guided by anatomy, including the diameter, length, and curvature of the canal. Importantly, the potential for safely removing a broken instrument is limited by root morphology, including the circumferential dimensions and thickness of dentin and the depth of an external concavity.[137,138] In general, if one-third of the overall length of an obstruction can be exposed, it can usually be removed. Instruments that lie in the straightaway portions of the canal can typically be removed. Separated instruments that lie partially around canal curvatures, although more difficult, can oftentimes be removed if straightline access can be established to their most coronal extents (Figure 23-55).[3,134] If the broken instrument segment is apical to the curvature of the canal and safe access cannot be accomplished, then removal is usually not possible and, in the presence of signs or symptoms, surgery or an extraction will, at times, be required. On occasion, an attempt to bypass the instrument can be successful (Figure 23-56).

The type of material comprising an obstruction is another important factor to be considered. As an example, stainless steel files tend to be easier to remove, as in general, they do not further fracture during the removal process. NiTi broken instruments may break again, albeit deeper within the canal, during ultrasonic efforts, presumably due to heat buildup.[8] Perhaps the most important factors central to successful instrument removal are knowledge, training, and competency in selecting the best presently developed and proven technologies and techniques. Importantly, no one removal method will always produce the desired result. As such, successful removal oftentimes requires patience, perseverance, and creativity. However, no removal method should be attempted until access has been made to the head of an intracanal obstruction.

Coronal and Radicular Access

Prior to commencing with efforts to remove a broken instrument, the clinician should thoughtfully observe different horizontally-angulated pre-operative radiographs. Coronal

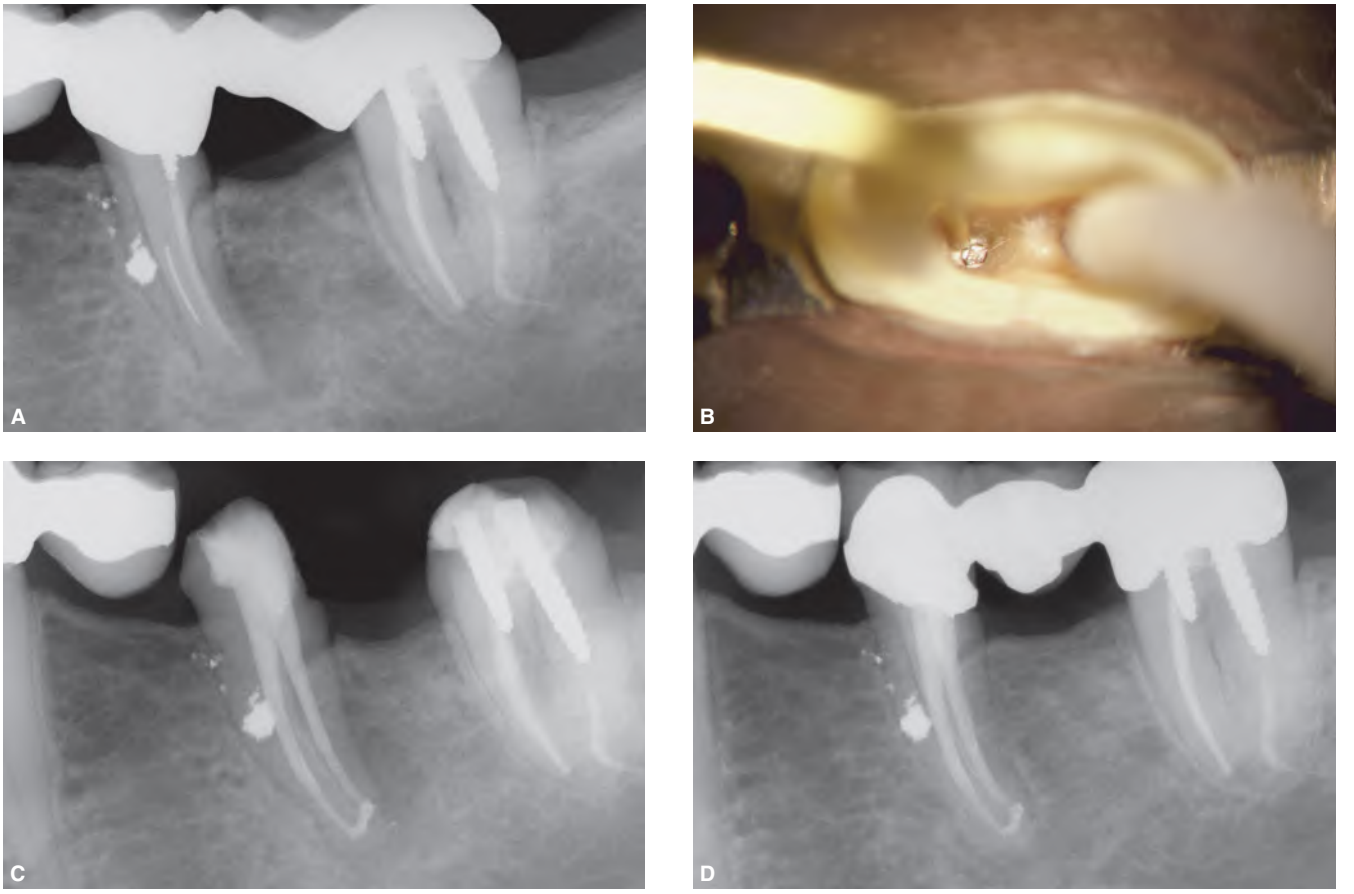


FIGURE 23-55 ***A.*** A failing endodontic treatment in a mandibular first molar. Note, a short screw post, a separated instrument and amalgam debris from the hemisection procedures. ***B.*** Photograph showing the splint removed, the post taken out, and an ultrasonic instrument trephining around the broken separated file. ***C.*** Postreatment radiograph reveals successful retreatment. Note the third mesial system between the MB and ML canals. ***D.*** Eight-year recall radiograph showing three-dimensional retreatment, a new bridge and excellent periradicular healing.

access is the first step in the removal of broken instruments. Highspeed, friction grip, surgical length burs are selected to create straightline access to all canal orifices. Special attention should be directed towards flaring the axial wall that approximates the canal holding the broken instrument in efforts to subsequently improve microsonic techniques below the orifice.

Radicular access is the second step required in the successful removal of a broken instrument. However, before commencing with radicular access, it is useful to review a few concepts that can potentially guide our clinical actions. With exceptions, the vast majority of teeth range from 19 to 25 mm in overall length. Most clinical crowns are about 10 mm and most roots range from 9 to 15 mm in length. If the root is divided into coronal, middle and apical one-thirds, then each third is between 3 and 5 mm in length. The question frequently asked is, how big can a canal be optimally flared without creating an iatrogenic problem? The answer is to review the dimensions of a typical preparation in a longer, narrower, and more curved root form. In this situation, if a 20 file is snug at length and each successively larger instrument uniformly moves away from the foramen in 1/2 mm increments, then the apical one-third of the canal would taper 10%. In this specific example, the diameter of the canal 4 mm coronal to the foramen would be equivalent to at least a 60 file or 0.60 mm. This analogy is useful and can serve to safely guide how big to prepare the apical one-third and the coronal two-thirds of a canal when there is a broken instrument.[139]

Clinical experience suggests the majority of broken files separate towards their terminal extents at between D_3, D_4, or D_5. Files most frequently break in the apical 3 to 5 mm because this is the region where a canal usually has its smallest diameter and exhibits its greatest degree of curvature or propensity to divide. Even if a file breaks at the working length, the position of the head of the instrument typically lies at about the junction of the middle and apical one-thirds. Fortuitously, straightline radicular access can generally be created through the coronal two-thirds of a canal to the head of a broken instrument.[3,140]

A number of different techniques may be employed to flare the canal coronal to an intracanal obstruction. However, experience suggests a predictable way to create safe radicular access is to initially use hand files, small to large, coronal to the obstruction. Hand files create sufficient space to safely accommodate NiTi rotary files, or preferably Gates Glidden (GG) drills (Dentsply Maillefer; Ballaigues, Switzerland). GG sizes 1 to 4 are most typically employed in furcated teeth and have maximum diameters of 0.50, 0.70, 0.90, and 1.10 mm, respectively. GGs are used to create radicular access and a uniform tapering funnel to the obstruction. GGs are more safely rotated at speeds

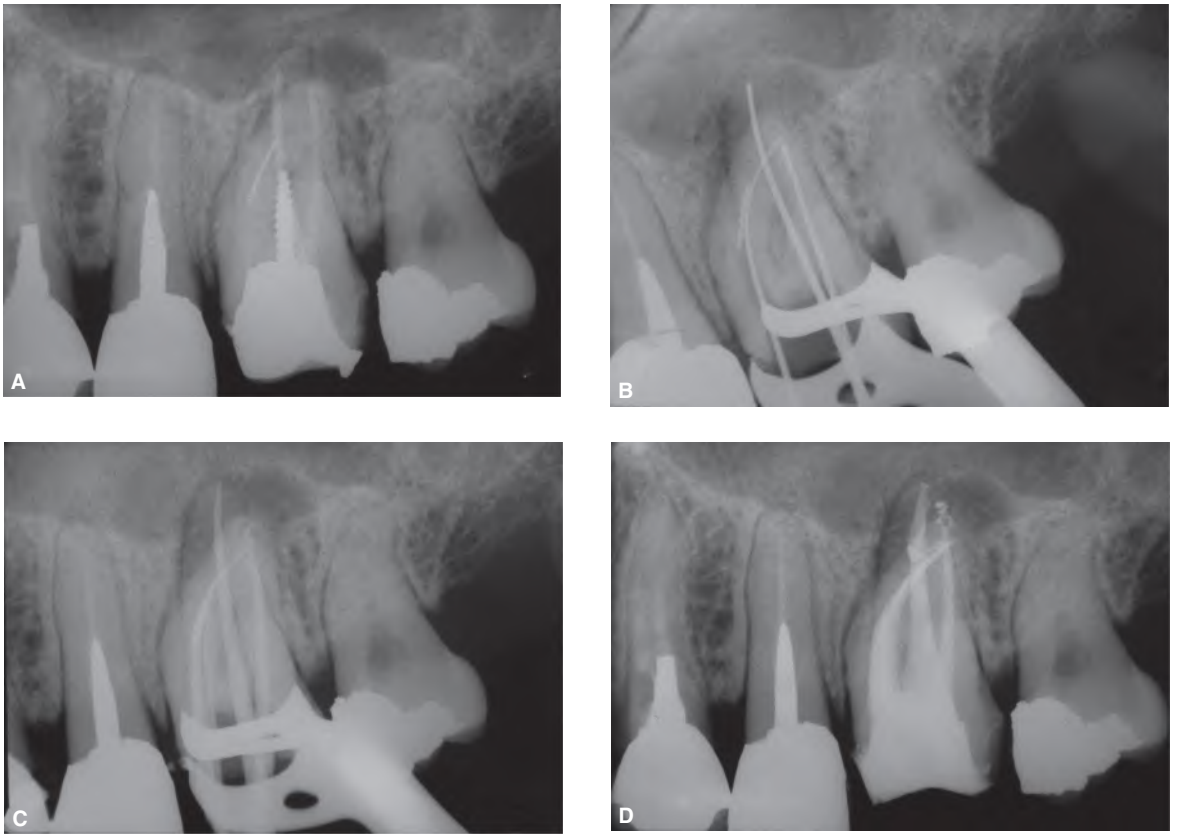


FIGURE 23-56 ***A.*** Preoperative radiograph of the first molar showing a separated instrument located beyond the curvature of the mesial root. ***B.*** After radicular access to the head of the fragment, the instrument could be bypassed with small hand files. ***C.*** During cleaning and shaping, the fragment was floated out with irrigation. Cone fit radiograph showing instrument removed. ***D.*** Postoperative radiogaph showing canal obturation.

of about 750 rpm and, importantly, are used with a *brushing motion* to create a tapered shape and maximize visibility.[3] Increasingly larger GGs are uniformly stepped out of the canal to create a smooth flowing funnel, that is largest at the orifice and narrowest at the obstruction. GG drills should be limited to the straightaway portions of the canal. Generally, a GG-1 or GG-2 can be carried to the depth of the head of a separated instrument. The GGs are used cautiously in approximation to the obstruction, with attention to brush-cutting out of the canal and away from furcal danger. Deliberately relocating the coronal one-third of a canal away from the furcation maximizes remaining dentin, produces a more centered preparation, and improves straightline radicular access.[3,140,141] The GG-3 is carried short of the level where the GG-2 was used and, in furcated teeth, the GG-4 is confined to a depth of no more than one bud length below the orifice. Importantly, radicular access should be performed so that the canal is pre-enlarged and ideally shaped no bigger than it would otherwise be prepared if there was no broken instrument obstructing the canal.

Creating a Staging Platform

When the canal has been optimally shaped, then microsonic techniques are usually the first option selected to remove a broken file segment. At times, when an ultrasonic instrument is introduced into a pre-enlarged canal, its activated tip does not have enough space, lateral to the broken file segment, to initiate trephining procedures. As such, if more lateral space is required, then the bud of a GG can be modified and used to create a circumferential staging platform.[3,8] The staging platform is made by selecting a GG drill with a maximum cross-sectional diameter slightly larger than the visualized instrument. The bud of the GG drill is altered by cutting it perpendicular to its long axis, at its maximum cross-sectional diameter (Figure 23-57). This modified GG is carried into the pre-enlarged canal, rotated at a reduced speed of approximately 300 rpm, and directed apically until it lightly contacts the coronal-most aspect of the obstruction. This clinical step creates a small staging platform which facilitates the introduction of an ultrasonic instrument. If properly performed, straightline coronal and radicular access, in conjunction with magnification and lighting, should enable the clinician to fully visualize the coronal-most aspect of a broken instrument. To facilitate excellent vision to the intraradicular obstruction, the canal should be vigorously flushed and thoroughly dried prior to beginning ultrasonic procedures.

Techniques for Removing Broken Instruments

A number of devices, technologies, and techniques have been reported to remove an intracanal obstruction, such as a broken instrument.[134,136] However, many of the removal techniques previously described in the literature did not have the benefit of the operating microscope. Today, virtually all broken

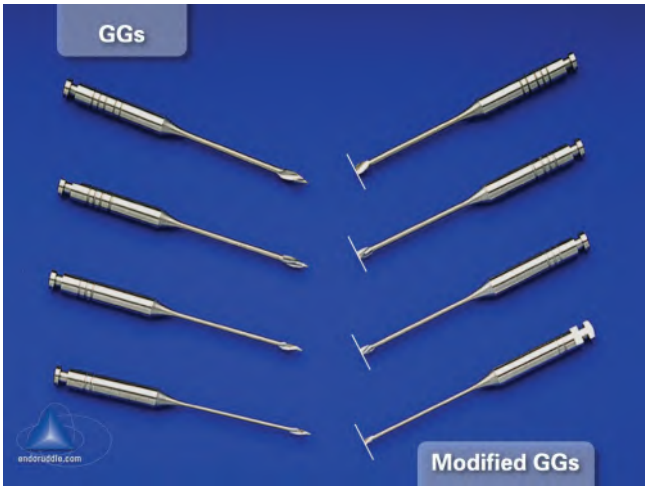


FIGURE 23-57 Photograph showing selected GG drills and their subsequent modification.

instruments can be eliminated if straightline access can be safely made to the coronal-most extent of a broken instrument.[3,134,140] The most important, predictable, and safe removal schemes utilize the microscope in conjunction with optimally designed ultrasonic instruments and/or a microtube method.[3,140]

Ultrasonic Techniques

Prior to performing any radicular removal techniques, it is wise to place cotton pellets over other exposed orifices, if present, to prevent the nuisance re-entry of the fragment into another canal. The first option to remove a broken instrument is to utilize piezoelectric ultrasonic technology and specific ultrasonic instruments (Figure 23-24). An ultrasonic generator should provide a broad range of power, precise adjustment within the lower settings, and electrical feedback to regulate amplitude and safe tip movement. Ideally, ultrasonic instruments should have a contra-angled design to provide access into all regions of the mouth, parallel-sided walls to create a line of sight between the instrument and the tapered canal, and non-aggressive coatings, such as zirconium nitride, to precisely sand away dentin during trephining procedures. Further, an appropriately-sized ultrasonic instrument is selected, such that its length will reach the broken obstruction and its diameter will passively fit and afford a favorable line-of-sight into the previously shaped canal.

The tip of this ultrasonic instrument is placed in intimate contact against the obstruction and typically activated within the lower power settings (Figure 23-58A). *The clinician should always work at the lowest power setting that will efficiently and safely accomplish the clinical task*. ALL ultrasonic work below the orifice is conducted DRY so the clinician has constant visualization of the energized tip against the broken instrument. To maintain vision, the dental assistant utilizes the Stropko three-way adapter with an appropriate luer-lock tip to collimate and direct a continuous stream of air and blow out dentinal dust. Microsonic techniques, as advocated for removing broken instruments, do not generally generate sufficient heat to become harmful to the attachment apparatus.

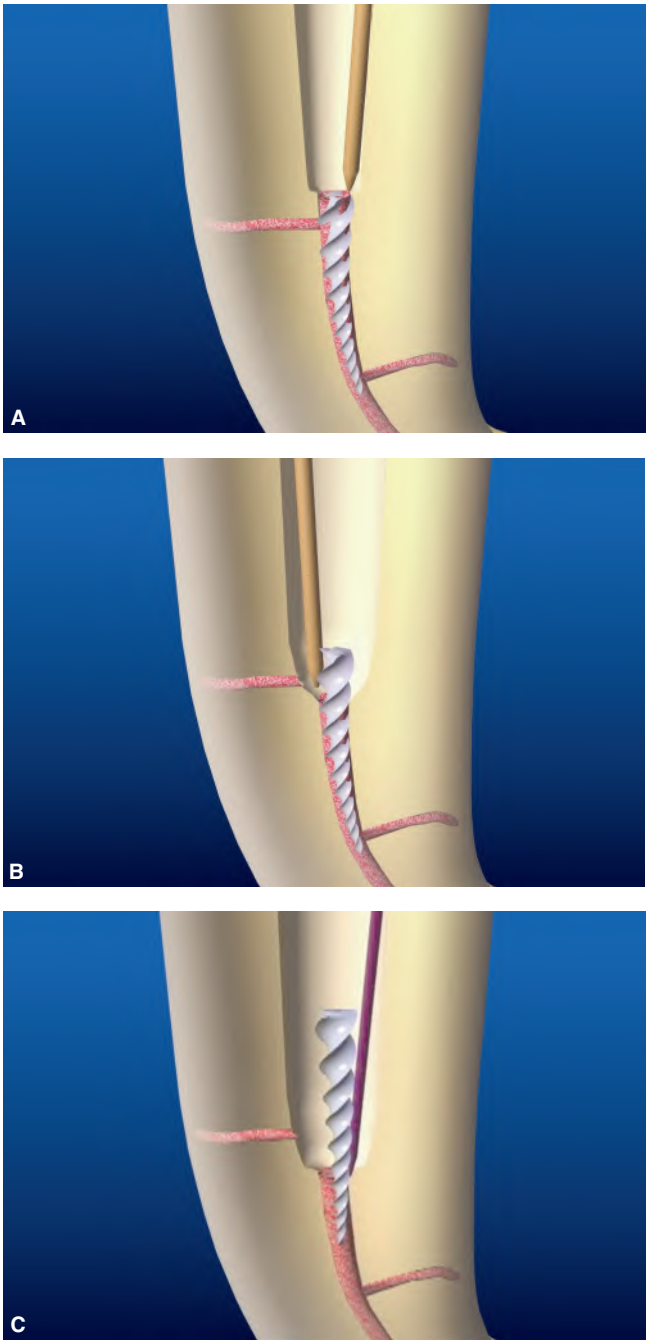


FIGURE 23-58 ***A.*** Illustration demonstrating the importance of coronal and radicular access, the staging platform and the ultrasonic instrument just laterally to the separated broken file. ***B.*** The ultrasonic instrument maintains contact with the separated file, precisely sands away dentin, and progressively exposes the coronal aspect of the file. ***C.*** A longer length and smaller diameter titanium instrument is selected to conserve dentin and successfully displace the separated instrument.

The selected ultrasonic instrument is moved lightly, in a CCW direction, around the obstruction, except when removing a file that has a left-handed thread, in which case the direction would be CW. This ultrasonic action trephines, precisely sands away dentin, and exposes the coronal few

millimeters of the obstruction (Figure 23-58B). Typically, during ultrasonic use, the obstruction begins to loosen, unwind, and then spin. Gently wedging the energized tip between the tapered file and canal wall oftentimes causes the broken instrument to abruptly jump out of the canal. In the instance where a broken file lies deep and ultrasonic procedures are restricted by root bulk and form, then select a longer length and smaller diameter, abrasively-coated ultrasonic instrument to promote safe retrieval efforts. In longer roots, or when space is even more restrictive, then an appropriately-sized titanium instrument may be chosen. Titanium instruments provide a smooth cutting action, that promotes safety when trephining deeper within a canal (Figure 23-58C). At times, weighing risk versus benefit, ultrasonic trephining procedures may have to be aborted. In these instances, the sharp cutting edges of a hypodermic needle may be safely used manually to further expose the head of a broken file.[142] Exposing 2 to 3 mm of the coronal-most aspect of an obstruction, or about one-third of its overall length, will generally produce the desired result. The clinical steps for broken instrument removal utilizing microsonics are shown in Figure 23-59.

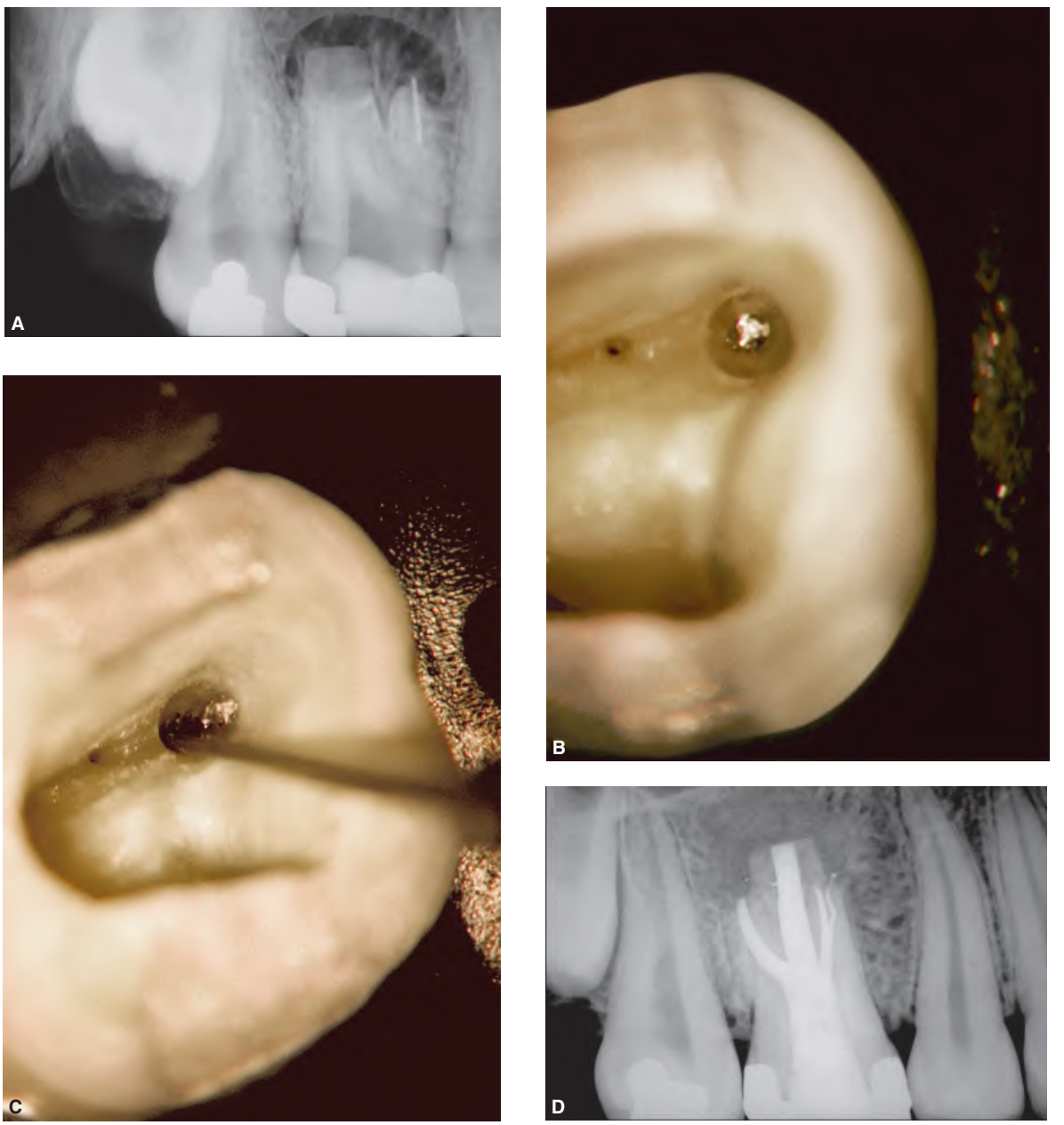


FIGURE 23-59 ***A.*** An off-angled radiograph of a maxillary first molar reveals a separated instrument, a possible MB-2 system and resected roots from a previous surgical procedure. ***B.*** Photograph showing access, an MB-2 orifice, the separated instrument, and the circumferential staging platform to facilitate ultrasonics. ***C.*** Documentation reveals an ultrasonic instrument has loosened and coronally displaced the separated instrument. ***D.*** A recall radiograph showing evidence of initial healing. Barriers were utilized in the DB and P canals to facilitate control and encourage three-dimensional filling.

On occasion, the clinician may create excellent coronal and radicular access, identify and expose the separated instrument, perform ultrasonic trephining procedures, and still be unable to loosen and jettison the instrument out of the canal. Further, it may be unsafe to continue trephining around a broken instrument due to lack of vision or anatomical restrictions. In this instance, small hand files may be used with an aqueous or viscous chelator, to partially or completely bypass, and hopefully remove a broken instrument through irrigation. Even when this tedious removal method is unsuccessful, oftentimes a little space can be created along a portion of the overall length of a broken instrument. To maximize efficiency and success, the handle from a stainless steel hand file can be intentionally removed and the shaft of the instrument inserted into a device called the File Adapter (Figure 23-60). The File Adapter threads onto the ultrasonic handpiece and its chuck will retain a 0.02 tapered hand file. Small stainless steel hand files can be precurved, if indicated, inserted into available space, and used at low power in an ultrasonic effort to remove a broken instrument. This technique is, at times, useful when the root is thin or a portion of the file lies apical to a canal curvature.[143]

Another clinical challenge is encountered when trying to remove a broken NiTi file that lies partially around a canal curvature. In these situations, it is axiomatic the head of a broken NiTi file will always lie against the outer wall even after optimal ultrasonic trephining procedures. Even when loose, the angle formed between the coronally flared canal and the head of the broken instrument oftentimes precludes its removal. This situation is best managed using a microtube removal method (Figure 23-61).[3,77,140]

The FRS Option

As previously mentioned in *"Silver Point Removal,"* there are several microtube removal methods that are designed to mechanically engage an intracanal obstruction. The File Removal System (FRS) has provided a breakthrough in the retrieval of broken instruments lodged deep within the root canal space.[3,77] The FRS is composed of variously-sized microtubes and insert wedges that are scaled to fit and work deep within the root canal space (Figure 23-62). The FRS is indicated when ultrasonic efforts prove to be unsuccessful and may be used to remove broken instruments that are lodged in the straightaway portions of the root or partially around the canal curvature. The black instrument has an outside diameter of 1.0 mm and is designed to work in the coronal one-third of larger canals, whereas the red and yellow instruments have outside diameters of 0.80 and 0.60 mm, respectively, and can be placed deeper into more narrow canals. Each complete instrument is comprised of a color-coordinated microtube and screw wedge (Figure 23-62B). Each microtube has a small-sized plastic handle to enhance vision during placement, a side window to improve mechanics, and a 45° beveled end to "scoop up" the coronal end of a broken instrument. Each screw wedge has a knurled metal handle, a left-handed screw mechanism proximally, and a solid cylinder that transitions into 0.02 tapered K-type file blades towards its distal end to facilitate engaging an obstruction.

FIGURE 23-60 Photograph showing that when the handle of an endodontic file is removed, its shaft can be quickly secured into the ultrasonically activated adapter.

As has been emphasized for any removal technique, straightline coronal and radicular access is required to expose and subsequently visualize the coronal-most end of the broken instrument. As previously described, the clinician utilizes ultrasonic instrumentation to circumferentially expose 2 to 3 mm of the separated file. However, ultrasonic instruments can only circumferentially trephine, sand away dentin, and expose the portion of the obstruction that lies in the straightaway portion of the canal. Therefore, the goal is to expose 2 to 3 mm, or about one-third of the total length, of a separated instrument (Figure 23-63A).

An FRS microtube is then selected that can passively slide through the pre-enlarged canal and drop over the exposed broken instrument. As previously mentioned, in a curved canal, the head of a broken NiTi file will always lie against the outer wall. In these instances, the microtube is inserted into the canal with the long part of its beveled end oriented to the outer wall of the canal to scoop up the head of the broken instrument and guide it into the microtube (Figure 23-63B). Once the microtube has been positioned, it is rotated 180° so the head of the file is oriented adjacent to the lateral window. The same color-coded screw wedge is then inserted and slid internally through the microtube's length until it contacts the obstruction. The obstruction is engaged by gently turning the screw wedge handle CCW. A few degrees of rotation will serve to tighten, wedge, and oftentimes, displace the head of the obstruction through the microtube window (Figure 23-63C). If any given color-coded screw wedge is unable to achieve a strong hold on the obstruction, then another color-coded screw wedge may be chosen to improve engagement and successful removal. When engaged, the obstruction can be potentially unwound and removed by rotating the microtube and screw wedge assembly CCW (Figure 23-63). The direction of rotation,

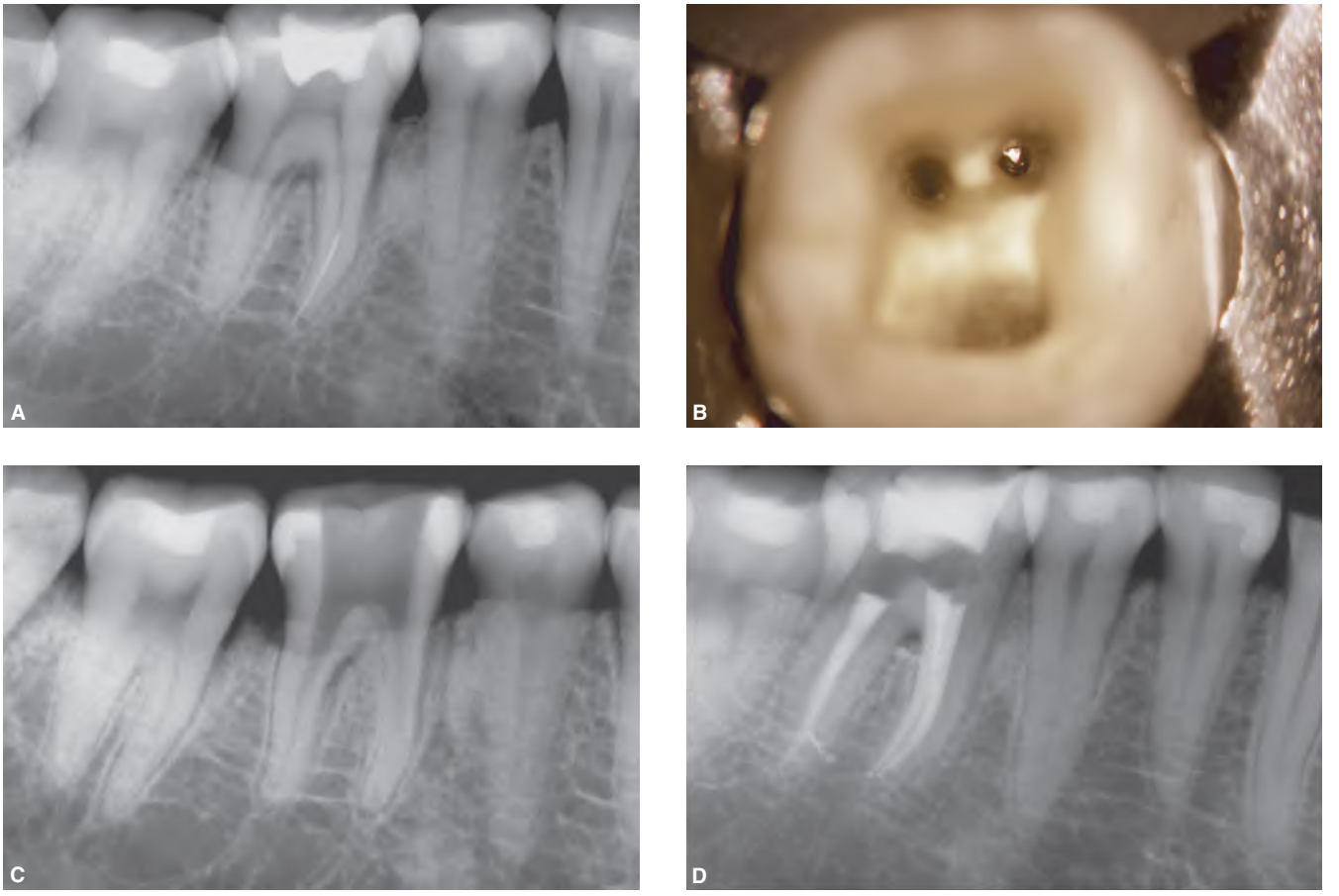


FIGURE 23-61 ***A.*** Preoperative radiograph of a mandibular left first molar with a separated instrument in the apical one-third of the mesial root. ***B.*** After refining access and completing ultrasonic procedures, a photograph reveals the head of the separated NiTi file predictably against the outer wall. ***C.*** A radiograph confirms straightline access and the removal of the separated file. ***D.*** Post-treatment radiograph shows the pack. Note, the furcal canal and that the distal system bifurcates apically.

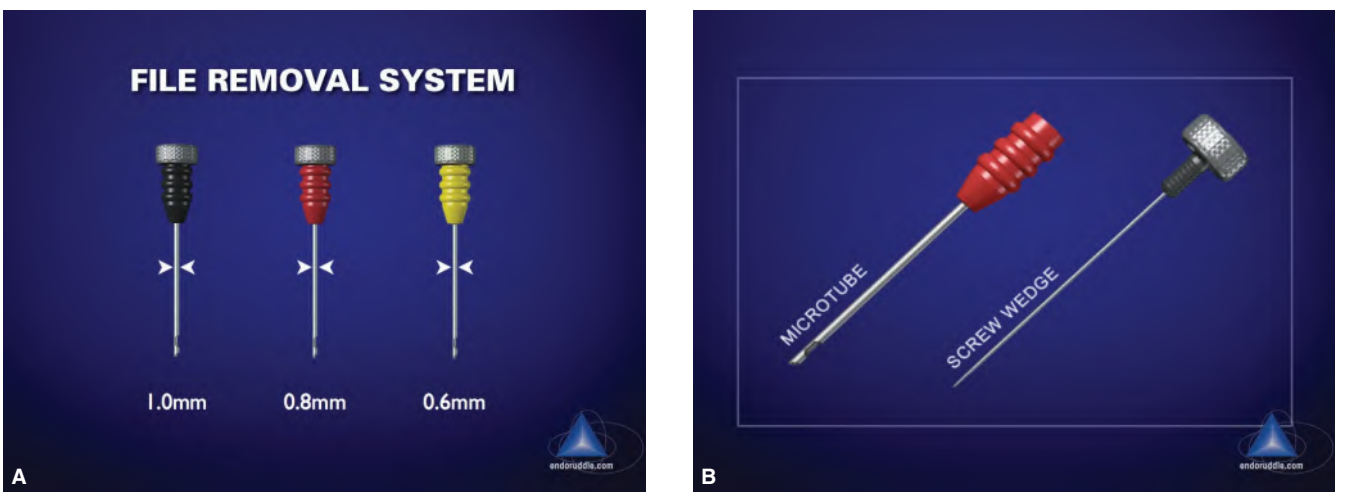


FIGURE 23-62 ***A.*** The Instrument Removal System (iRS) is a set of devices utilized for removing separated instruments or other intracanal obstructions. ***B.*** Each iRS instrument is comprised of a microtube and an internal screw wedge designed to mechanically engage and remove intracanal obstructions.

in the instance of a broken file, is generally CCW, but ultimately should be appropriate to the thread design of the obstruction. If difficulty is encountered when rotating the microtube and screw wedge assembly CCW, then proceed with a limited CW rotation of 3° to 5°, which will promote staying engaged, followed by turning the assembly CCW until snug. This repeated reciprocating handle motion will serve to loosen and facilitate the removal process. Placing

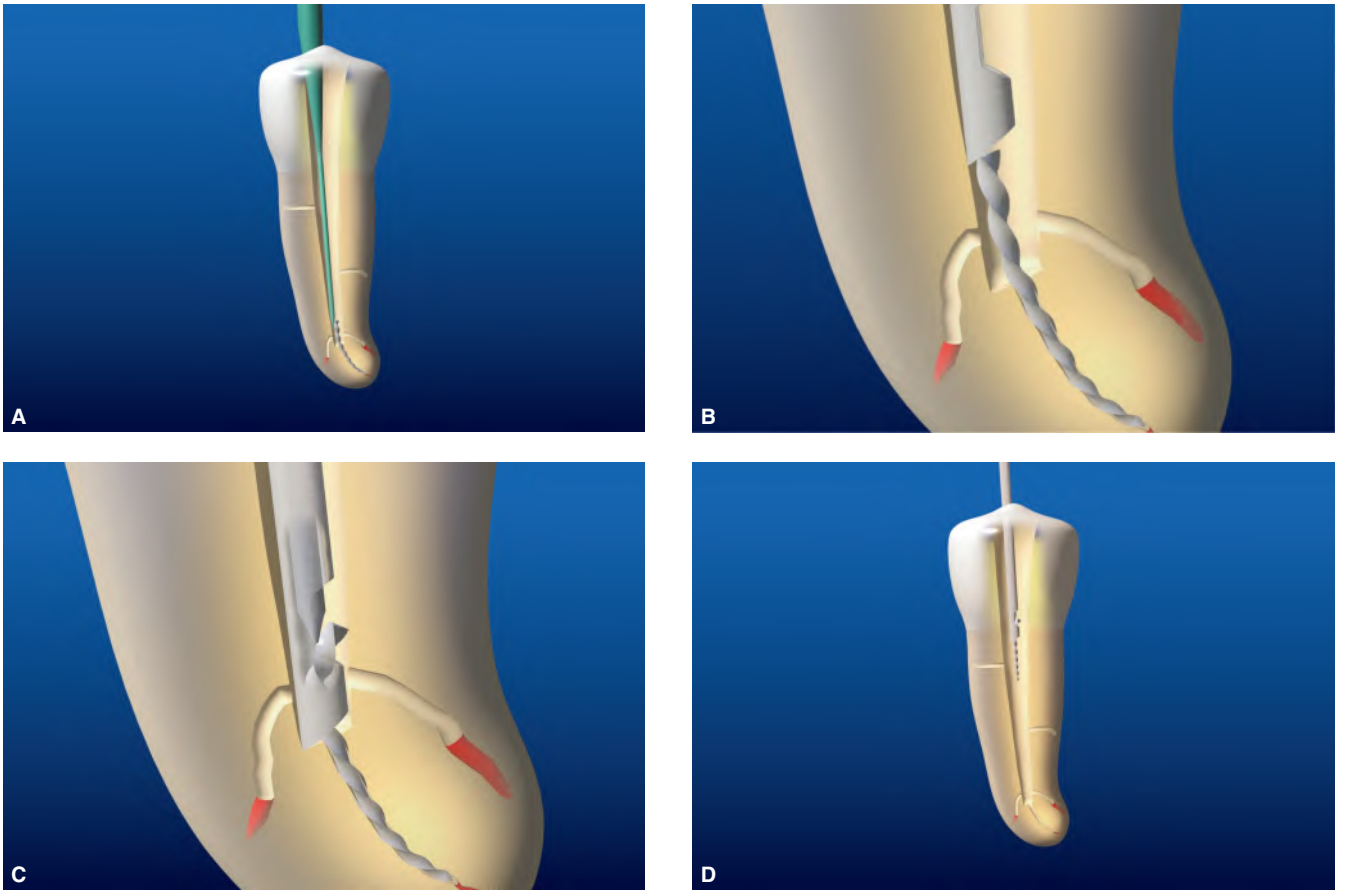


FIGURE 23-63 ***A.*** Illustraion demonstrating a titanium ProUltra ENDO-8 trephining deep around the coronal aspect of the separated file. ***B.*** When ultrasonic procedures prove ineffective, the beveled end of an iRS microtube is designed to "scoop up" the head of the separated file. ***C.*** Introduction of the screw wedge rotated counterclockwise to engage and potentially displace the head of the file out the side window. ***D.*** The iRS can form a strong purchase on, unwind and remove a separated instrument.

an activated ultrasonic instrument on the engaged assembly is another potent adjunct that will oftentimes promote removal success. If a microtube cannot be placed over a broken instrument such that the head of the obstruction lies within the side window, then in these instances, the microtube's beveled end can be easily reduced or eliminated to achieve better mechanics. A clinical case utilizing the FRS option is shown in Figure 23-64.

The Endo Rescue Kit 4601 Option

The Endo Rescue kit (Komet; Rock Hill, South Carolina, USA) has recently launched for the removal of broken instruments.[144] The kit is comprised of five instruments: namely, a cylindrical bur with a noncutting tip, two short Gates Glidden drills (GG-3 and GG-4), a dedicated centering drill, and a trephine. The centering drill has the same external diameter as the GG-3 (0.90 mm) and its concave tip is designed to go over the fragment and keep the preparation centered (Figure 23-65). The trephine has the same external diameter as both the GG-3 and the centering drill (0.90 mm). Specifically, the trephine has an internal lumenal length of 5 mm and a diameter of 0.50 mm. The trephine must be used in a CCW rotation.

After creating straightline access to the broken instrument with the cylindrical bur, the GG-3 is used to reach the head of the fragment. The centering drill is then used at 300 rpm, such that its outer blades bore and cut dentin around the broken file. The correspondingly-sized trephine is used in a CCW rotation, either mechanically at 300 rpm or manually, with the mandrel included in the kit. Trephining serves to cut a 2 mm-length thin trench around the head of the broken fragment. The activity of trephining serves to produce dentin chips that in turn, jam between the inner walls of the trephine and the fragment. When successful, the fragment is pulled inside the trephine. If unsuccessful, the microtube technique may be considered. In this instance, a microtube is selected that has the same outside diameter as the previously used trephine (Ultradent; South Jordan, UT, USA). This microtube is prefit over the fragment, filled with a self-curing composite core material, and then replaced in the canal and over the fragment. Once the composite has set, the microtube is rotated CCW to encourage the removal of the broken fragment (Figure 23-66). A radiograph is taken to confirm instrument removal.

When using the microtube technique, care should be taken to dry the canal with pure alcohol. Further, only the

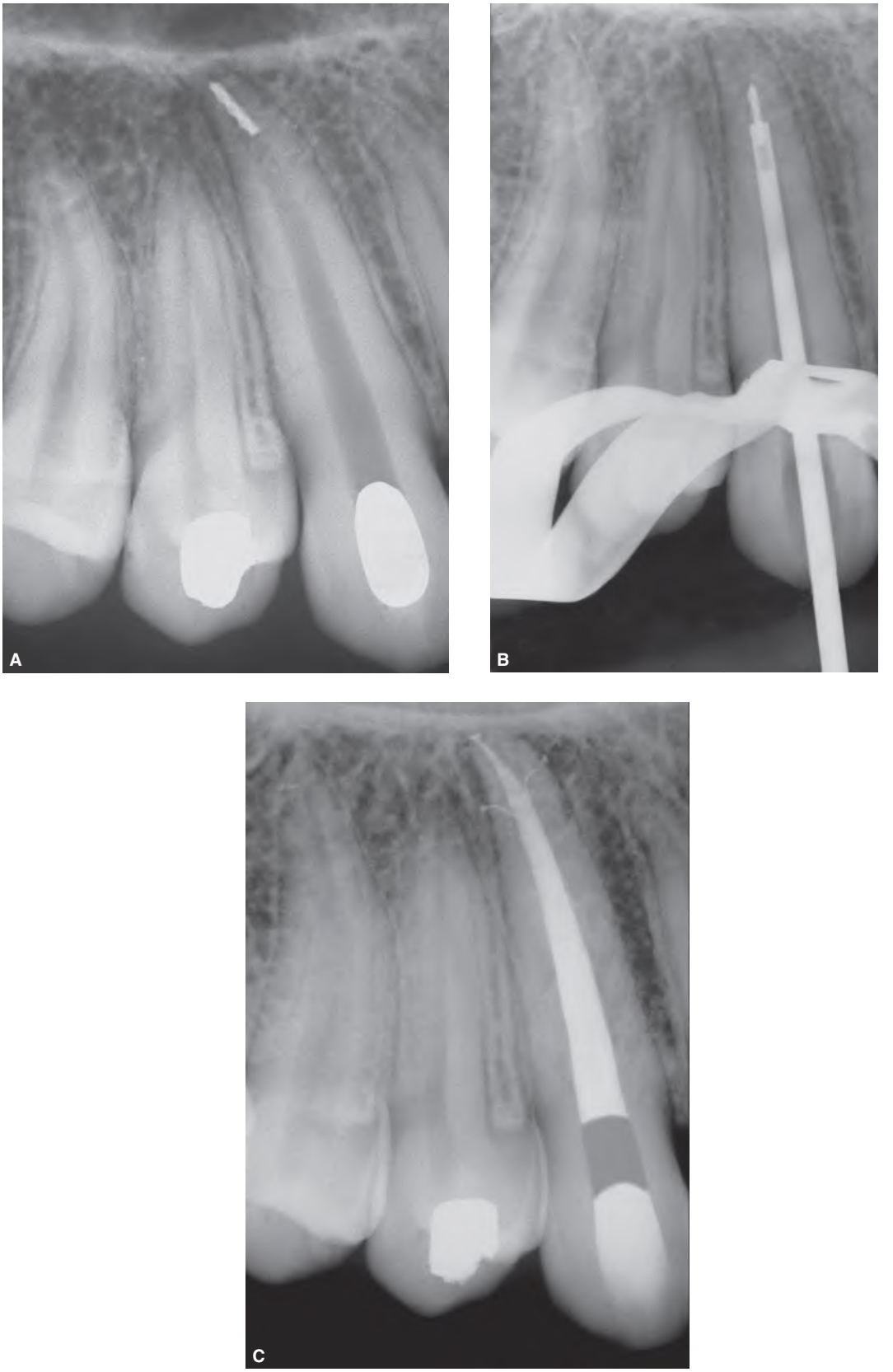


FIGURE 23-64 ***A.*** Preoperative radiograph of a maxillary canine showing a temporized canal with a separate instrument deep in the apical one-third. ***B.*** A working radiograph shows that the 21 gauge iRS has successfully engaged and partially elevated the deeply positioned file segment. ***C.*** Postoperative radiograph showing the retreatment completed with a densely packed canal system that exhibits three apical portals of exit.

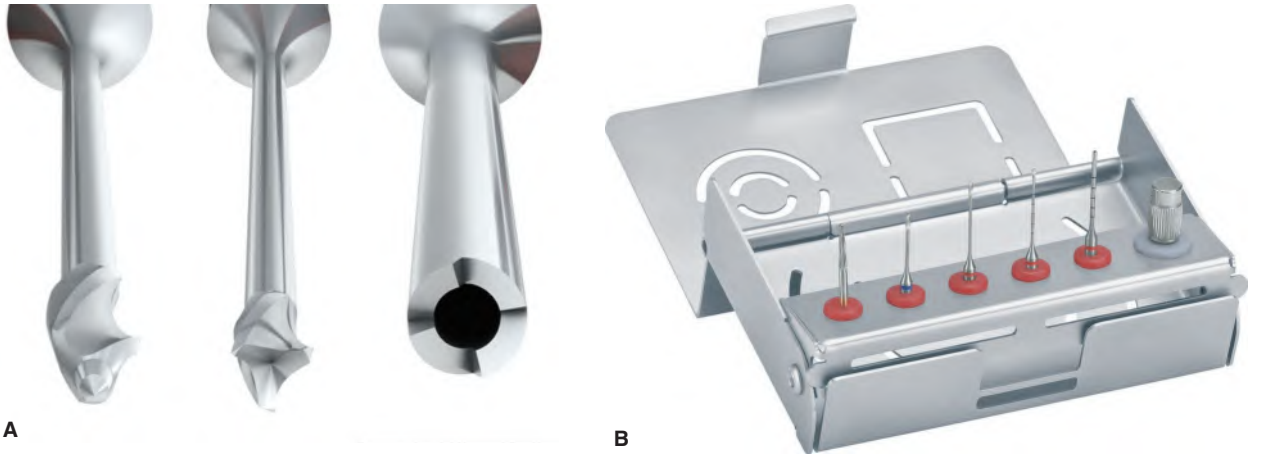


FIGURE 23-65 ***A.*** The main components of the Endo Rescue kit. ***A.*** The three instruments, Gates Glidden drill no.3 (i.e., 0.9 mm), Centering drill and Trephine have the same external diameter. The internal diameter of the trephine is 0.5 mm. Note the concave tip of the centering drill. ***B.*** The complete kit.

internal lumen of the microtube should be filled with composite, without surplus. In thin roots, when a staging platform is not practical, NiTi file fragments may be abraded with thin ultrasonic tips to facilitate utilizing a bypass technique (Figure 23-67).[145]

Broken Instrument Prevention

The best antidote for a broken file is prevention. Adhering to proven concepts and utilizing safe techniques during root canal preparation procedures will virtually eliminate the broken instrument procedural accident.[3,141] Prevention may also be greatly facilitated by thinking of negotiating and shaping instruments as disposable items. Simply discarding all instruments after the completion of each endodontic case will reduce breakage, lost clinical time, and prevent upsets. However, on occasion, an instrument will break and the treating dentist must decide on the best treatment option.[146] Weighing risk versus benefit, certain broken file segments may not be able to be retrieved or bypassed. However, "Failing is not always failure," so, in these instances, the canal should be cleaned up to the fragment and sealed. The case should be followed over time (Figure 23-68), and in the presence of clinical symptoms and/or radiographic pathology, surgery,[56] or extraction may be the best treatment option.

BLOCKS, LEDGES, & APICAL TRANSPORTATIONS

It is important to understand and appreciate the biological and mechanical objectives for shaping canals and cleaning root canal systems.[139,147] Failure to respect these objectives increases frustration and predisposes to needless complications such as blocks, ledges, apical transportations, and potentially, perforations. Regrettably, these iatrogenic events *do occur* and are clinically encountered (Figure 23-69). Perhaps the most overlooked and unsaid method to negotiate a blocked canal, bypass the ledge, or manage a transportation is determination, perseverance, and patience.

Techniques for Managing Blocks

Generally, when managing blocked and/or ledged canals, the shortest file that can reach working length is selected. Shorter instruments provide more stiffness and move the clinician's fingers closer to the tip of the instrument, resulting in greater tactile control. Appreciate that canals are frequently more curved than the roots that hold them. The armamentarium needed to successfully negotiate through a canal blockage is generally a series of 21 mm-length stainless steel files in sizes 0.06, 0.08, and 0.10 mm. Importantly, the obstructed and blocked canal is filled with full-strength sodium hypoclorite or a lubricant, such as Glyde, ProLube, or RC Prep.

The first step is to precisely locate the level of the block, then preshape that portion of the canal that is coronal to this blockage. During this exercise, care should be exercised to avoid contacting the block. An abrupt curve is placed on the apical extent of a size 0.06 or 0.08 K-type file. This precurved file can now be passed through the pre-enlarged canal and will reach the obstruction, curved. C Pilot files (*VDW, United Dental Manufacturers, Inc; Tulsa, Oklahoma, USA)* are available in sterile blister packs of six instruments and are recommended because they have a high resistance to bending, due to a special thermal hardening process. The precurved file is placed in contact with the block. A single back-and-forth reciprocating motion is used in conjunction with light apical pressure. The file is immediately removed from the root canal with an envelope movement.[148] The goal is to disintegrate the block with the tip of the file. Irrigate with 1.0 ml of NaOCl, repeat the same procedure three times, and then discard the file. A new file is selected and precurved, as the reciprocating action described dulls the tip of the file. Indeed, a new file ensures a sharp tip, which is mandatory for a successful outcome. The same handle motion and file movement is continued with as many files as required until a light catch is felt. At this stage, a size 0.08 C-Pilot file is usually selected and utilized, as

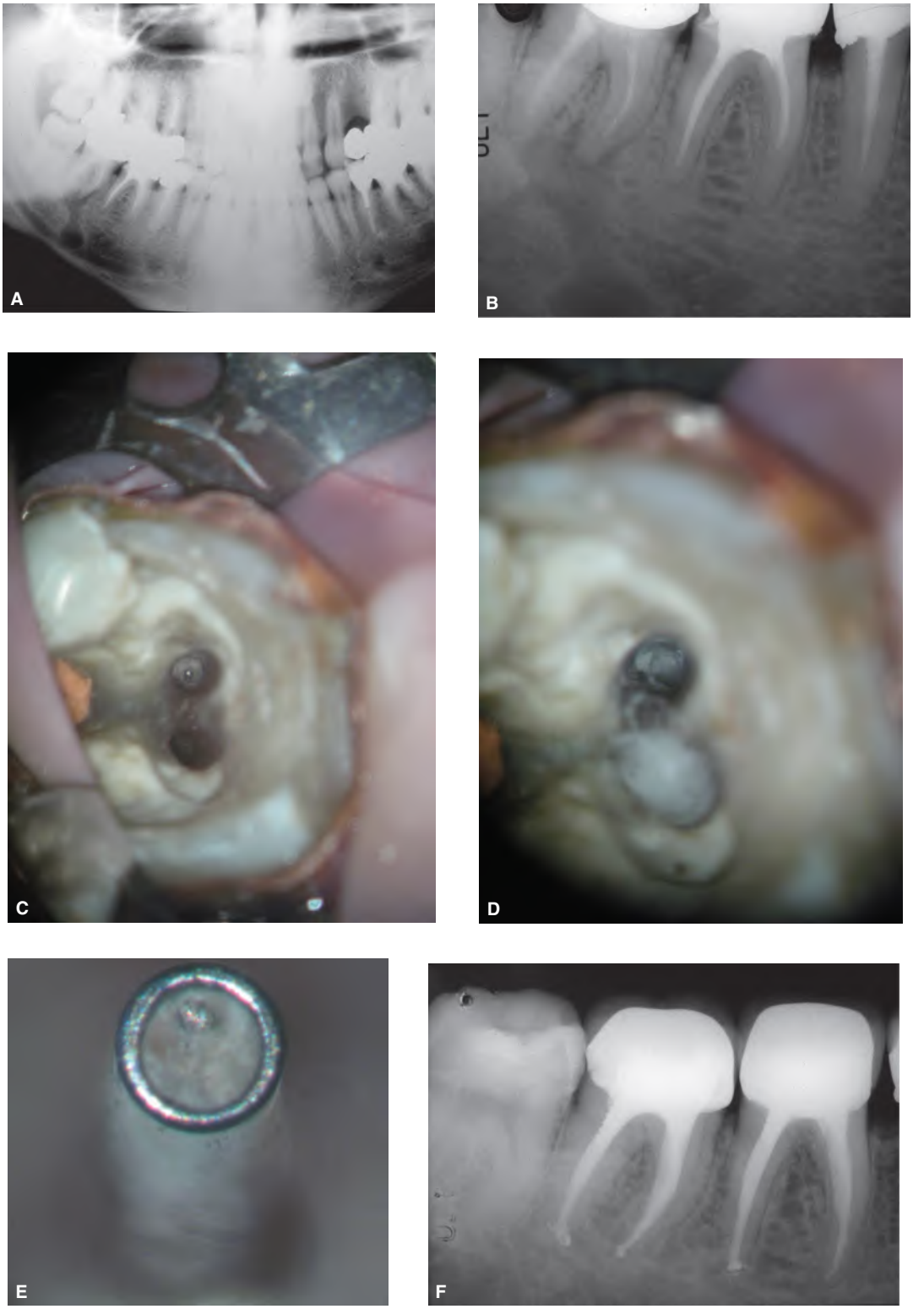


FIGURE 23-66 ***A***. Following an acute apical abcess on the mandibular right second molar, the patient developed numbness of the hemi-mandible. A bad quality Panorex shows an apical radiolucency in contact with the alveolar nerve. ***B***. Preoperative radiograph showing a separated Lentulo spiral located beyond the curvature of the mesial root. Patient was advised that there was no guarantee for successful removal of the separated instrument. ***C***. A good centering of the fragment was obtained with the dedicated drill and trephine from the Endo Rescue Kit. ***D***. The separated instrument was removed with the microtube technique. Complete cleaning and shaping was done. A week later, the paresthesia completely disappeared and the canals were obturated. ***E***. Photograph of the lumen of the tube showing the separated instrument frozen in a sea of composite. ***F***. One-year recall radiograph showing evidence of complete healing of the lesion.

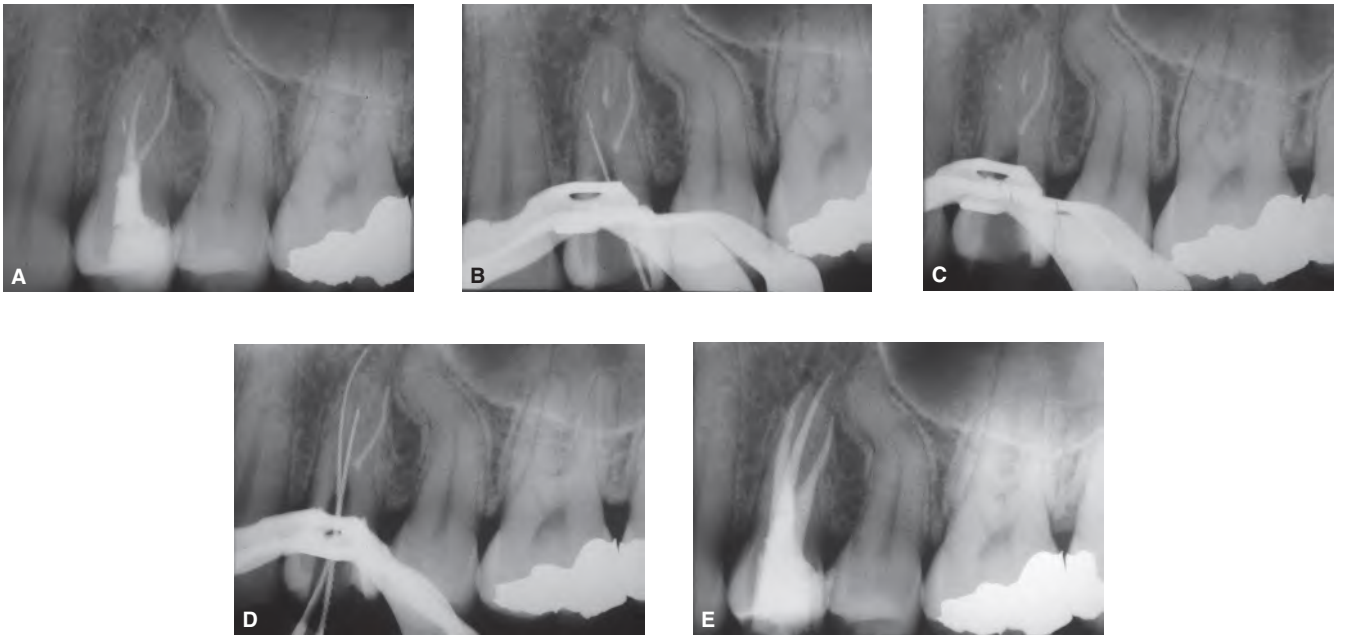


FIGURE 23-67 ***A.*** Preoperative radiograph of a three-rooted maxillary premolar with a failed root canal treatment and a separated NiTi instrument in the MB canal. ***B.*** A conservative radicular access was created up to the instrument. ***C.*** A thin ultrasonic tip was used to abrade the instrument. ***D.*** The residual fragment was bypassed. A ledge was present in the palatal canal. ***E.*** Postoperative radiograph showing the retreated case. (Courtesy of Dr. Dominique Martin, Paris, France.)

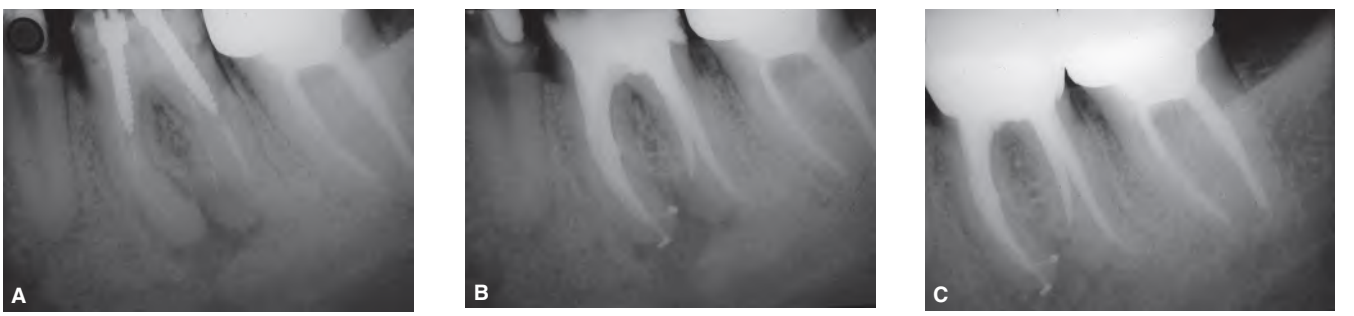


FIGURE 23-68 ***A.*** Retreatment was attempted on the first mandibular molar. The preoperative radiograph showed two large screw-posts and a broken separated instrument in the second distal canal. ***B.*** The separated instrument was removed but negotiation of the canal terminus was unsuccessful. A wait and see approach was adopted. ***C.*** Despite a less than ideal result, a one-year recall radiograph showing evidence of ongoing healing.

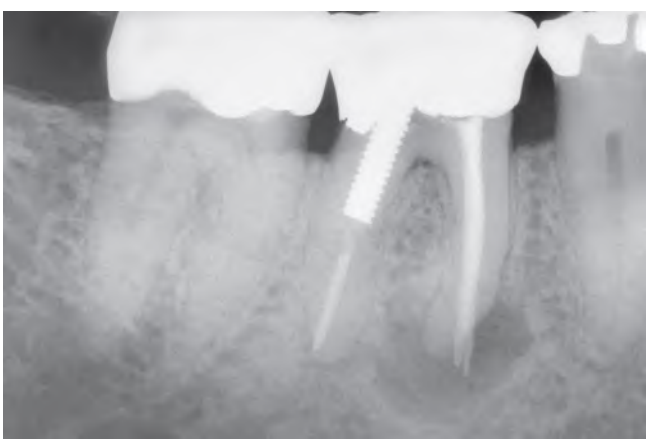


FIGURE 23-69 Preoperative radiograph of a mandibular right first molar showing a variety of restorative and endodontic breakdowns.

described, to expand the pathway following the use of the size 0.06 K files. In some clinical situations, up to a dozen files may be needed to negotiate through and bypass a blockage. Once the canal has been negotiated and patency established, the file is repeatedly used in a slip, slide, and glide manner to refine, expand, and smooth the glide path. Clinicians need to demonstrate perseverance when blocked canals are encountered, as oftentimes patience will be rewarded (Figure 23-70).

If no progress is made after diligent efforts over a timeframe of approximately two to three minutes, then the sodium hypochlorite is removed from the root canal system and replaced with a viscous chelator, such as RC Prep, Glyde, or ProLube. As the chelator begins to work deeper into the canal, the same technique as described above is then utilized.

At times, there are clinical situations where the aforementioned techniques have been carefully attempted, but either the file is not progressing apically or is not tracking the

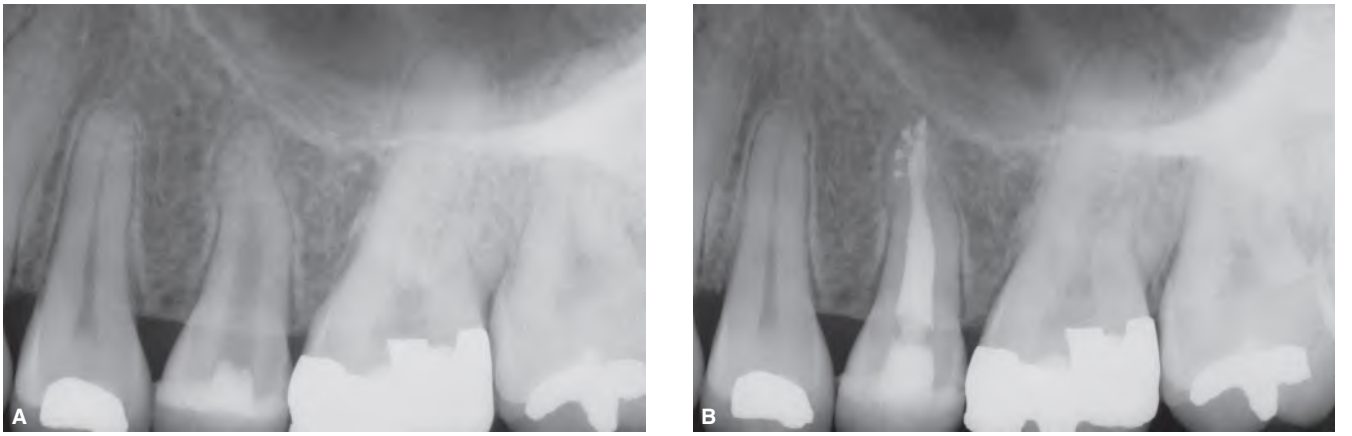


FIGURE 23-70 ***A.*** A preoperative radiograph of a maxillary left second premolar showing previous access and pre-enlargement of the canal in its coronal two-thirds. ***B.*** Postoperative radiograph provides an explanation as to the etiology of the original block. Note, the canal bifurcates apically and has four portals of exit.

pathway of the physiologic canal. What to do next requires a thoughtful treatment decision.[56] If the patient is truly asymptomatic and symptoms are not masked by a pharmaceutical, and if the periodontium is healthy and there are no lesions of endodontic origin, then the preparation is finished to the level of the obstruction and packed. The patient is informed of this less-than-ideal outcome, the importance of periodic recalls, and the possible need for future surgery. If the blocked canal is not negotiable and if clinical symptoms, periodontal breakdown, and/or a lesion of endodontic origin are present, then three-dimensionally pack the case, for at times post-operative films demonstrate hydraulics and obturation material to length. Regardless of the packing result, the patient needs to be advised of the importance of recall and that future treatment options include surgery, re-implantation, or extraction.

Techniques for Managing Ledges

An *internal* transportation of the canal is termed a "ledge" and frequently results when clinicians are preparing a curved canal and work short of length (Figure 23-71A). Many ledges are successfully bypassed using the techniques described for blocks. Once the tip of the file is apical to the ledge, it is moved in and out of the canal utilizing very short push-pull movements with emphasis on staying apical to the defect. When the file moves freely, slightly longer push-pull strokes are used to reduce the ledge and confirm the presence or absence of any residual internal canal irregularities. If the file is sliding easily, it is turned CW upon withdrawal because this motion will tend to straighten the apical one-third of a stainless steel file and allow it to rasp, reduce, smooth, or eliminate the ledge, which is typically on the outer wall of the canal curvature. During these procedures, try to keep the file coronal to the full working length so the apical foramen is handled delicately and kept as small as practical. When the ledge can be predictably bypassed, then efforts are directed towards establishing patency with a size 10 file. Gently passing a .02 tapered size 10 file 1.0 mm through the foramen increases its diameter to 0.12 mm and paves the way for the size 15 file.

Advantageous to ledge management is the utilization of ProTaper NiTi hand files (Dentsply Maillefer; Ballaigues, Switzerland). The major advantage of using NiTi files of greater taper to remove a ledge is their tapers are three to six times larger than conventional 0.02 tapered files. Once a precurved NiTi hand file has been inserted into the canal and its precurved tip has successfully bypassed the ledge, then its larger taper oftentimes eliminates or reduces the extent of the ledge using a minimum number of files. NiTi files are *not* introduced into the canal until the ledge has been bypassed, the canal negotiated, and patency established. Bypassing the ledge and negotiating the canal up to a size 15, and if necessary, a size 20 file, creates a pilot hole so the tip of the NiTi instrument can passively follow this secured glide path. To move the apical extent of a NiTi file past a ledge, the instrument must be precurved, preferably with the Bird Beak Pliers (Hu-Friedy; Chicago, Illinois, USA) (Figure 23-71B). To successfully precurve a NiTi instrument, the working end of the NiTi file is securely grasped between the jaws of the pliers and the handle is pulled through a radius of between 180 and 270 degrees. NiTi has shape memory and efforts to bend it must be exaggerated so that when the instrument is released from the pliers, it will have the desired curvature in its apical extent. The rubber stop on a manual NiTi instrument is oriented so the file's precurved working end can be directed to bypass and move apical to the ledge (Figure 23-71C). The ProTaper Shaping files, termed S1 and S2, may be used in this secured region of the canal until the full working length is reached (Figure 23-71D). Following the use of the S1 and the S2, the size 10 hand file is slidded down the canal to check if the ledge has been either reduced or eliminated. Regardless, the canal needs to be further enlarged to at least the ProTaper F1, and ultimately in accordance with the ProTaper finishing criteria.[149,150] A clinical case depicting ledge management is illustrated in Figure 23-72.

Based on preoperative radiographs, the clinician must make a decision whether to continue shaping procedures in hopes of eliminating the ledge or to abort if continued

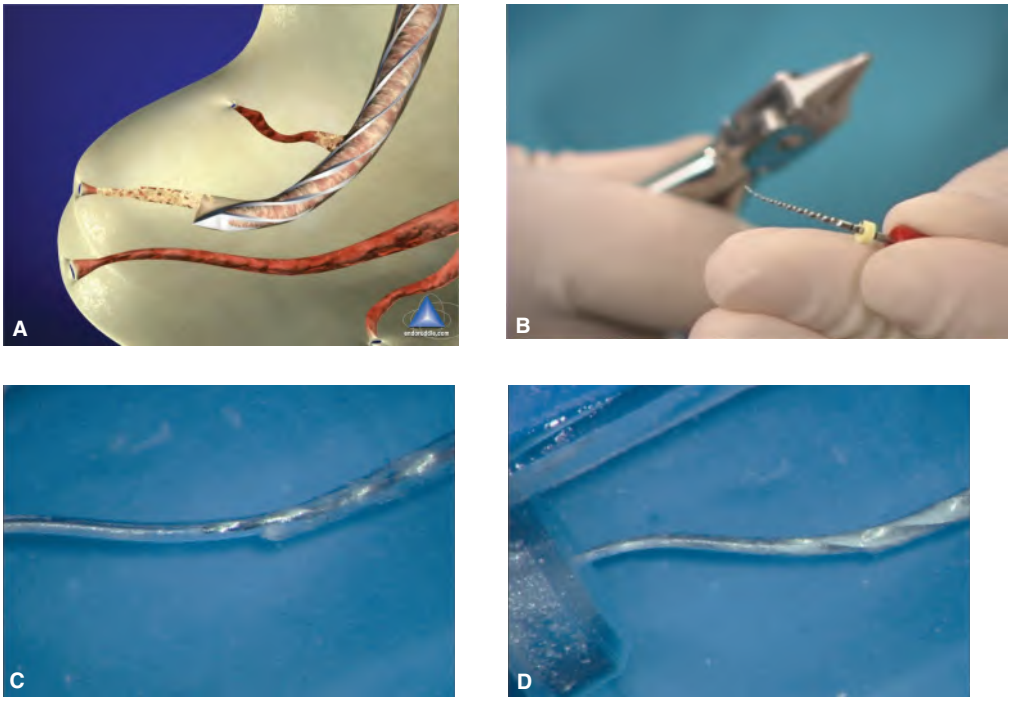


FIGURE 23-71 ***A.*** A ledge is created in a curved canal when pressure is applied on the file to reach the working length. ***B.*** Photograph showing how the Bird Beak Pliers can precurve a manual NiTi ProTaper file. ***C.*** A plastic block image demonstrates a manual, precurved ProTaper S1 file apical to the ledge. ***D.*** A plastic block image demonstrates a manual, precurved ProTaper S2 file has eliminated the ledge and is at length.

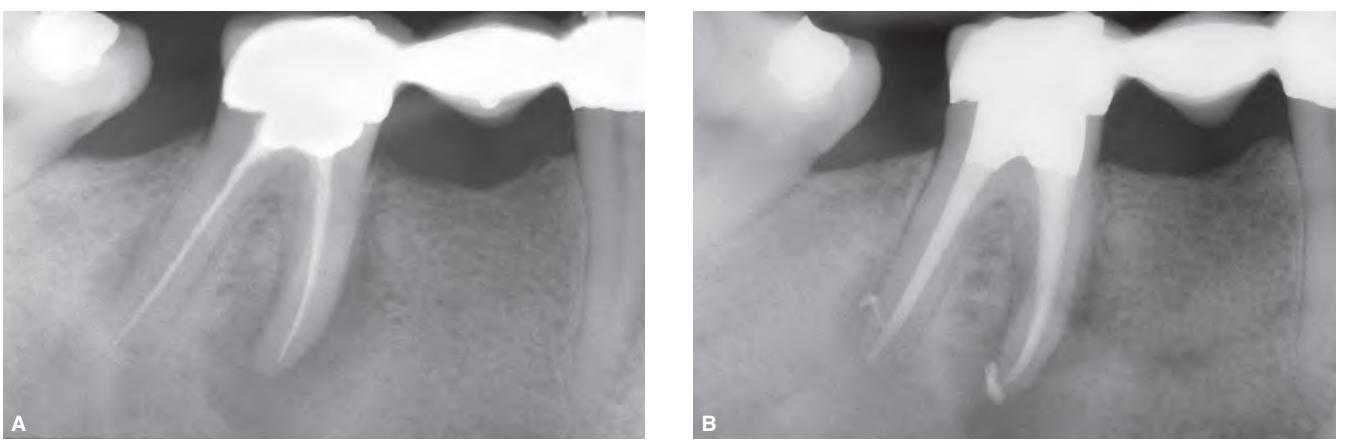


FIGURE 23-72 ***A.*** Preoperative radiograph showing an endodontic failure in a mandibular molar serving as a posterior bridge abutment. Note, the amalgam in the pulp chamber and that the mesial root appears to have been ledged. ***B.*** Post-treatment radiograph showing ledge management with the obturation materials following the root curvature.

efforts will weaken or perforate the root. Not all ledges can or should be removed. Clinicians must weigh risk versus benefit and make every effort to maximize remaining dentin. Ultimately the five mechanical objectives of cleaning and shaping should guide each clinician's actions.[139,141,147]

Cone fit is usually possible when the ledge is located in the middle portion of the canal. In the instance when the ledge cannot be eliminated in the apical one-third, fitting the master gutta-percha cone can be challenging. In these cases, the master cone is trimmed so its terminal diameter equals the D_0 diameter of the file that was snug at length. The cone is then precurved to simulate the curvature of the canal and its radicular portion placed in a dappen dish of 70% isopropyl alcohol. The master cone is removed after a few seconds and the clinician will appreciate the significant increase in its rigidity. An orientational notch is then placed on the butt-end of the master cone so as to identify working length and the direction of the cone's curvature. This technique greatly

facilitates the placement of the master cone during the packing procedure.

Techniques for Managing Apical Transportations

Moving the position of the canal's physiologic terminus to a new iatrogenic location on the external root surface equates to a transportation of the foramen.[151] Foraminal rips, zips, or tears are caused by carrying progressively larger and stiffer files to length.[151,152] If a transportation has occurred, then the canal exhibits reversed apical architecture and fails to provide resistance form to hold gutta-percha. This contributes to poorly packed cases that are vertically overextended, but internally underfilled.[3,77,153] In general, apical transportations can be categorized into three types, each requiring a specific treatment.

Type I

A *Type I* transportation represents a *minor* movement of the position of the physiologic foramen, resulting in an iatrogenic relocation on the external root surface (Figure 23-73A). In this instance, the clinician must weigh risk versus benefit when trying to create positive apical canal architecture. Generating shape coronal to the foramen requires the additional removal of dentin and could predispose to root weakening or to a lateral strip perforation. If sufficient residual dentin can be maintained and shape created above the foramen, then some of these iatrogenic cases can be three-dimensionally cleaned, shaped, and packed) (Figure 73B–D).[3] Regretfully, many canals whose foramens have been progressively transported are not amenable to a Type I-related treatment approach and will require an escalation in procedural steps.

Type II

A *Type II* transportation represents a *moderate* movement of the physiologic position of the foramen, resulting in a bigger iatrogenic relocation on the external root surface (Figure 23-74). As such, the apical one-third of the canal exhibits more reverse architecture than in a Type I transportation. In these instances, wet canals are common and attempting to create more coronal shape would risk weakening and/or perforating the root. In treating these canals, a barrier must be selected to control bleeding and provide a backstop to pack against during subsequent obturation procédures.[3]

The barrier of choice for a Type II transportation is mineral trioxide aggregate (MTA) (ProRoot, Dentsply Maillefer; Ballaigues, Switzerland) (Figure 23-75A).[154,155] MTA represents an extraordinary breakthrough for managing radicular repairs and can be used in canals that exhibit reverse apical architecture, such as those in immature roots and in Type II transportations. Additionally, MTA is used for nonsurgical and surgical perforation repairs or as a surgical root-end filling material. Remarkably, cementum grows over this nonresorbable and radiopaque material, thus allowing for a normal periodontal attachment apparatus.[156] Although a dry field facilitates visual control, MTA is apparently not compromised by slight moisture and typically sets brick-hard within 4 to 6 hours, creating a seal as good as or better than the best materials used today.[156]

MTA is easy to use and the powder is mixed with anesthetic solution, or sterile water, to a heavy viscous consistency. A fiberless 2 × 2 gauze may be used to wick-off surplus moisture and achieve the ideal viscosity.[77] A small aliquot of this cement is picked-up and introduced into the prepared canal with any microtube carrying device (Figure 75B and C). Generally, these devices will allow MTA to be placed in the middle one-third of virtually any posterior root and well-shaped canal. MTA is then gently tamped and coaxed down the canal to approximate length using a customized nonstandard gutta-percha cone as a flexible plugger. The gutta-percha cone should be trimmed apically to have sufficient diameter to effectively condense MTA.[3,77] This flexible gutta-percha plugger may be utilized to move MTA around root canal curvatures and into root defects.

At times, when pressing MTA into narrowing cross-sectional canal diameters, the material will become dehydrated and not move. Pressing on MTA with an apically trimmed, flexible gutta-percha plugger tends to compress the material and squeeze out water content from the MTA. In these instances, the MTA should be rehydrated in order to get it to slump, move, and adapt into any given root defect. Rehydration is accomplished by dispensing one or two drops of water into the canal. Gently insert a size 10 or 15 hand file into the MTA. Pump the file in short 1-2 mm strokes within the MTA to rehydrate the material so it can be readily moved and adapted. At this point, if there is excess water, simply wick it off using paper points. It is of critical importance to tamp a 3 to 5 mm aliquot of MTA to within 3 to 4 mm of any given defect or root apex.

When a sufficient volume of MTA is radiographically confirmed to be in position, it can be vibrated and moved into the defect and to length using the sonic EndoActivator system. As previously mentioned, the EndoActivator handpiece drives highly flexible, noncutting, polymer tips and three tip sizes are available in different D0 diameters and tapers. The tip selected is based on the length and diameter of the canal and position of the root defect. The tip is vibrated at the lowest power setting and is never placed closer than 2 to 3 mm from the root end. The sonic EndoActivator vibratory energy will generate a wave-like motion that will encourage MTA to slump, move, and adapt to the configurations of the canal laterally, as well as control its movement to and gently against the periapical tissues. Prior to initiating subsequent procedures, a dense 4 to 5 mm zone of MTA in the apical one-third of the canal should be confirmed radiographically.

MTA needs moisture to drive the cement to set and become brick-hard. Fluids are present external to the canal

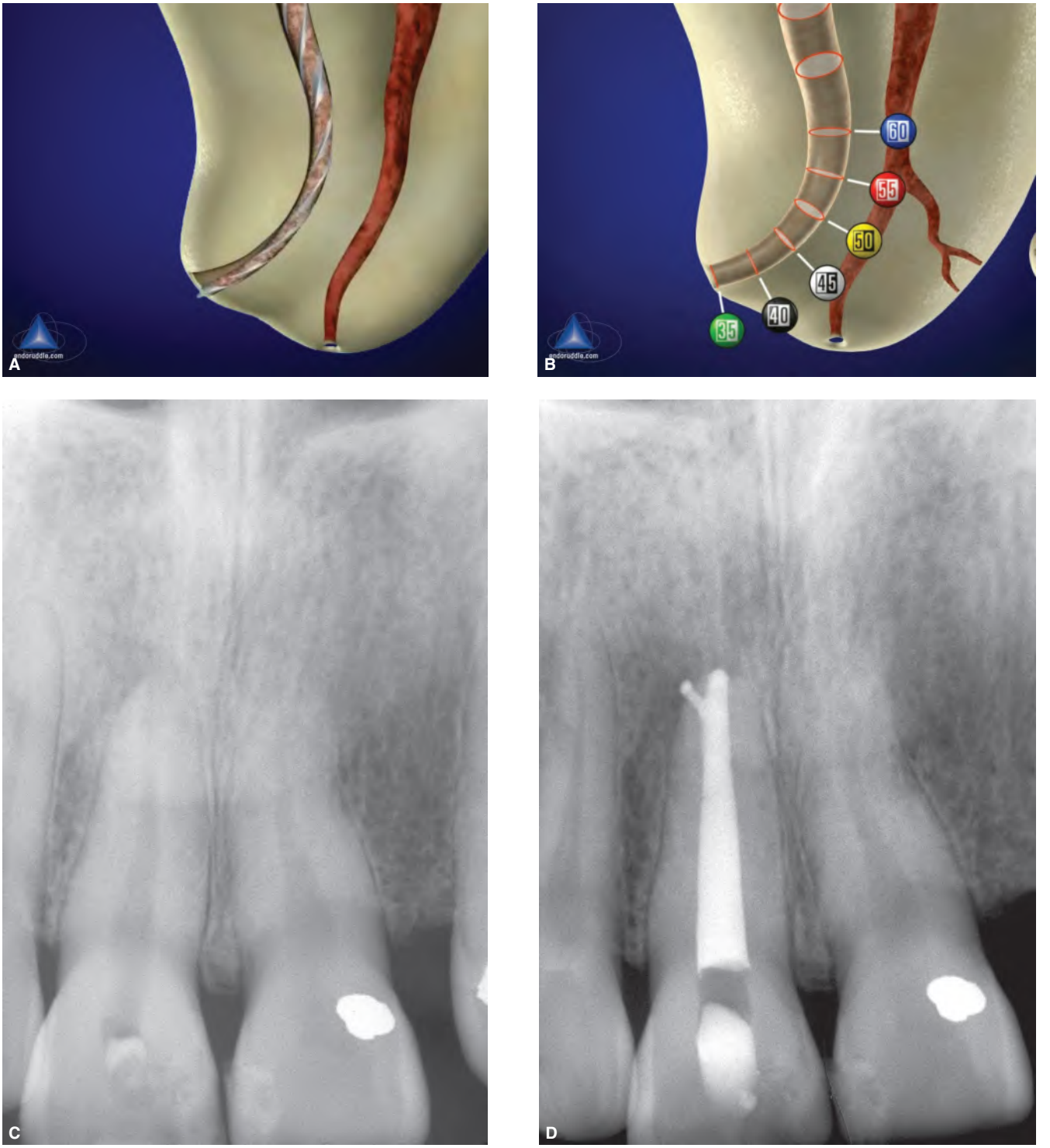


FIGURE 23-73 ***A.*** Illustration demonstrating a Type I transportation. Note, there has been a minor "shift" of the foramen. ***B.*** Management of a Type I transportation is directed towards creating shape above the foramen. ***C.*** Preoperative radiograph showing that the maxillary central incisor has been previously accessed, over-instrumented, and its canal exhibits reverse apical architecture. ***D.*** Post-treatment radiograph showing treatment of a *Type I* transportation. Shaped canals provide resistance form to hold gutta-percha.

and will fulfill the moisture requirement for the apical aspect of the positioned MTA. However, a cotton pellet will need to be sized, moistened with water, and placed against the coronal most aspect of the MTA that is within the canal. The tooth is then temporized and the patient dismissed. At a subsequent appointment, the temporary filling is taken out and the wet cotton pellet removed. The MTA cement is probed with a sharp explorer to determine its hardness. Typically, the material is brick-hard and the clinician can then obturate against this barrier. If the material is soft, it

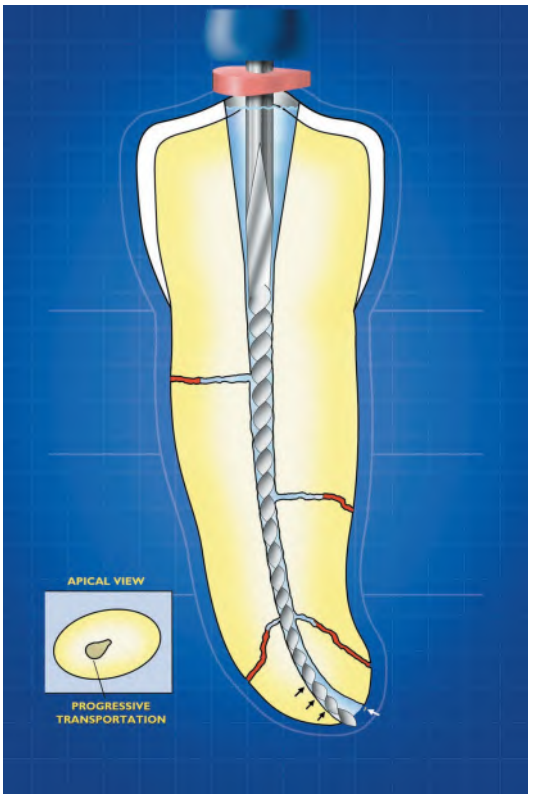


FIGURE 23-74 Illustration of a *Type II* transportation. Note, there has been a moderate "shift" of the foramen.

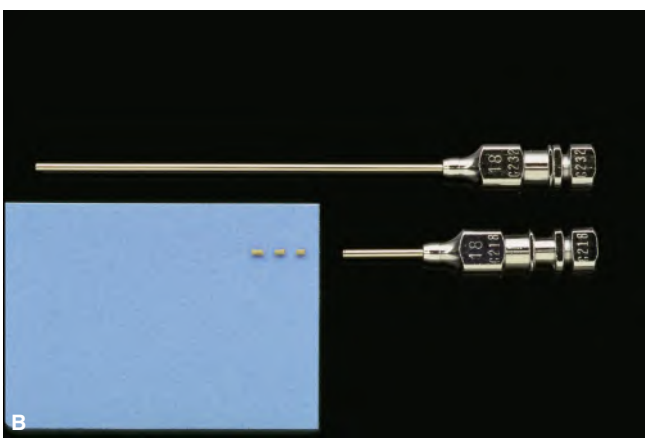
should be removed, the area flushed, dried, and a new mix of MTA placed. Upon subsequent re-entry, a hard barrier should exist which will provide a backstop against which to pack. The clinical steps for managing a *Type II* transportation are illustrated in Figure 23-76.

Type III

A *Type III* transportation represents a *severe* movement of the physiologic position of the foramen, resulting in a significant iatrogenic relocation on the external root surface (Figure 23-77). In this situation, the terminal extent of the canal is so badly mutilated that a barrier technique is usually not feasible and hence, three-dimensional obturation is impossible. If a tooth with a *Type III* transportation is to be salvaged, it requires obturation as best as possible with follow-up corrective surgery (Figure 23-78). Severe foraminal transportations that cannot be treated surgically are extracted.

ENDODONTIC PERFORATIONS

Perforations represent pathologic or iatrogenic communications between the root canal space and the attachment apparatus. The causes of perforations are resorptive defects, caries, or iatrogenic events that occur during and after endodontic treatment. Regardless of etiology, a perforation is an invasion into the supporting structures that initially incites inflammation and loss of attachment, and ultimately may compromise



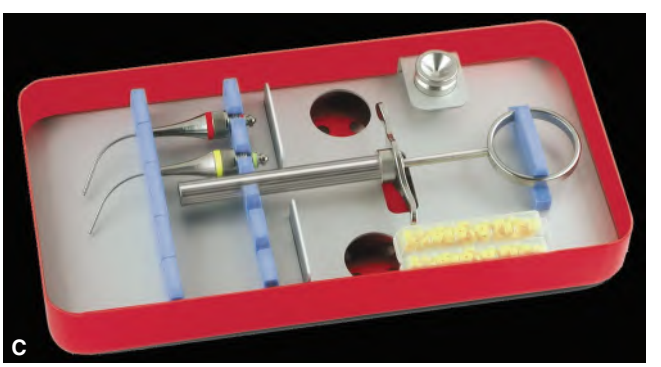


FIGURE 23-75 ***A.*** ProRoot, or MTA, is packaged in a powder form and then mixed with sterile water to a heavy cake-like consistency. ***B.*** A regular length 18 or 20 gauge spinal tap needle is appropriately shortened and utilized to carry small aliquots of MTA. ***C.*** The MAP system® (Micro Apical Placement system), Tulsa, Oklahoma, U.S.A.

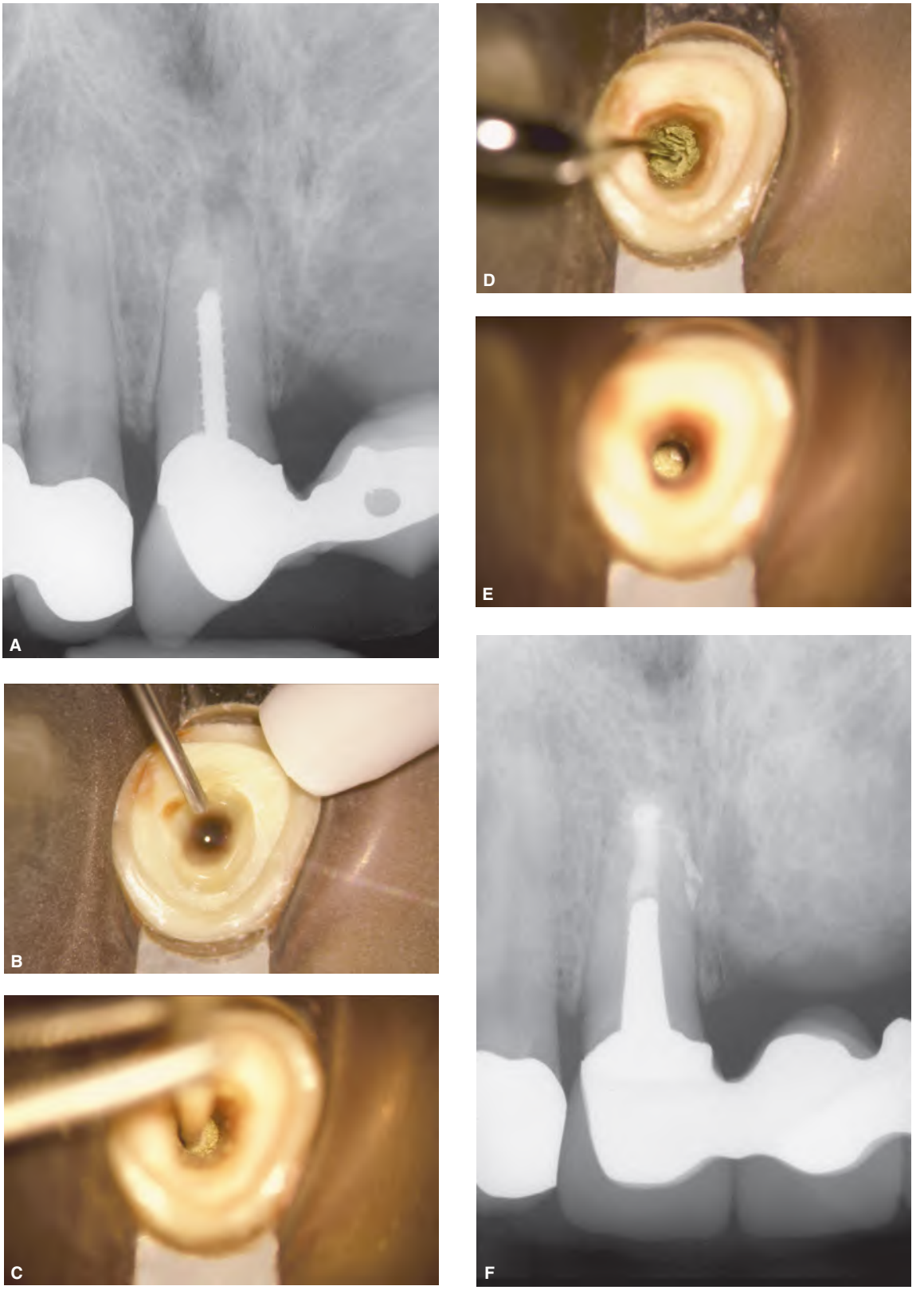


FIGURE 23-76 ***A***. Preoperative radiograph showing the maxillary right central incisor, serving as a bridge abutment, with a post and an empty canal system that exhibits reverse apical architecture. ***B***. Photograph at 8× magnification confirms disassembly. The foramen is visible following cleaning and shaping procedures. ***C***. Photograph showing tamping MTA into the apical one-third with a gutta-percha cone used as a flexible plugger. ***D***. The ProUltra ENDO-5 ultrasonic instrument vibrating MTA densely into the apical one-third of the canal. ***E***. Photograph taken on a subsequent visit shows brick-hard MTA. ***F***. Six-year recall radiograph showing a new bridge, post, the nonsurgical efforts, and excellent osseous repair.

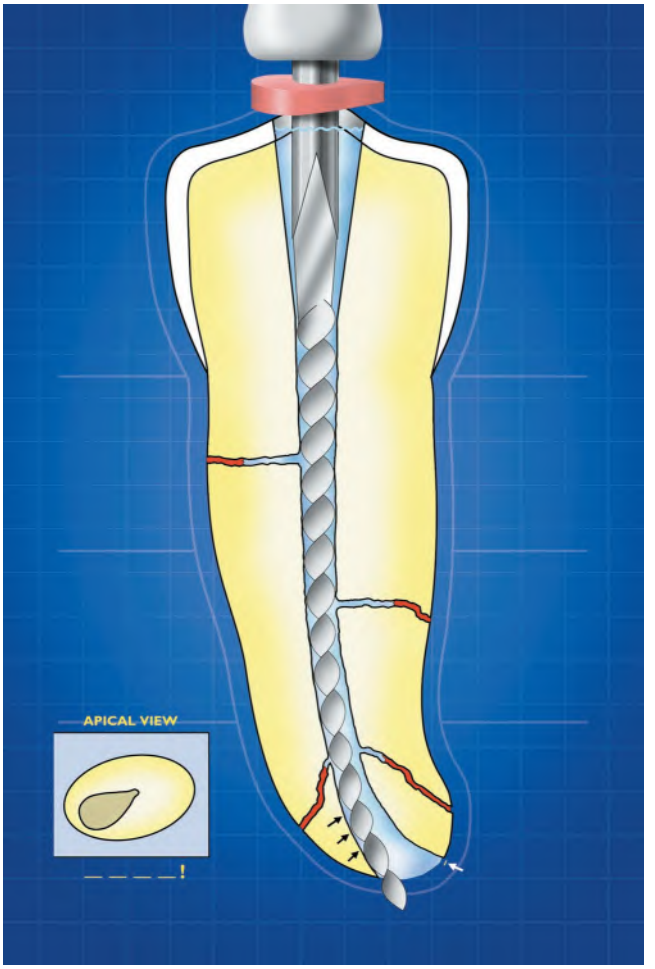


FIGURE 23-77 Illustration demonstrating a *Type III* transportation. Note, the foramen has been massively ripped and relocated from its original position.

the prognosis of the tooth. Perforations at or below the crest of bone certainly pose a serious threat to an otherwise favorable endodontic prognosis. The interdisciplinary team must decide whether to extract or direct efforts towards nonsurgical retreatment, surgical correction, or both.

Considerations Influencing Perforation Repair

When evaluating a perforated tooth, there are a number of variables that must be considered individually and collectively to properly guide treatment. Treating clinicians must identify the four dimensions of a perforation and understand how each of these entities critically affects treatment selection and prognosis.[3] Microscopes, paper points, electronic apex locators, and a suitable diagnostic radiopaque contrast solution are useful in determining the level, location, and extent of a perforation and the potential for successful management.[3,77] The four dimensions of a perforation always occur in combination, which synergistically complicates treatment outcomes.

1. *Level*: Perforations occur in the coronal, middle, and apical one-thirds of roots. Furcal perforations have similar considerations as coronal one-third perforations. Perforations at this level threaten the sulcular attachment and pose different treatment challenges than more apically occurring perforations. In general, the more apical the perforation, the more favorable the prognosis.
2. *Location*: Perforations occur circumferentially on the buccal, lingual, mesial, and distal aspects of roots. The location of the perforation is not so important when nonsurgical treatment is selected, but its position is critical and may preclude surgical access if this approach is considered.
3. *Size*: Perforation size greatly affects the clinician's ability to establish a hermetic seal. The area of a circular shaped perforation can be mathematically described as πr^2. Therefore, doubling the perforation size with any bur or instrument increases the surface area to seal by four-fold. Clearly, many perforations are ovoid in shape due to the nature of occurrence and represent enormous surface areas to effectively seal.
4. *Time*: Regardless of etiology, a perforation should be repaired as soon as possible to discourage further loss of attachment and prevent sulcular breakdown. Chronic perforations exhibiting a loss of sulcular attachment pose treatment challenges that potentially escalate to surgical correction and efforts directed toward guided tissue regeneration procedures.[157–159]

Periodontal Condition

Teeth that have been perforated must be thoroughly examined periodontally. Specifically, the sulci of these teeth must be carefully probed.[160,161] If the attachment apparatus is intact, then timing is critical, and the treatment is ideally directed toward nonsurgically repairing the defect. However, if there is periodontal breakdown with resultant loss of attachment, then interdisciplinary consultations, including ortho, perio, endo, and restorative, should guide treatment planning, sequencing, and prognosis. In these instances, a decision must be made between a nonsurgical repair versus surgical correction, recognizing that, at times, combined treatment may be required to salvage the tooth. Importantly, extraction may be wise and a fixed bridge or restored implant considered.

Esthetics

Perforations in the anterior region can definitely impact esthetics. Patients that exhibit a high lip line can be esthetically compromised by soft tissue defects such as clefts, recessions, or discrepancies in the inciso-gingival dimensions of a crown, as compared to the adjacent teeth.[162,163] Importantly, tooth-colored restoratives must be carefully chosen in areas that demand esthetics and are selected from the best materials currently available in adhesion dentistry.[164] Sadly, certain traditional restoratives contributed to tooth discoloration, soft tissue tattoos, and a serious compromise in esthetics.

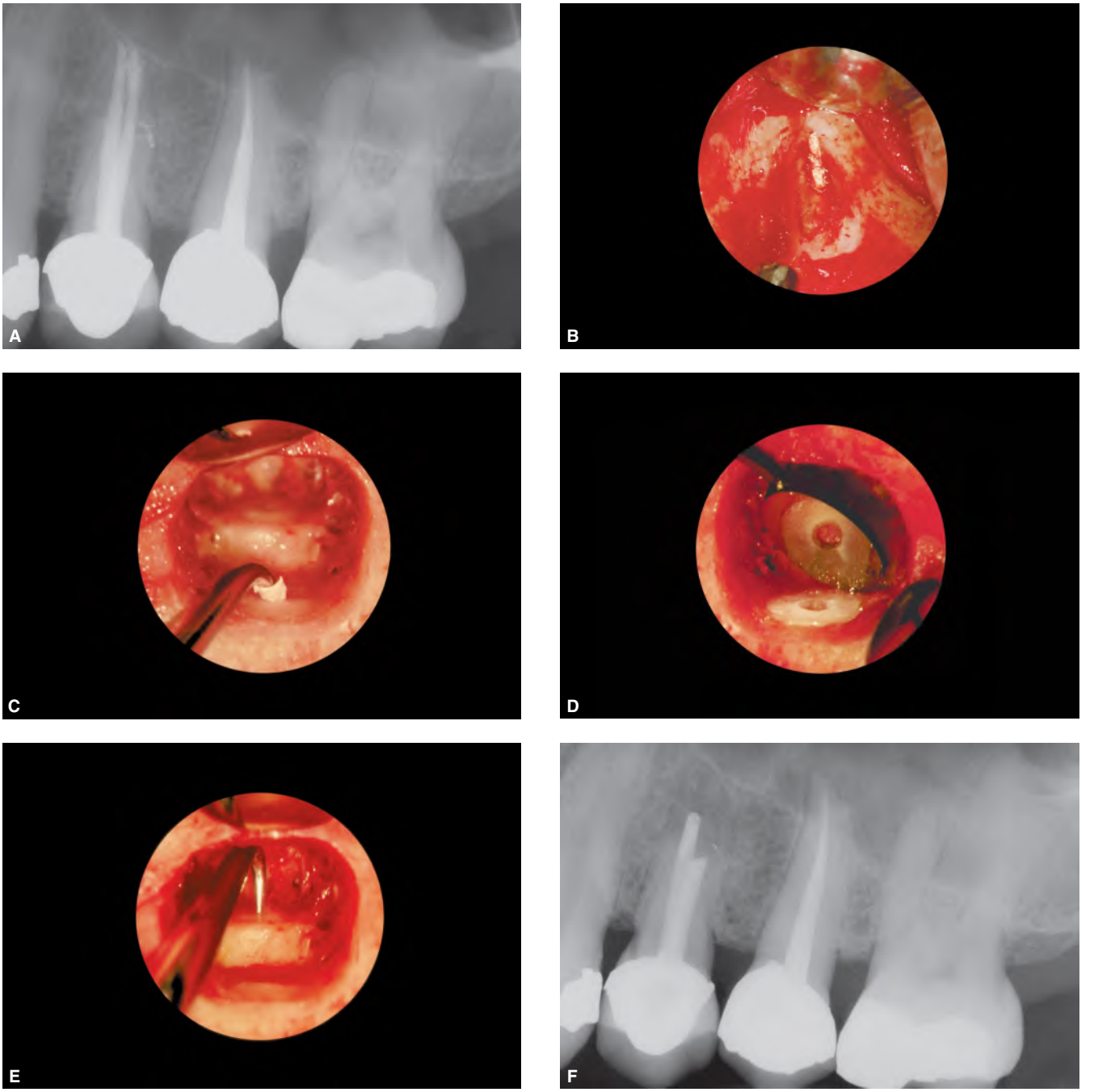


FIGURE 23-78 ***A.*** Preoperative radiograph of a maxillary left first premolar. Note, the packed buccal canal does not conform to the mechanical objectives of cleaning and shaping. ***B.*** Following flap elevation, a surgical photograph at 12× magnification shows gutta-percha exiting through a significantly ripped and relocated foramen. ***C.*** Surgical photograph at 12× magnification shows osteotomy, apicoectomies, and ultrasonic root-end preparation of the buccal canal. ***D.*** Surgical photograph at 12× magnification through a sapphire-plus micro-mirror depicts the finished buccal root-end preparation. ***E.*** Ultrasonic root-end preparation of the lingual canal. ***F.*** Four-year recall film showing the apically corked canals and excellent osseous repair.

Vision

Magnification glasses, headlamps, and transilluminating devices facilitate vision and are important adjuncts in addressing perforations. However, the dental operating microscope has become the standard of vision and may be utilized to more predictably repair perforation defects nonsurgically, thus reducing the need for surgical intervention and its associated risks.[3,77]

Treatment Sequence

When there is a perforation and the canal has not been fully prepared, then the defect should be repaired prior

to proceeding with definitive endodontic treatment. Repairing the perforation will enable the clinician to control bleeding into the canal, confine irrigation, and achieve a controlled and hydraulic fill. However, any given perforated canal should be optimally enlarged and prepared, when necessary, to improve access to the defect, enhance visualization, and to importantly minimize post-repair instrumentation. When repairing a perforation, it is important to maintain the pathway to the physiologic canal because the barriers and restoratives utilized could inadvertently block the canal. A gutta-percha segment or a collagen plug can by placed apical to the defect to prevent canal blockage during the perforation repair procedures. In failing endodontic cases exhibiting a perforation, the existing obturation material may be used to hold the position of the canal. This allows the clinician to first repair the perforation before proceeding with endodontic disassembly and retreatment efforts. However, in this sequence of treatment, caution must then be exercised not to disrupt the perforation repair material during subsequent disassembly, canal preparation, and obturation procedures.

Materials Utilized in Perforation Repair

Hemostatics

Many perforation defects exhibit massive bleeding upon re-entry. As such, clinicians need to be familiar with a few hemostatic agents and materials that can predictably arrest bleeding.[165] A dry field enhances vision while creating an environment for the predictable placement of a restorative agent. Calcium hydroxide, a time-honored material, can be passively syringed into the canal, hydraulically moved to place, and allowed to remain in the canal/defect for four to five minutes or longer. The calcium hydroxide is then flushed from the field using sodium hypochlorite. Two or three applications of placing and then removing calcium hydroxide usually begins to control the bleeding. When the clinician can not obtain hemostasis, calcium hydroxide can be advantageously left in the canal until a future appointment.[166] Other increasingly important materials that can be used to achieve hemostasis by various mechanisms include collagen, calcium sulfate, freeze-dried bone, and mineral trioxide aggregate (MTA).[159,165,167] There are other hemostatics that exist, but they are not generally selected due to factors such as cost, ease of handling and placement, or their byproducts. Ironically, some of the best hemostatics, like ferric sulfate, leave a coagulum behind which may promote bacterial growth, compromise the seal at the tooth-restorative interface, and jeopardize the prognosis.[169]

Barrier Materials

The two main challenges a clinician faces when attempting to repair a perforation are hemostasis and the controlled placement of a restorative material. Barriers help produce a "dry field" and also provide an internal matrix or "backstop" against which to condense restorative materials.[3] In general, barriers can be divided into resorbable and nonresorbable; however, it is critical to note that the restorative material utilized oftentimes dictates the barrier that is selected.

Resorbable Barriers Bleeding into the tooth must be arrested in order to successfully manage perforations. Hemostasis is accomplished by passing a resorbable barrier nonsurgically through the access cavity, internally through the perforation defect, and ideally into a three-walled osseous defect. Resorbable barrier materials are intended to be placed within the bone and not left inside tooth structure. The barrier should conform to the anatomy of the furcation or root surface involved. Although a variety of resorbable barriers exist, collagen and calcium sulfate materials are best employed due to supporting research, ease of handling, and observed clinical results.

- *Collagen materials*, such as CollaCote (Sulzer/Zimmer Dental; Carlsbad, California, USA), exhibit excellent working properties that provide complete hemostasis.[165] CollaCote is biocompatible, supportive of new tissue growth, resorbable in 10 to 14 days, and left in situ.[158,167] Based on the size of the defect and the available access, pieces of CollaCote are cut to appropriate sizes and carried into the access cavity. The material is incrementally placed through the tooth and *into the osseous defect* until a solid barrier is established at the cavo surface of the root. Hemostasis is typically achieved in two to five minutes. Collagen barriers have been widely used in conjunction with amalgam, super EBA, and other non-bonded restoratives.[169] CollaCote is contraindicated as a barrier if adhesion dentistry is contemplated, as it absorbs moisture and will contaminate the restorative.
- *Calcium sulfate*, such as Capset (Life Core Biomedical; Chaska, Minnesota, USA), can be used as both a barrier and hemostatic material in perforation management.[159,169,170] Calcium sulfate creates a tamponade effect, mechanically plugging the vascular channels once it sets. Capset is remarkably biocompatible, does not promote inflammation, and is bioresorbable in 2 to 4 weeks. This material is syringed through the tooth and into the osseous defect utilizing a microtube delivery system. During its placement, the calcium sulfate will fill the osseous defect and a portion of the space within the root defect. Calcium sulfate rapidly sets brick-hard and may be sanded back flush to the external root surface with ultrasonic instruments. Calcium sulfate is the barrier of choice when utilizing the principles of wet bonding.[164,170,171] Importantly, the perforation defect can be rinsed of contaminants in preparation for adhesion dentistry.

Nonresorbable Barriers: Calcium-Silicate based Cements

- *Mineral Trioxide Aggregate (MTA)*, available in gray and white colors, exhibits excellent tissue biocompatibility and can be used both as a nonresorbable barrier and restorative material (Figure 23-75A).[172] MTA has

many clinical applications and represents an extraordinary breakthrough for managing radicular repairs.[173–175] MTA is the barrier of choice when there is potential moisture contamination or when there are restrictions in technical access and visibility. Furthermore, MTA can be used as the sole radicular restorative material or as a barrier against which to pack another material. The mixing and use of MTA was previously discussed in the *"Techniques for Managing Apical Transportations"* section earlier in this chapter.

- *Biodentine™*[176,177] is a new calcium silicate-based cement, initially created as a dentine substitute (Figure 23-79). It is a synthetic nano-particle multipurpose material, indicated to replace damaged dentine, both in the crown and in the root. Its chemical composition is different from that of MTA, as it has no gypsum and less water. Biodentine combines the biocompatibility of MTA with many additional and desirable characteristics. These characteristics include bioactivity-inducing hard tissue formation, good handling characteristics, self-adhesion to dentine, no shrinkage, and a better antibacterial action.

Further, Biodentine has a setting time significantly shorter than MTA (12–15 minutes). In endodontics, the indications for using Biodentine are the same as for MTA; namely, pulp capping, partial pulpotomy, deciduous teeth pulpotomy, apexification, perforation, and apical surgery.

Restoratives

Central to success when repairing a perforation is to select a restorative material that is easy to use, nonresorbable, biocompatible, esthetically pleasing, and one that provides a complete seal. The materials commonly employed to repair perforations include the time-honored but decreasing in popularity amalgam, SuperEba resin cement (Henry J. Bosworth; Skokie, Illinois, USA), composite bonded restoratives (Geristore, DenMat Corporation; Santa Maria, California, USA) calcium phosphate cement, MTA, and Biodentine.[175–180] The choice of the restorative repair material is based on the technical access to the defect, the ability to control moisture, and the esthetic considerations.

FIGURE 23-79 The Biodentine™ package.

Techniques for Repairing Perforations

The specific barrier and restorative selected to repair a perforation site should be based on sound research, judgment, experience, training, esthetics, ease of handling, and the advantages or disadvantages of a particular material in a specific clinical setting. This section describes the armamentarium, materials, and techniques required to repair perforations.

Management of Coronal One-Third and Furcal Perforations

The major difference between *coronal one-third* and *furcal floor* perforations is the shape of the resultant root defect. Mechanical perforations that occur in the furcal floor are generally round, while those occurring in the lateral aspects of roots are ovoid by nature of occurrence. When managing these perforations, the clinician must first isolate the perforation site. Generally, if the perforation is mechanical and has just occurred, it is non-infected and hence clean. In this situation, and if hemostasis is achieved, the defect can be immediately repaired. However, if the perforation is chronic and exhibits microleakage, then the defect needs to be cleaned and prepared before receiving the restorative. Ultrasonic instruments are ideal for preparing perforation sites because of their geometries and coatings, and because they afford unsurpassed vision.

Once the defect has been properly prepared, an appropriate barrier material and restorative are then selected based on the following esthetic considerations:

- In a coronal one-third perforation where esthetics is a concern, a calcium sulfate barrier in conjunction with adhesion dentistry is generally utilized.[171]
- Historically, amalgam and more recently SuperEBA have been utilized to repair coronal one-third perforations when esthetics was not an issue. Presently, MTA is rapidly becoming the material of choice for repairing non-esthetic coronal one-third defects because of its many desirable attributes. Biodentine is a good alternative to MTA.

Following perforation repair, the tooth can be three-dimensionally shaped, cleaned, and packed if this has not already been accomplished (Figure 23-80).

Management of Perforations in the Middle One-Third

Iatrogenic perforations in the *middle one-third* of roots are generally caused by endodontic files, Gates Glidden drills, or large misdirected posts. By nature of occurrence, these defects are ovoid in shape and typically represent relatively large surface areas to seal.

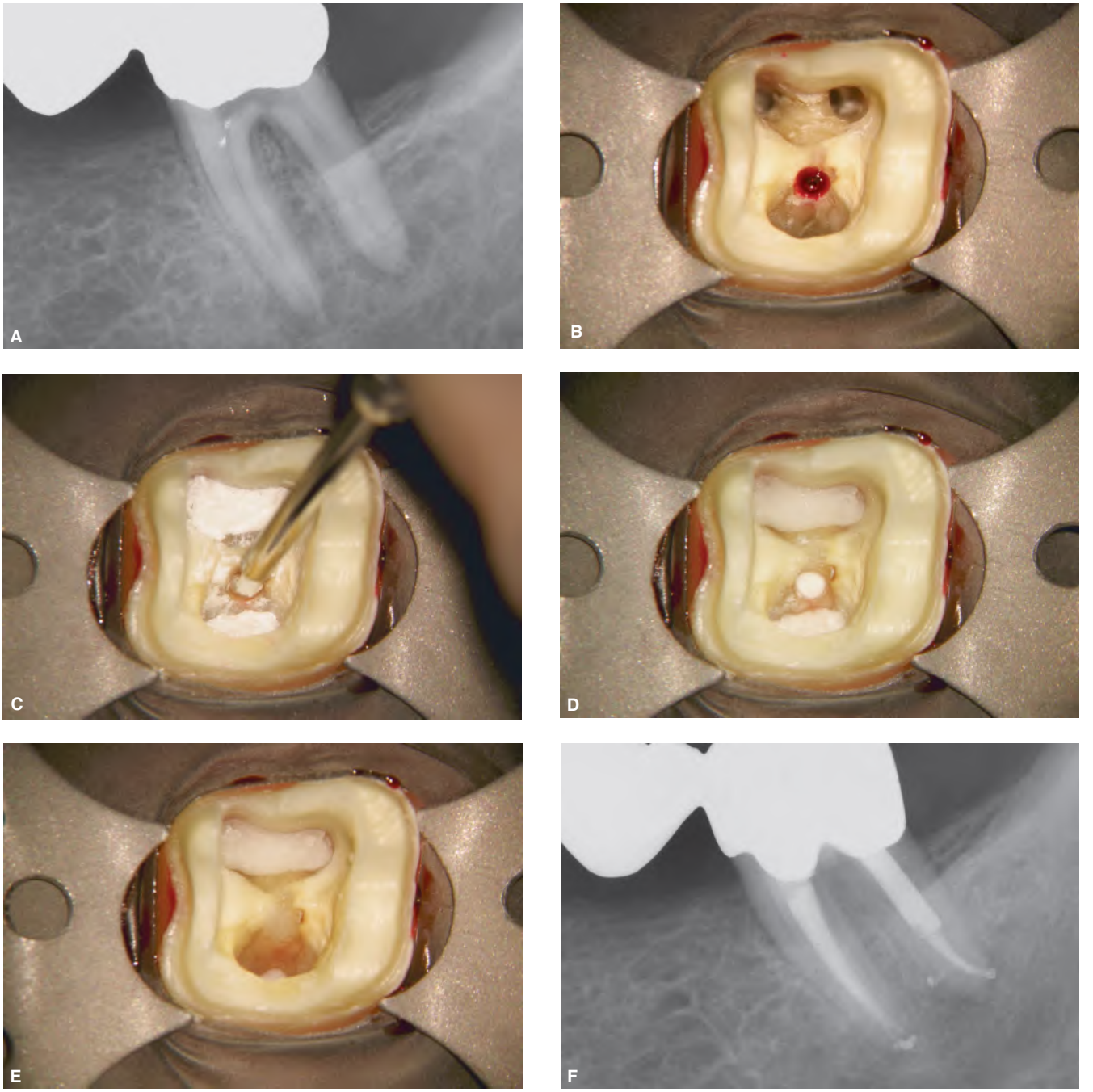


FIGURE 23-80 ***A.*** Preoperative radiograph of an endodontically involved mandibular left second molar serving as a bridge abutment. Note the previous access and possible pulp chamber floor perforation. ***B.*** Photograph showing the identified canal orifices and a furcal floor perforation. ***C.*** Segments of Collacote overlying the canal orifices and calcium sulfate being introduced into the perforation via an STN. ***D.*** Furcal management. The absorbable barrier is placed flush to the external cavo root surface. ***E.*** Perforation repair utilizing a dual cured composite restorative material. ***F.*** Five-year recall radiograph showing a new bridge and osseous repair furcally and apically.

Middle one-third or strip perforations have the same technical considerations as coronal one-third perforations, *except* the clinician is now dealing with defects located deeper and further away from the occlusal table. The factors that must be addressed in order to successfully treat these more apically-positioned perforations are hemostasis, access, utilization of micro-instrumentation techniques, and the selection of best materials in a challenging environment. When managing deeper defects, positioned on the lateral walls of canals, vision is enhanced when direct access exists or can

be safely created. In some instances, direct access may not be possible without irreversibly compromising the structural integrity of the tooth and indirect repair techniques are required. Generally, perforations that occur secondary to overzealous canal instrumentation are sterile and do not require further modification utilizing micro-instrumentation procedures. However, failing endodontic cases are associated with microleakage and bacterial invasion and may require ultrasonic instrumentation to clean and refine the defect in preparation for repair.

When the middle one-third perforation is a small defect, if the bleeding can be arrested and the canal dried, then the perforation can be sealed and repaired during three-dimensional obturation. However, if the defect is large and there is nuisance moisture, and/or the canal cannot be definitively dried, then the perforation must first be repaired before three-dimensional obturation. It is wise to prepare the canal as optimally as possible prior to initiating the perforation repair procedures. As has been stated earlier, a prepared canal will facilitate access to the defect *and* minimize post repair instrumentation. To prevent obstructing the root canal space during the repair procedures, any readily retrievable material is placed in the canal and apical to the defect before the perforation is repaired.

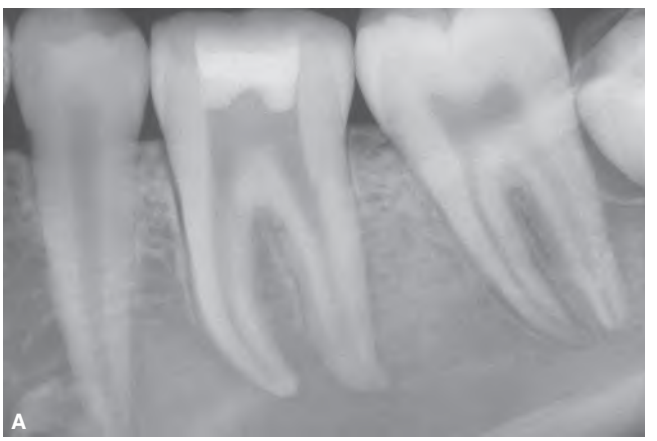
In these teeth, due to the difficult access, limited visibility, and the uncertainty of a moisture-free environment, the restorative/barrier material of choice is MTA. MTA is mixed and carried into the field and managed in accordance with the techniques discussed earlier in this chapter. Upon reappointment, MTA will invariably be hard and the clinician can proceed with the required treatment (Figure 23-81). A similar case was treated with Biodentine (Figure 23-82).

Management of Perforations in the Apical One-Third

Perforations occurring in the apical one-third of roots primarily result from iatrogenic shaping procedures. Blocks and ledges invite deep perforations and result from inadequate irrigation, inappropriate instrumentation, and failure to maintain patency. Roots perforated in their apical one-third can pose surprises and frustration during nonsurgical retreatment. It is quite common that a root perforated in its apical one-third holds a canal that is both blocked and ledged. Recognizing the etiology of this type of perforation has led to surgical correction utilizing apicoectomy and root-end-type procedures. However, it is generally best to first attempt nonsurgical retreatment to enhance the existing endodontic treatment and, if present, to identify and treat missed canals.

The clinician should attempt to negotiate the physiologic terminus with the concepts, instruments, and techniques previously discussed under *"Techniques for Managing Blocks"* and *"Techniques for Managing Ledges."* On occasion, the apical extent of the file will stick, handle flutter will be noticed, and the instrument will begin to track along the true canal pathway. The file is gently worked to negotiate the physiologic pathway, establish patency, and pave the way for the next successively larger instrument. The next sequentially larger precurved file is then inserted and carried apical to the perforation, but not necessarily to length. This "holding file" maintains the pathway of the true canal and prevents it from being blocked during subsequent repair.

MTA is the material of choice for repairing deep perforations, especially when a dry environment and technical access are not possible. MTA is placed, as has been previously described. To prevent the holding file from being frozen in a sea of MTA as it hardens, the instrument is grasped with Stieglitz pliers and moved up and down in short 1 to 2 mm amplitude strokes. The loosened holding file is then sectioned so its coronal-most aspect is below the occlusal table. A radiograph should be taken to confirm the position of the MTA and the quality of the repair. A wet cotton pellet is placed within the pulp chamber and against the MTA, the tooth is provisionalized, and the patient dismissed. Upon re-entry, the holding file is removed, and if the MTA is brick-hard, then



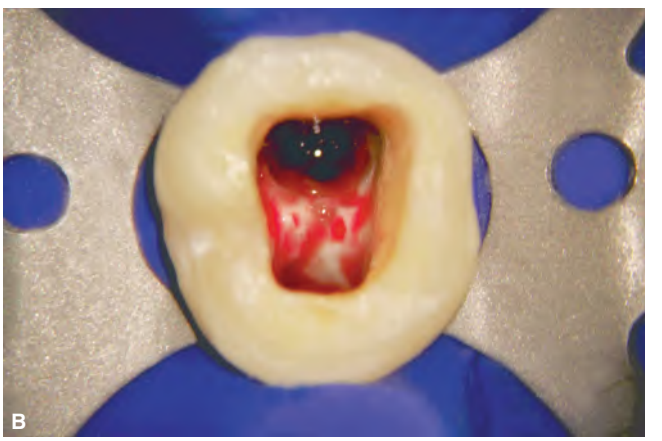


FIGURE 23-81 *(Continued on facing page)*

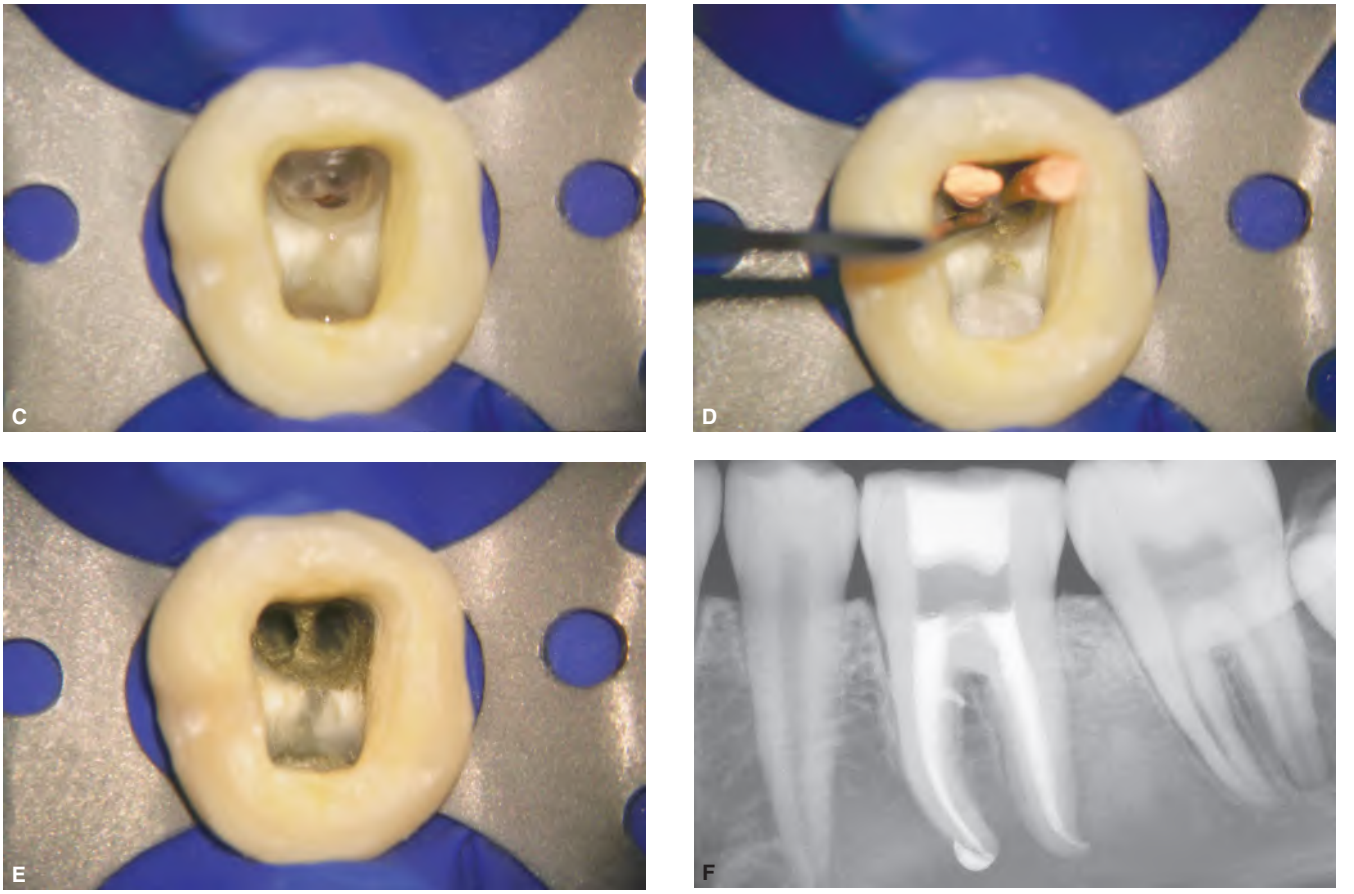


FIGURE 23-81 ***A.*** Preoperative radiograph of an endodontically involved mandibular left first molar. Note the previously over-enlarged mesial root. ***B.*** A photograph taken upon re-entry showing significant bleeding emanating from the mesial root. ***C.*** Photograph demonstrating hemostasis and the position of the strip perforation. ***D.*** Gutta-percha cones in the MB/ML canal systems to prevent their blockage. MTA is being vibrated into the perforation defect. ***E.*** At the subsequent visit, the gutta-percha cones are removed, the perforation repaired and the canals ready to pack. ***F.*** Postoperative radiograph showing the provisionalized tooth, four canal systems packed and the apical one-third anatomy.

voluminously irrigate and gently finalize the preparation, fit a gutta-percha master cone, and three-dimensionally obturate. It is wise to provisionalize these cases and follow up periodically before placing a definitive restoration. The clinical steps for managing apical one-third perforations are illustrated in Figure 23-83. It is also important to acknowledge that not all perforations can be corrected nonsurgically, even when the best technologies are utilized in the hands of the most skilled practitioners. Certain cases will still require surgical treatment or extraction (Figure 23-84).

FUTURE

As we have seen in this chapter, a variety of techniques exist to address endodontically failing teeth and the ultimate aim of orthograde retreatment is to improve the previous treatment (Figure 23-85). However, not all failures are amenable to successful nonsurgical retreatment. Clinicians need to weigh risk versus benefit and recognize that, at times, a referral, surgery, or extraction might be in the patient's best interest. The future of endodontics is bright and the demand for initial treatment and retreatment will grow significantly in the years immediately ahead. This growth will be attributable to better trained general dentists and specialists, alike, advancements in technologies, and the public's growing recognition that the naturally retained tooth is the ultimate dental implant. New advancements in root canal disinfection methods will continue to move our discipline ever closer, toward eliminating all the contents from both the instrumentable and noninstrumentable portions of the root canal system.[181–183] Properly performed, endodontic treatment is the cornerstone of restorative and reconstructive dentistry.

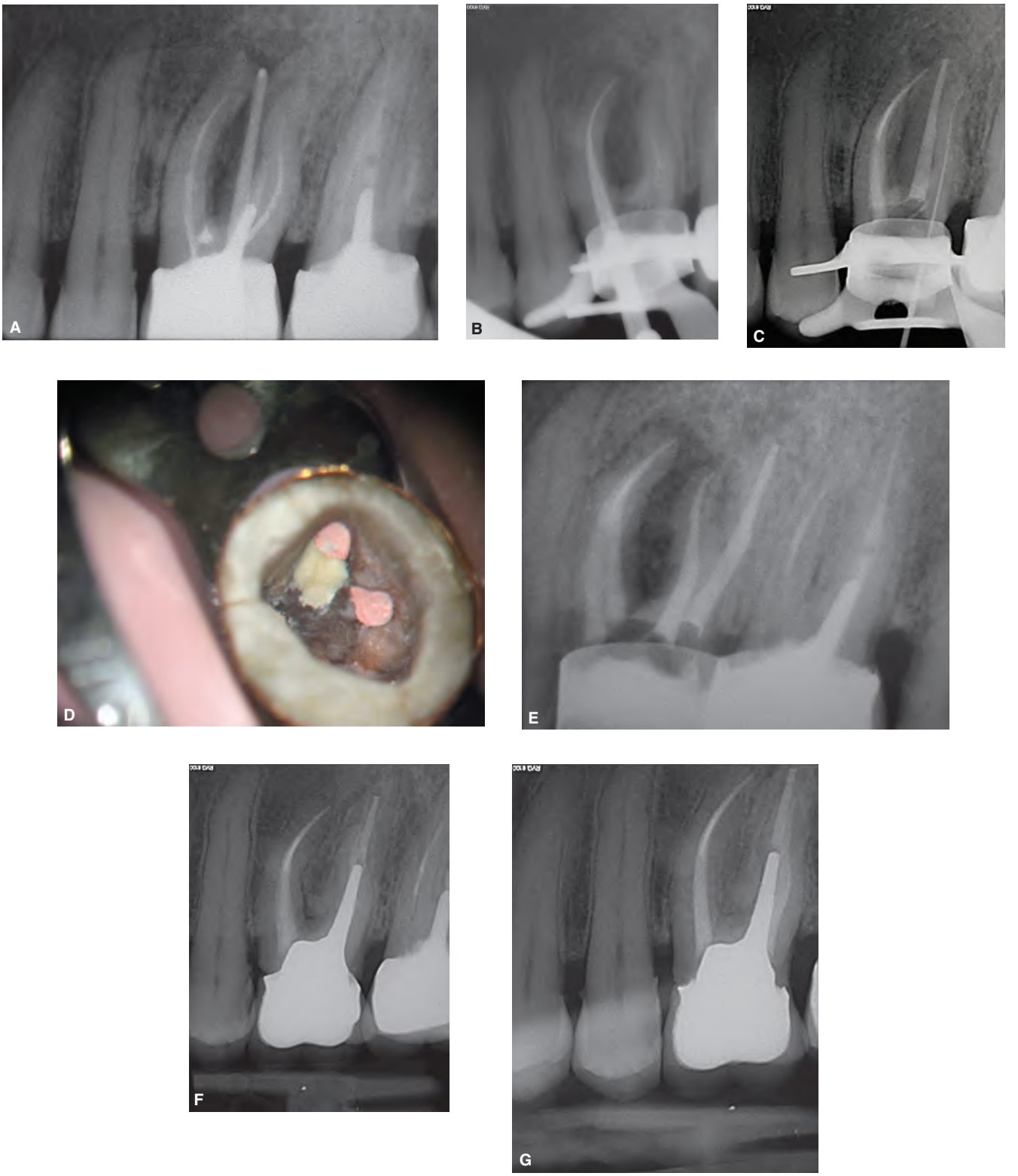


FIGURE 23-82 ***A.*** Preoperative radiograph showing a large intraradicular lesion and a sinus tract associated with the maxillary left first molar. A two-millimeter perforation of the inner wall of the MB canal was present below the orifice. ***B.*** After shaping of the MB-1 and MB-2 canals, the gutta-percha points serve as a matrix for placement of Biodentine to repair the defect. Note the separated instrument in the DB canal. ***C.*** After Biodentine setting, the gutta-percha points were removed and the mesial canals obturated. The DB canal was negotiated after removal of the broken instrument. Note the lower radio-opacity of Biodentine as compared to gutta-percha. ***D.*** A photograph of the pulp chamber showing repair of the perforation. ***E.*** Postoperative radiograph after completion of the treatment. ***F.*** One-year recall radiograph showing evidence of healing of the intra-radicular lesion. ***G.*** Two-year recall radiograph showing evidence of complete healing and formation of a new periodontal ligament in the furcation area.

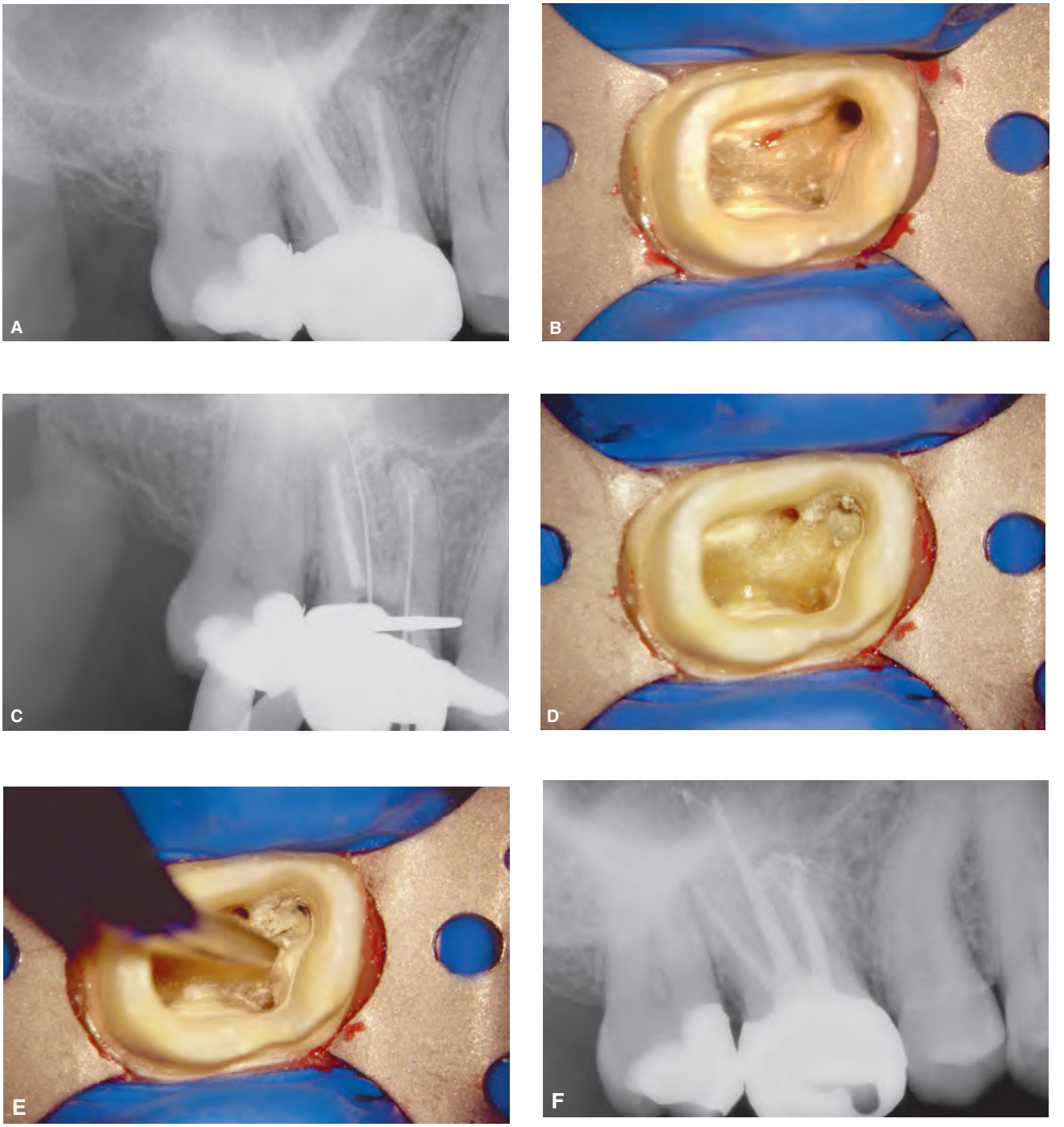


FIGURE 23-83 ***A.*** Preoperative radiograph of a failing endodontic treatment in a maxillary right first molar. Note the MB root is perforated in its apical one-third. ***B.*** Photograph showing crown removal, the MB-1 canal orifice and bleeding emanating from the MB-2 canal system. ***C.*** A no. 10 file at the terminus of the MB-2 canal system. ***D.*** The prepared MB-2 canal system, a holding file within the MB-1 canal intentionally sectioned below the occlusal surface, and MTA utilized to repair the perforation defect. ***E.*** At a subsequent visit, the MTA was brick-hard. At this point the holding file is removed from the MB-1 canal system. ***F.*** A Post-treatment radiograph showing the treated MB-1 and MB-2 canal systems, the perforation repair and the block/ledge management of the DB and palatal canal systems.

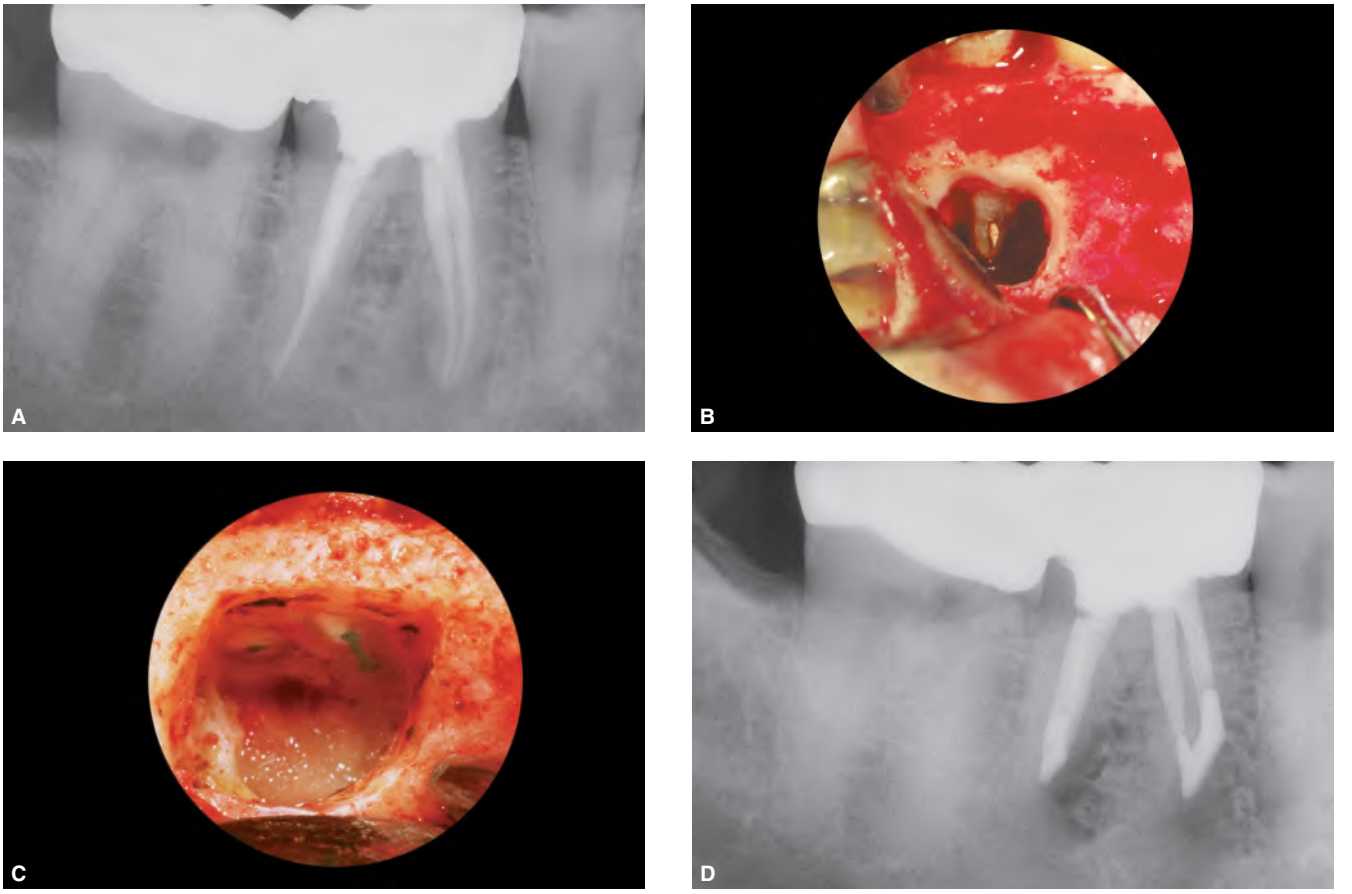


FIGURE 23-84 ***A.*** Preoperative radiograph of a failing endodontic treatment in a mandibular right first molar. This specific radiographic angle does not reveal the etiology of failure. ***B.*** Photograph showing gutta-percha perforating the buccal aspect of the mesial root. ***C.*** The apicoectomy and retrograde procedures. Note the management of the isthmus between the MB and ML foramina. ***D.*** Post-treatment radiograph showing the surgical efforts.

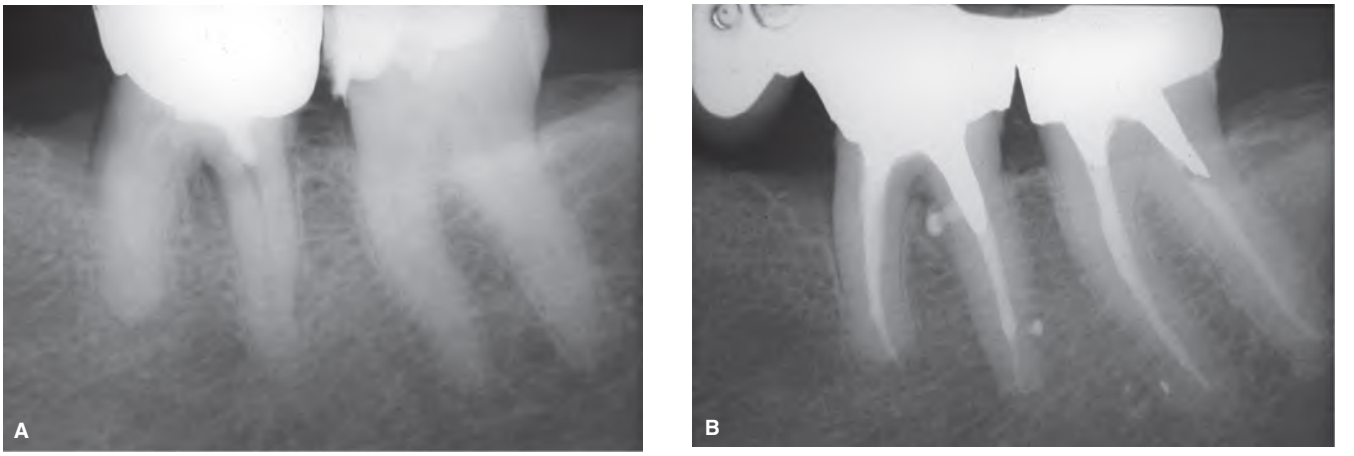


FIGURE 23-85 ***A.*** The mandibular left first molar is scheduled to be retreated prior to initiating a full mouth restoration. ***B.*** Five-year recall radiograph showing evidence of complete healing of the furcation area.

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(Continued on facing page)

Chapter 24

Surgical Endodontics

Mahmoud Torabinejad, Mohammad Sabeti, Gerald N. Glickman

Nonsurgical initial root canal treatment is a highly successful procedure when diagnosis and technical aspects are carefully performed (Figure 24-1). Several studies with large sample sizes have reported that nonsurgical root canal therapy has a survival rate of over 90% when adequately performed.[1–3] Lazarski et al.[1] reported a functional survival rate of 94% for 44,613 cases over 3.5 years in the United States. Salehrabi and Rotstein[2] reported 97% survival for 1.1 million patients over eight years in the United States, and Chen et al.[3] reported 93% survival for 1.5 million teeth over 5 years in Taiwan. Torabinejad et al.,[4] in a systematic review, showed that initial root canal treatment is associated with a very high survival rate of 97% over six years. Iqbal and Kim[5] found similar results in another systematic review when they compared the survival rates of restored endodontically treated teeth with those of implant-supported restorations. When initial nonsurgical root canal therapy is unsuccessful, the treatment alternatives are nonsurgical retreatment (revision) and surgical treatment (Figures 24-2 and 24-3). When these are not feasible or practical, extraction with or without replacement is indicated. Studies have shown that between 77% and 89% of retreated cases are successful after retreatment of the original root canal therapy.[6–8] However, there are situations in which surgical treatment (surgical endodontics) is needed to retain a tooth that would otherwise be extracted.

Surgical endodontics is a delicate procedure, involving more than simply cutting off the apex of a root and placing a filling at the root end(s). The purposes of surgical endodontics are the sealing of all portals of exit from the root canal system, eliminating microorganisms and other antigens from contaminating the periapical tissues, providing an environment for complete regeneration of periapical tissues, and saving natural dentition. During the past two decades, the science and art of surgical endodontics have significantly changed. With the use of the operating microscope, ultrasonic tips, and new root-end filling materials, surgical endodontics has enabled clinicians to retain many teeth that might otherwise be extracted.[9]

This chapter describes the history of surgical endodontics, indications and contraindications, procedures involved in incision for drainage, periapical surgery, and corrective surgical procedures such as root resection, hemisection, and root

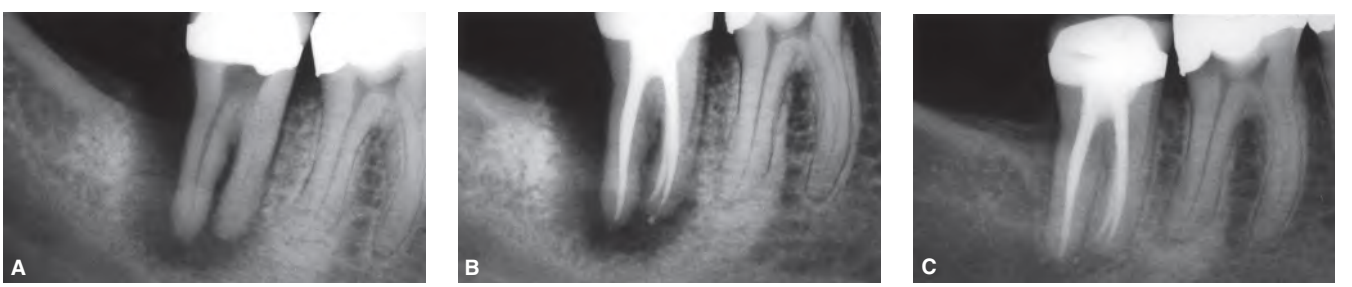

FIGURE 24-1 ***A.*** Preoperative radiograph of a second molar with pulpal necrosis and evidence of severe asymptomatic apical periodontitis. ***B.*** Postoperative radiograph of the tooth. ***C.*** Postoperative radiograph two years after root canal treatment shows complete resolution of the periapical pathosis. (Courtesy of Dr. David Steiner, Gig Harbor, Washington, U.S.A.)

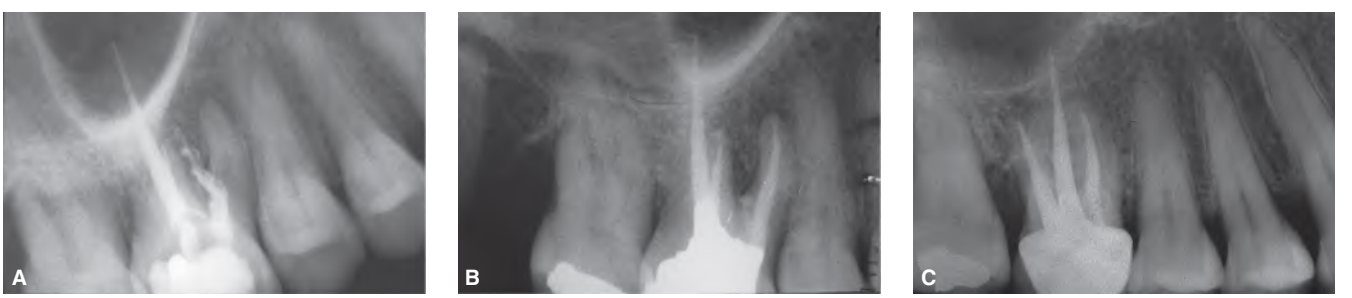

FIGURE 24-2 ***A.*** A search for the MB canal in a partially calcified chamber resulted in a furcation perforation and extrusion of filling materials into the periapical tissues. An apex locator reading or an angled radiograph would have detected this type of error. ***B.*** The initial treatment was redone and the perforation was sealed with MTA. ***C.*** Radiograph taken three years later shows no evidence of pathosis in the repaired area. (Courtesy of Dr. George Bogen, Los Angeles, California, U.S.A.)

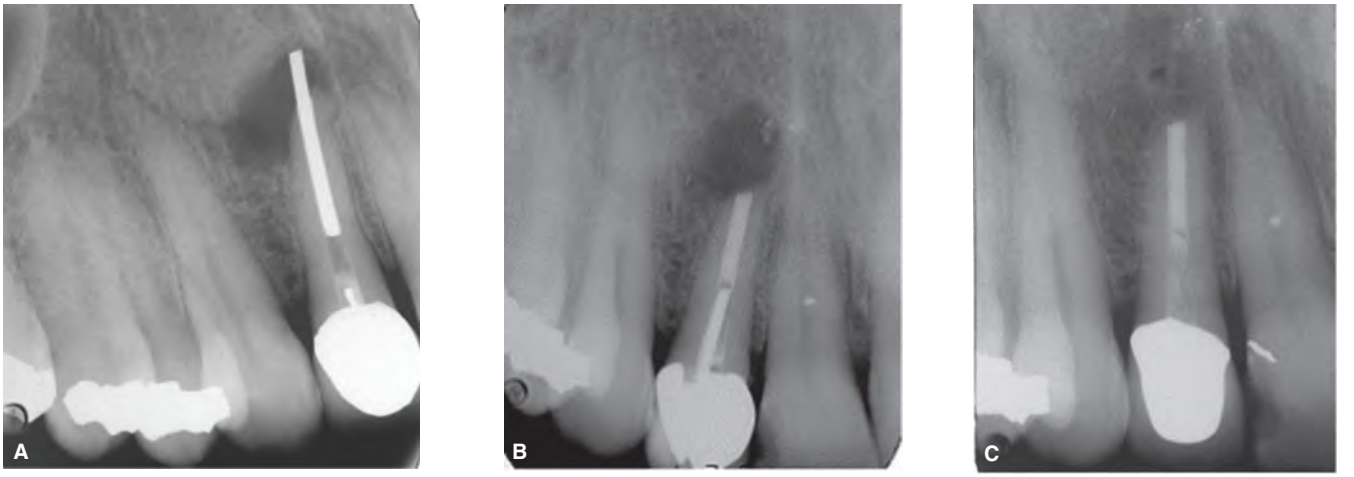

FIGURE 24-3 ***A.*** Periapical radiograph shows extensive radiolucency around the apex of the maxillary right incisor with an overextended silver point and an inadequate coronal seal. ***B.*** The root canal treatment was retreated nonsurgically and surgically using MTA as root canal filling material. ***C.*** Radiograph taken three years later shows complete resolution of the periapical lesion. (Courtesy of Dr. Christopher Sechrist, Redlands, California, U.S.A.)

extraction, bicuspidization, tooth replantation, transplantation, as well as the outcomes of these procedures.

HISTORY OF SURGICAL ENDODONTICS

Surgical endodontics has a long history (Table 24-1). The first recorded endodontic surgical procedure is related to the replantation of a tooth in the 11th century by Abu al-Qasim al-Zahrawi (Albucasis).[10] Hunter[11] discussed replantation and emphasized the need for preservation of the periodontal ligament for successful replantation. There are reports indicating that the first root resection was performed by C.S. Smith[12] in 1871. However, there are some documents contradicting these reports, supporting an argument that root-end resection was being performed for more than a century before Smith. For example, Berdmore[13] performed root-end resection in the middle of the 18th century.

In 1886, G.V. Black recommended the resection of the apex of the root of a tooth by using a fissure bur and saving the tooth.[14] During the annual session of the American Dental Association, Rhein[15] suggested that the treatment of a tooth with chronic apical abscess should include filling of the root canal followed by removal of the abscess. Ottolengui,[16] in 1892, recommended root-end resection following the filling of a root canal.

The period from 1900 to 1939 was the era of focal infection theory and wholesale extraction of teeth with pulpal and periapical diseases. William Hunter discussed and supported "the role of sepsis and of antisepsis in medicine" in his presentation and supporting focal infection theory. Due to the clinical success of surgical techniques, focal infection theory did not impact surgical endodontics in Europe. In two separate articles in the 1890s, Partsch[17,18] described root-end resection techniques by using a semilunar incision (Partsch incision). The endodontic surgical techniques, especially molar surgeries, were developed, practiced, and published in Europe. In 1915, Neumann[19] discussed both the theoretical and clinical techniques of mandibular molar surgeries. He provided the details of anatomical considerations for periapical surgery. Neumann described a surgical flap in attached gingiva that was similar to that proposed by Oschenbein and Luebke. A review of surgical flap designs was discussed by Hofer[20] in 1936.

In 1936, Peter[21] published a textbook on surgical endodontics that served as a prelude for today's surgical

TABLE 24-1 Examples of Historical Reports Related to Endodontic Surgery (1884–1998)

Authors	Year	Described Techniques
Farrar	(1884)	Root amputation
Black	(1886)	Apicectomy
Rhein	(1890)	Root amputation
Schamberg	(1906)	Flap design
Koch	(1909)	Fistulation
Buckley	(1914)	Flap design
Lucas	(1916)	Amalgam as a root-end filling
Fawn	(1927)	Causes of surgical endodontic failures
Coolidge	(1930)	Cementum formation over resected roots
Hill	(1931)	Cementum formation over resected roots
Maxmen	(1959)	Scope of periapical surgery
Leubke, et al.	(1964)	Indications for endodontic surgery
Arens, et al.	(1981)	(Text)
Gutmann & Harrison	(1991)	(Text)
Bellizzi & Loushine	1991	(Text)
Arens, et al.	1998	(Text)

endodontics. This book reviewed the literature, elaborated on historical developments, outlined the indications of surgical endodontics, and described the flap design for posterior teeth. Peter[21] discussed the position of the inferior alveolar canal in relation to the mandibular molar roots and the relationship of the maxillary sinus to the apices of the maxillary teeth. Different techniques for management of root-end resection and preparation were also developed in this era. Von Hippel[22] in 1914 described the vertical slot root-end preparation. Rudd[23] proposed a retentive groove, and Matsura[24] later on introduced this concept for contemporary surgical endodontics.

Root resection is a procedure that has been performed since the 1800s. In a 10-year study on the evaluation of the success of root resections, Langer[25] described this procedure for maintenance of a multi-rooted tooth that has a furcation defect. Bauer[26,27] and Kronfeld[28] recommended histological evaluations of root-end resections, based on the findings of their studies. Cavina[29] introduced the histological findings of Bauer to clinical procedures in 1930. At the same time, Gottlieb[30] and Steinhardt[31] discussed the healing of periapical tissues and provided a histological report that addressed the regeneration of the cementum and periodontal ligament. Hartzel recommended obturation of the root canal providing a "sterile" canal prior to root-end resection.[32]

In an article by Grossman,[33] many different surgical endodontic procedures were discussed that were available at the time. For example, he discussed hemisection, radisectomy (root amputation, root resection), endodontic implants, tooth replantation, transplantation, and implantation.[33] At that time, he stated that a transplanted tooth would exfoliate within two to three years because of resorption. However, advances and understanding at that time were making it possible to maintain a transplanted tooth. Grossman discussed the indications of tooth replantation in 1966. They include complex anatomy, extruded materials, or to repair a perforation when apical surgery is not feasible.[34]

In 1964, endodontics was recognized as a specialty in the United States and multiple universities established training programs. The surgical endodontics chapter became an integral part of endodontic textbooks. Various aspects of surgical endodontics such as indications, contraindications, anesthesia, flap designs, root-end resection and preparation, root-end filling materials, and outcomes were reported.

The first modern endodontic textbook on surgical endodontics was published by Arens, Adams, and DeCastro,[35] followed by other textbooks in 1998.[36–39]

The results of scientific investigation and the clinical application of the techniques and concepts developed during the second half of the 20th century represent the foundation of what is known and are being practiced in the 21st century. However, surgical endodontics is dynamic and it is imperative that scientific investigation continues; concepts, techniques, and materials used in "microsurgical endodontics" must be continually evaluated, modified and more emphasis must be placed on the assessment of long-term clinical outcomes with a better understanding of the molecular basis for surgical wound healing.[40]

Surgical endodontic procedures consist of fistulative surgery (incision and drainage, cortical trephination, and decompression procedures), periapical surgery and corrective surgery (perforation repair, management of periodontal defects by root resection, hemisection and root extraction, bicuspidization) as well as tooth replantation and transplantation.

INCISION AND DRAINAGE

The objective of incision and drainage is to evacuate exudates and purulence from a soft tissue swelling. The objective of incision for drainage is to evacuate inflammatory exudates and purulence from the soft tissue swelling and reduce discomfort from the buildup of pressure. This procedure not only results in patient's comfort, but it also speeds healing and recovery.

Indications

The best treatment for swelling originating from an acute apical abscess of pulpal origin is the establishment of drainage through the offending tooth (Figure 24-4). When adequate drainage is not accomplished through the tooth itself, drainage through the soft tissue incision should be attempted. Occasionally drainage is performed through the soft tissue without obtaining drainage through the offending tooth. The reason for this is the presence of separate, non-communicating abscesses to the apices of the offending tooth. Drainage through the soft tissue is accomplished most effectively when the swelling is fluctuant (Figure 24-5).

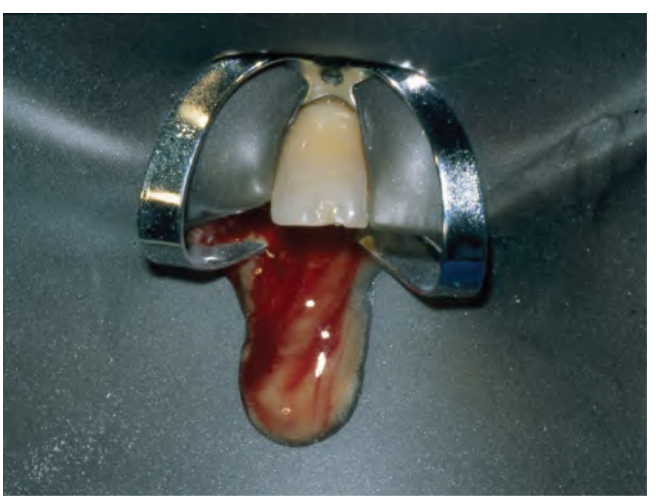

FIGURE 24-4 Establishment of drainage through the offending tooth with an acute apical abscess is the first step in treating these conditions.

A fluctuant swelling is a fluid-containing mass in which a wave-like sensation is felt when pressure is applied. Incisinga fluctuant swelling releases purulence immediately and provides rapid relief. If the swelling is non-fluctuant or firm, incision for drainage often results in drainage of only blood and serous fluids. Incision and drainage of a non-fluctuant abscess reduce pressure and facilitates healing by reducing irritants and increasing circulation in the area.

Contraindications

There are very few contraindications for the incision for drainage. Patients with bleeding disorders must be approached with caution. Hematological screening is indicated for these patients. Anatomic landmarks such as the mental foramen, maxillary sinus, incisive foramen, and an inferior alveolar canal near the apical abscess should be approached carefully during incision and drainage.

Procedures

Anesthesia

Obtaining profound anesthesia is difficult when severe inflammation and swelling are present. Because achieving infiltration anesthesia is difficult and painful in some cases, regional block anesthetic techniques such as mandibular blocks for posterior areas, bilateral mental blocks for the anterior mandible, posterior superior alveolar blocks for the posterior maxilla, and infraorbital blocks for the premaxillary area should be attempted.

After the application of topical anesthetic, the anesthetic solution should be injected slowly with limited pressure away from the center of the abscess. This injection is then followed by additional injections in previously anesthetized tissue, moving progressively closer to the center of the swelling. Using this technique usually results in improved anesthesia without causing extreme pain or discomfort for the patient.

Use of topical ethyl chloride has been suggested to reduce pain during injection, incision, and drainage of acute apical abscesses.[41] A stream of ethyl chloride liquid solution is directed onto the swelling from a distance, permitting the liquid to volatilize on the tissue surface. Within seconds, the tissue at the site of application becomes white and numb, allowing the operator to perform anesthesia, incision, and drainage. When none of these procedures provide adequate anesthesia, nitrous oxide sedation, oxygen sedation, or intravenous (IV) sedation should be considered as adjunctive treatments for obtaining adequate anesthesi and maximum patient comfort.

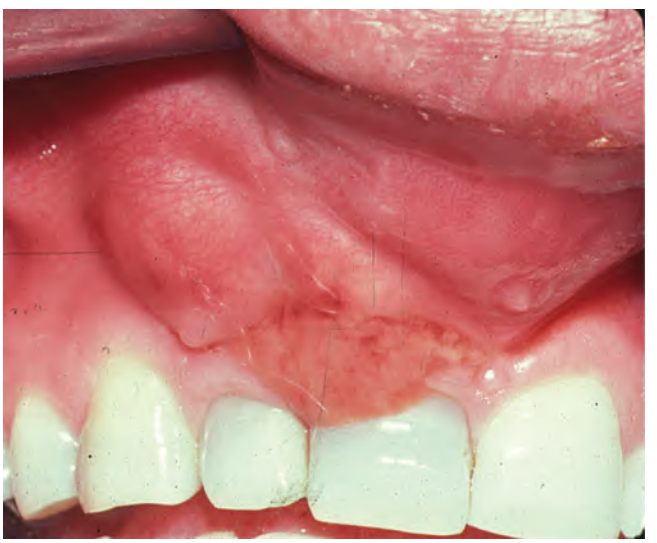

FIGURE 24-5 A fluctuant swelling in a tooth with an acute apical abscess.

Incision

After obtaining adequate anesthesia, the incision is made vertically with a No. 11 scalpel at the height of swelling (Figure 24-6A and B). A vertical incision should be made firmly through the periodontium to the underlying bone. If the swelling is fluctuant, drainage is usually obtained immediately. The drainage starts with pus followed by blood. If the swelling is nonfluctuant, the drainage is only blood.

Drainage

After making an incision, a small hemostat can be placed in the incision line to enlarge the draining tract.[37] To maintain a path for drainage, a drain cut from a dental dam can be sutured in the incision line (Figure 24-6C). The drain should be removed two to three days after the procedure.

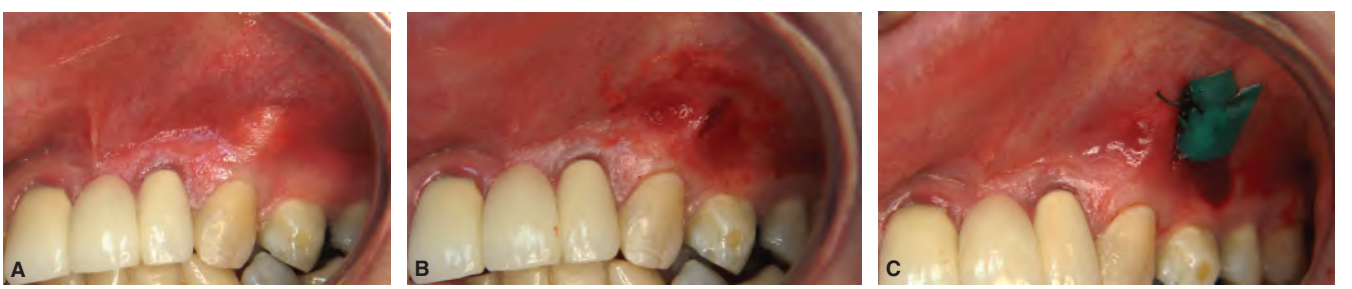

FIGURE 24-6 ***A.*** A fluctuant swelling caused by an acute abscess originating from the maxillary left canine. ***B.*** A vertical incision is made. ***C.*** A drain is sutured in the incision line to maintain a path for drainage for a few days after I&D.

PERIAPICAL SURGERY

The purpose of performing periapical surgery (PAS) is to stop egress of irritants from the root canal system into the periapical tissues. This is accomplished by gaining access to the root end or any part of it and sealing the portals of exit with a biocompatible material that allows complete regeneration of periapical tissues.

Indications

The indications for periapical surgery have undergone dramatic changes in the last two decades.[40] These changes have been evident, especially when dealing with the treatment of failed nonsurgical endodontic treatments. The primary modality for endodontic treatment failure should be non-surgical endodontic retreatment whenever possible. The importance of thorough and meticulous pre-surgical planning cannot be overemphasized. Not only must the practitioner and staff be thoroughly trained, but also all necessary instruments, equipment, and supplies must be readily available in the treatment room. This requires that every step of the procedure be carefully planned and analyzed. The potential for possible complications must be anticipated and incorporated into the presurgical planning. Good patient communication is essential for thorough surgical preparation. It is important that the patient understands the reason surgery is needed as well as other treatment options available. The patient must be informed of the prognosis for a successful outcome and the risks involved in the surgical procedure in addition to the benefits. It is also important that the patient is informed of the possible short-term effects of the surgery such as pain, swelling, discoloration, and infection. Signed consent forms are essential.

The indications for PAS are the anatomical complexities of the root canal system that prevent complete cleaning, shaping, and obturation of the root canal system through the coronal access, symptomatic cases, irretrievable materials in the root canal, procedural accidents, and exploratory surgery.

Anatomical Complexity of the Root Canal System

These complexities include unusual canal anatomies such as severe curvature, complex anatomy, and canal calcifications (Figure 24-7).

Irretrievable Filling Materials

Most filling materials can be removed from the roots through a coronal access of a tooth. However, there are situations when obturating materials or posts cannot be removed without jeopardizing the whole tooth (Figure 24-8). Insistence on removing every filling material at the expense of weakening the root is a contraindication for nonsurgical retreatment.

Persistent Symptoms

Performing endodontic treatment usually results in relief of pain and symptoms in most patients. However, there are cases when they remain symptomatic after cleaning and shaping or following obturation. These are indications for surgical retreatment (Figure 24-9).

Procedural Accidents

Like other complex disciplines of dentistry, performing root canal treatment can result in unwanted or unforeseen challenges that are collectively termed procedural accidents. Examples of these mishaps include root perforation, ledge formation, separated instruments, and underfilled or overfilled canals. Most of these accidents can be corrected nonsurgically as described in Chapter 23, "Endodontic orthograde retreatment and management of mishaps." When unfeasible or impractical, periapical surgery is indicated to retain these teeth (Figure 24-10).

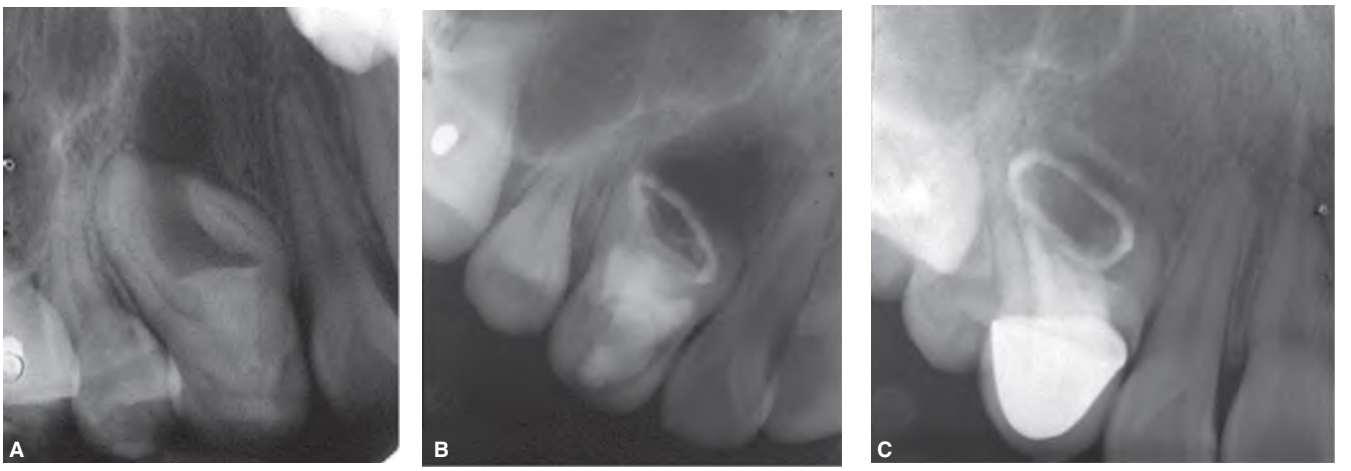

FIGURE 24-7 ***A.*** Preoperative radiograph of the right region shows presence of dens invaginatus in the canine. ***B.*** Because of the presence of this anomaly in the tooth and a large lesion, in addition to the inability to perform nonsurgical root canal treatment, periapical surgery was performed. ***C.*** Postoperative radiograph taken 20 months later shows complete resolution of the lesion associated with the canine.

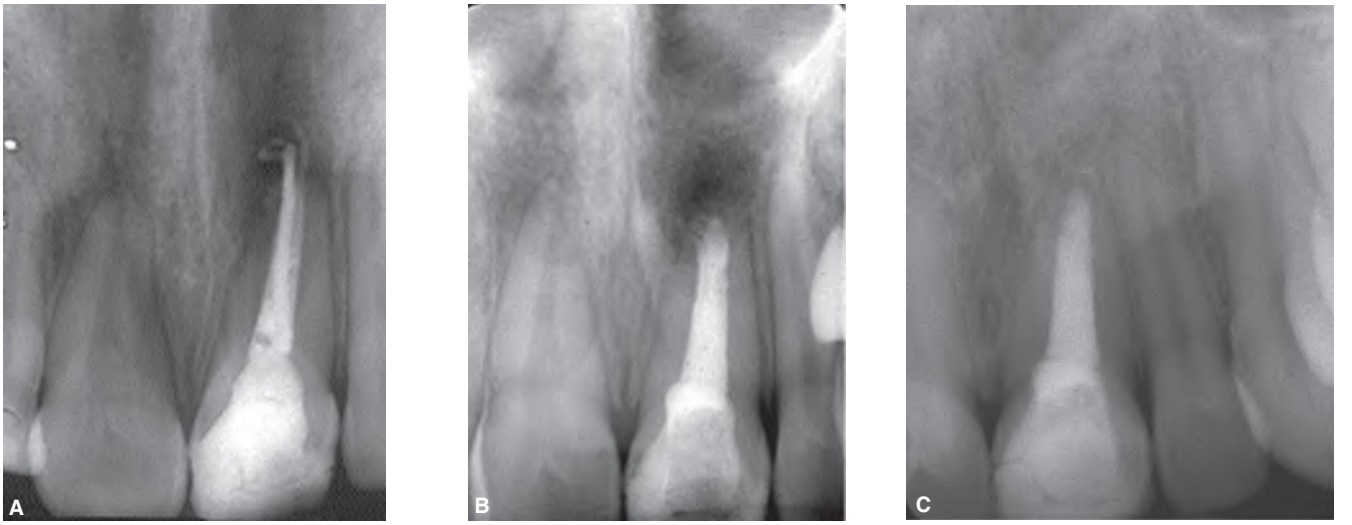

FIGURE 24-8 ***A.*** Preoperative radiograph of a maxillary central incisor shows presence of gutta-percha segments in the bone. ***B.*** Because of the patient's discomfort and pain, periapical surgery was performed on this tooth. ***C.*** Follow-up radiograph taken nine years later shows complete resolution of the lesion.

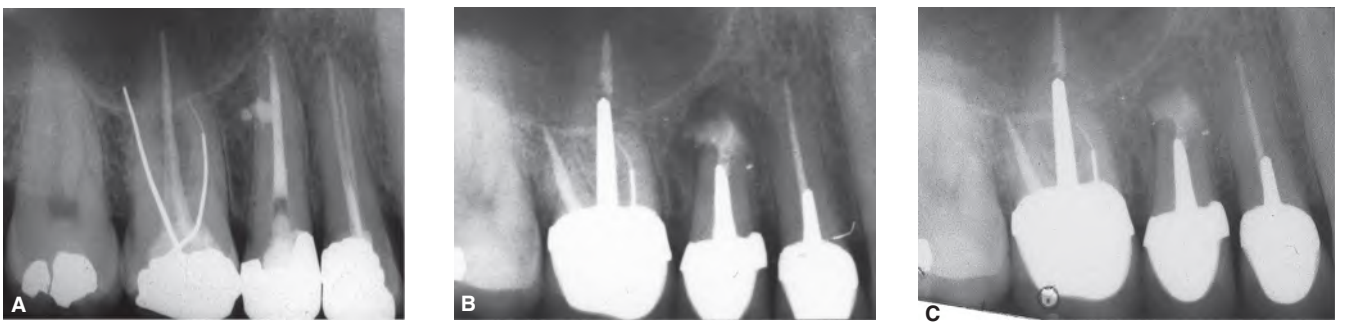

FIGURE 24-9 ***A.*** Preoperative radiograph of a maxillary second premolar shows presence of gutta-percha and extrusion of root canal sealer into the periapical tissues of this tooth. ***B.*** Because of the patient's discomfort and pain, periapical surgery was performed on this tooth. ***C.*** Radiograph taken nine months later shows complete resolution of the lesion. (Courtesy of Dr. Ahmad Fahid, Beverly Hills, California, U.S.A.)

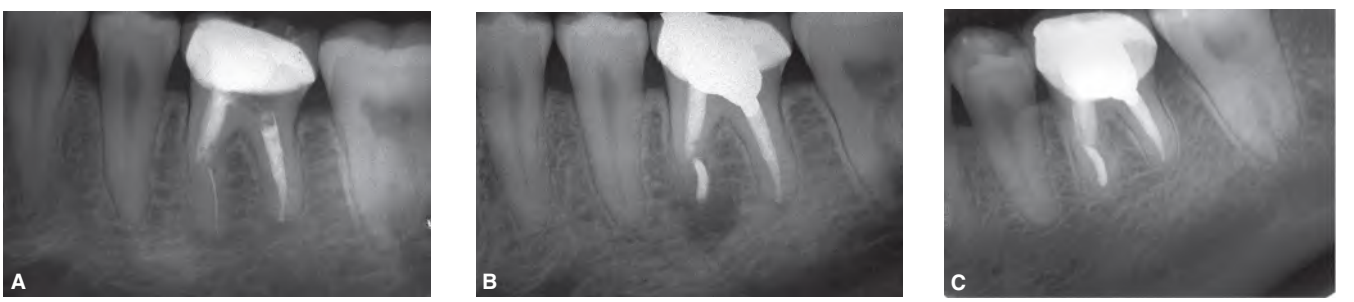

FIGURE 24-10 ***A.*** Nickel-titanium file is separated inside the mesiobuccal canal of the mandibular first molar. ***B.*** Because of patient discomfort, surgery was performed to remove this instrument surgically. MTA was used as a root-end filling material. ***C.*** Periapical radiograph taken 32 months later shows complete healing.

EXPLORATORY SURGERY

Studies have shown that the majority of periapical lesions are caused by infection of root canal systems.[42] However, there are some radiolucencies that are not caused by root canal infection. If suspected, exploratory surgery is indicated to determine the cause of these pathological entities (Figure 24-11).

Contraindications

There are very few contraindications for PAS. They include (1) medical or systemic complications, (2) indiscriminate use

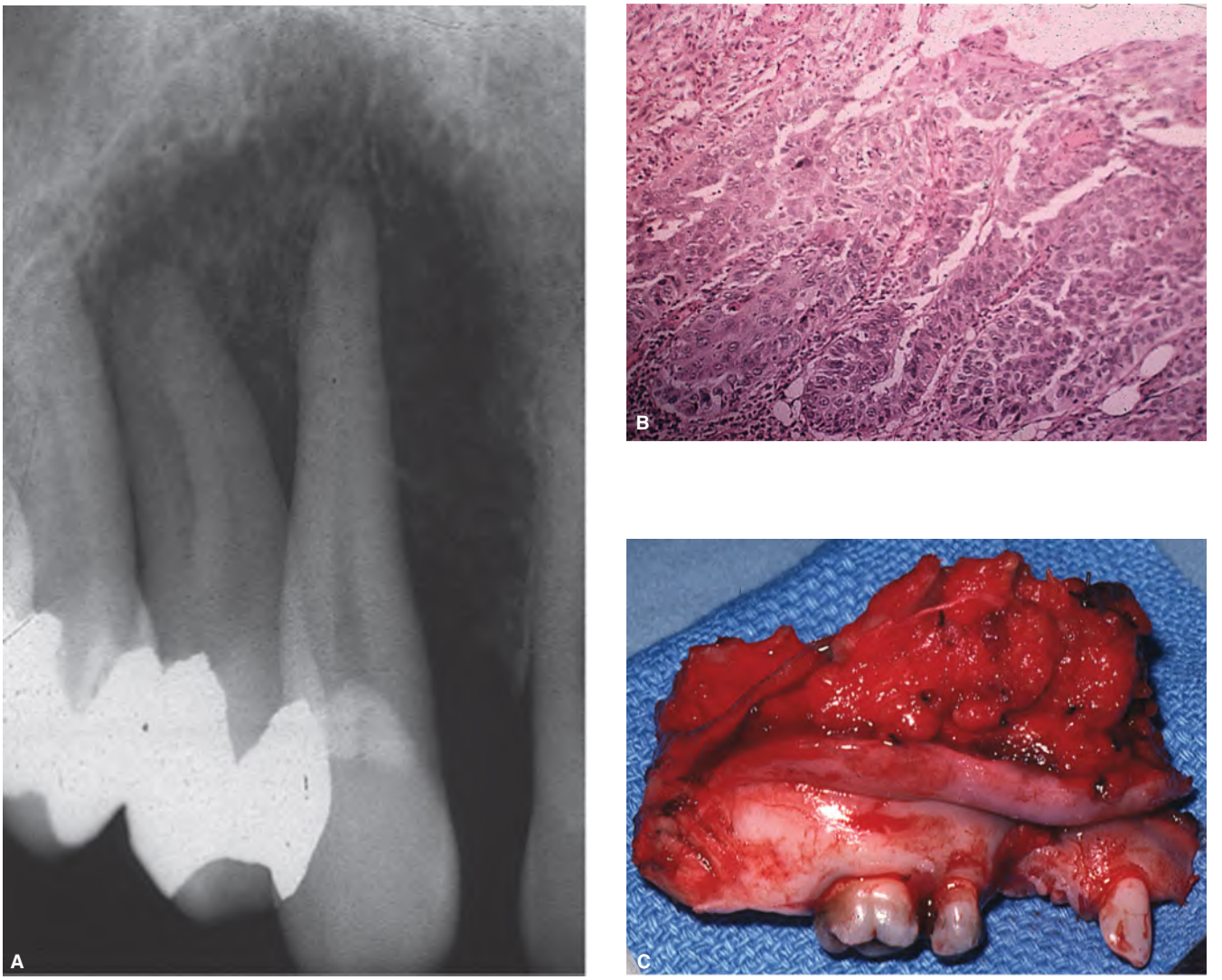

FIGURE 24-11 ***A.*** Periapical radiolucent lesion of nonpulpal origin. ***B.*** The pulp responded normally to pulp sensibility tests. Histologic examination of the tissue confirmed a diagnosis of carcinoma. ***C.*** The maxilla was completely removed to save this patient.

of periapical surgery, (3) anatomic factors, and (4) an unidentified cause of treatment failure.[37]

Medical or Systemic Complications

Serious systemic diseases in patients with blood disorders, terminal diseases, uncontrolled diabetes, severe heart diseases, and compromised immune systems are contraindications for PAS. For more details, see Chapter 31, "Management considerations for the medically complex endodontic patient."

Indiscriminate Use of Surgery

Periapical surgery is not indicated when a nonsurgical approach is feasible and would probably result in successful outcomes. Indiscriminate use of PAS for those cases that can be handled with a nonsurgical approach is unethical and contraindicated (Figure 24-12). A systematic review comparing treatment outcome for surgical endodontics versus

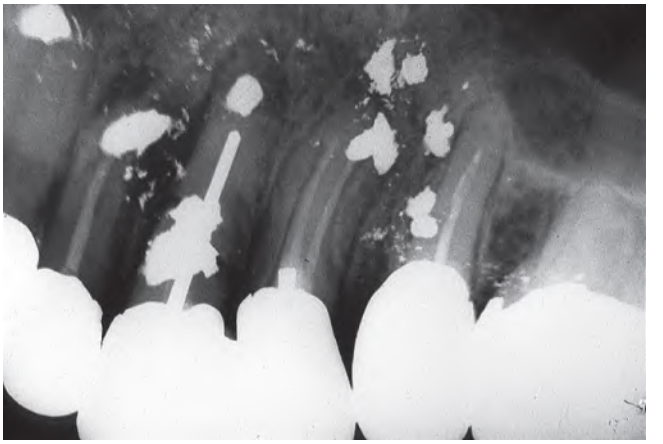

FIGURE 24-12 Indiscriminate use of periapical surgery in patients is unethical and is one of the contraindications of this procedure.

nonsurgical retreatment showed an initial higher success rate for surgery at two to four years, but a significant shift in favor of nonsurgical retreatment at four to six years.[7] This finding supports the concept that nonsurgical retreatment should be the first choice before attempting PAS.

Anatomic Factors

Anatomical structures that may be of importance during surgical endodontics include the neurovascular bundle associated with the greater palatine foramen, the mandibular canal and the mental foramen, the floor of the nose, the maxillary sinus, and any other anatomical structures that limit or compromise visualization and access to the surgical site.[40] During surgical procedures on maxillary incisors, the floor of the nose will infrequently be encountered unless the roots of the incisors are extremely long or the periapical lesion extends superiorly and has eroded through the bony floor of the nose. The greater palatine foramen (Figure 24-13) is most frequently located between the maxillary second and third molars in a position approximately one centimeter from the margin of the palatal gingiva toward the midline of the palate. The greater palatine foramen and its contents generally do not pose a problem because so few apical surgical procedures are performed on the palatal root of maxillary second and third molars.

Exposure of the maxillary sinus may occur when apical surgical procedures are performed on the roots of any of the teeth located in the maxillary posterior quadrants. Eberhardt et al.[43] reported that the apex of the mesiobuccal root of the maxillary second molar is closest to the floor of the maxillary sinus (mean = 0.83 mm), and that the apex of the buccal root of the maxillary first premolar is furthest away (mean = 7.05 mm). The root apices of the maxillary second premolar and the mesiobuccal and distobuccal root of the maxillary first molar were all approximately 2.8 mm from the floor of the sinus. Twelve autopsy specimens and 38 human patients were subjected to computed tomography to arrive at these means.

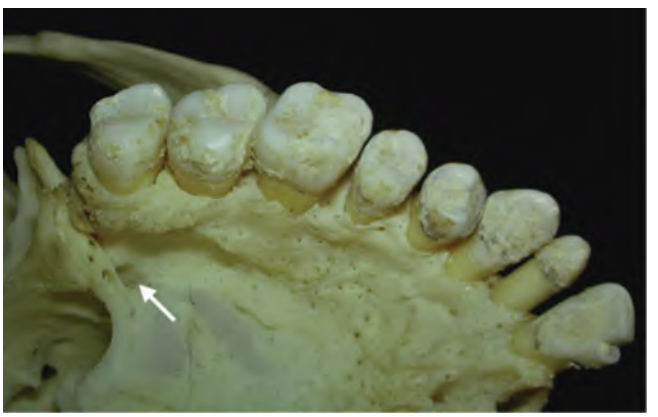

FIGURE 24-13 Opening of greater palatine foramen (arrow). The neurovascular bundle courses in posterior portion of the palate.

In a clinical study, Ericson et al.[44] noted in 314 apical surgical procedures performed on maxillary canines, premolars, and molars in 276 patients that oroantral communication occurred in 41 cases (13%). Sinus communication did not impair healing, as there was no difference in the success rate between those cases with or without sinus exposure. In another clinical study by Rud and Rud,[45] a sinus perforation occurred in 50% of 200 maxillary first molars treated with an apical surgical procedure. Of the cases with a sinus exposure, only two developed postoperative sinusitis.

When performing surgical procedures on teeth in situations where the apical lesion has not penetrated the cortical plate, the surgeon should be aware of the mean thickness of bone that must be penetrated to reach the root apices.

Jin et al.[46] calculated the mean distance from the root apices to the buccal or palatal cortical plate for the root or roots of every tooth group except third molars. One thousand eight hundred and six teeth were evaluated using computed tomography in 66 Asian patients. The greatest mean distance between a root apex and the buccal cortical plate reported in the two studies was for the distal root of the mandibular second molar with a mean distance of 8.51 mm. The tooth with the root apex closest to the buccal cortical plate was the maxillary canine (mean = 1.64 mm) in one study and the buccal root of the maxillary first premolar (mean = 1.63 mm) in the second study.

When performing apical surgery on mandibular premolars and molars, it is not only important to know the location of the root apices in relationship to the buccal cortical plate, but also to note the relationship of the root apices to the mandibular canal. The distal root of the mandibular second molar is located furthest from the buccal cortical plate and as one progresses forward in the arch, the root apices of each succeeding tooth become progressively closer to the buccal cortical plate.[46,47] The mandibular canal follows an "S-shaped" pathway as it moves from a position inferior and buccal to the distal root apex of the mandibular second molar to a more lingual position inferior to the mesial root apex of the mandibular second molar and the apices of both roots of the mandibular first molar. The canal then crosses back to a more buccal position beneath the apex of the mandibular second premolar.[48] Denio et al.[49] found that the root apices of the mandibular second molar (mean = 3.7 mm) and the mandibular second premolar (mean = 4.7 mm) were closest to the mandibular canal. The mesial root apex of the mandibular first molar was found to be furthest from the canal (mean = 6.9 mm). Similar results were noted in the study by Littner et al.[48]

The anatomy and location of the mental foramen have been studied by multiple investigators. The mental foramen may be located anywhere between the apex of the mandibular first premolar and the mesial root of the mandibular first molar. In two reports[50,51] Tebo and Telford as well as Tebo where 100 adult mandibles were studied, the most frequent

location was apical to the mandibular second premolar (Figure 24-14). This was also the most frequent location in another study that looked at 75 dry mandibles.[52] In a study of 105 cadaver specimens, the most frequent location was found to be between the apices of the mandibular first and second premolars.[53] In another study[54] that reviewed 1,000 full-mouth series of radiographs, 70% were found to be located between the apices of the two premolars. It is obvious that the mental foramen can be quite variable in location for different individuals.

Phillips et al.[52] compared the size of the mental foramen as measured on dry mandibles with the size measured on periapical and panoramic radiographs. The mean horizontal measurement on the 75 dry mandibles was 4.6 mm and the mean vertical measurement was 3.4 mm. The periapical radiographic measurement was smaller with a mean of 2.6 mm for the horizontal and 2.3 mm for the vertical. The measurements from the panoramic film were slightly larger than those obtained from the periapical radiographs (horizontal mean = 2.9 mm and vertical mean = 2.5 mm). In two-thirds of the 75 dry specimens, the foramen was found to exit in a posterior and superior direction.

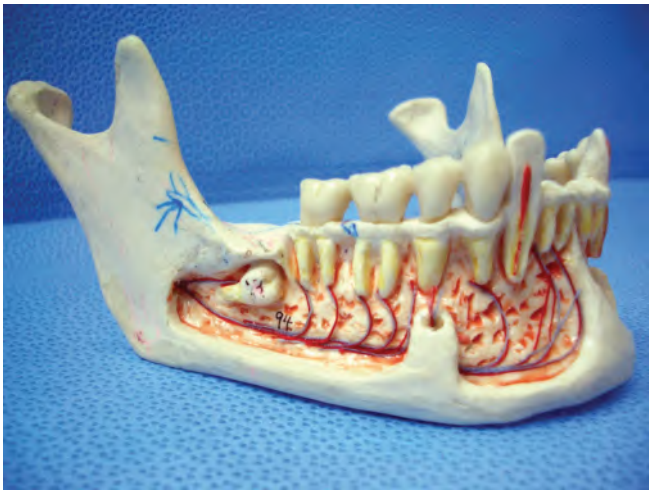

FIGURE 24-14 The mental foramen is usually located inferior to the apex of the root of the mandibular second premolar.

When performing apical surgery on a root containing more than one root canal system, the surgeon must be aware that an isthmus may exist between the canals. Weller et al.[55] were among the first to point out the significance of the isthmus in surgical endodontics. In mesiobuccal roots of the maxillary first molar with two root canal systems, they reported the highest incidence of an isthmus occurred in the apical 3 to 5 mm, and that an isthmus was present 100% of the time at the 4-mm level. In a more recent clinical study by von Arx,[56] an isthmus was found in 76% of mesiobuccal roots of maxillary first molars with a 3 to 4 mm apical root resection. In a microcomputed tomographic study by Mannocci et al.,[57] an isthmus was found to be present 85% of the time in the mesial root of the mandibular first molars evaluated. This incidence of isthmuses in the mesial root of mandibular first molars is very close to the 83% reported in a clinical surgical study.[56] In von Arx's clinical study, an isthmus was found 36% of the time when the distal root of mandibular first molars presented with two canals.[56]

Proximity to the neurovascular bundles in the mandibular posterior teeth can be, in some cases, a contraindication for PAS. These cases require caution and skill to handle surgically (Figure 24-15). The presence of a thick external oblique ridge associated with a mandibular molar or apices contiguous with the mandibular canal can compromise surgical access. These cases are indications for other surgical procedures such as tooth replantation, transplantation, or extraction with or without replacement.

Unidentified Cause of Treatment Failure

Using PAS to correct a failed nonsurgical case without a clear cause is a contraindication for this procedure.

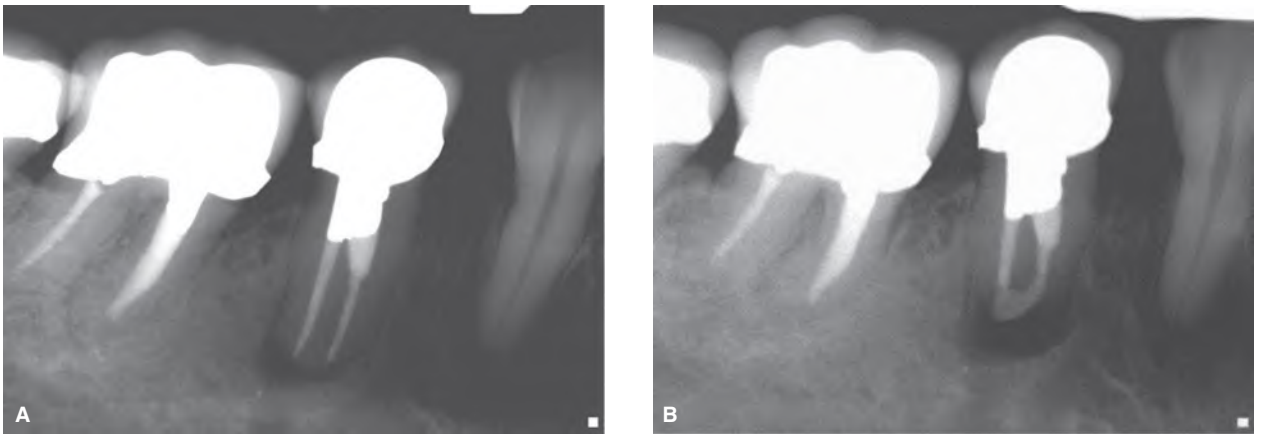

FIGURE 24-15 ***A.*** Proximity of the apex of the mandibular first premolar to the neurovascular bundle can be a contraindication for periapical surgery, and ***B.*** It requires caution and skill if surgical endodontics is performed. (Courtesy of Dr. Tory Silvestrin, Loma Linda, California, U.S.A.)

RECENT ADVANCES IN SURGICAL ENDODONTICS

A number of advances in PAS techniques, instruments, and materials have occurred within the last two decades.[58] These include enhanced magnification and illumination, ultrasonic tips, micro-instruments, and newer root-end filling materials. Enhanced magnification and illumination have greatly improved the ability of the operator to perform PAS more efficiently and predictably. Using magnification in PAS has led to the miniaturization of endodontic surgical instruments (Figure 24-16). Development of biocompatible root-end filling materials with better sealing ability such as MTA has increased the predictability of PAS. The cone beam computed tomography (CBCT) in PAS provides high-resolution images in three dimensions. The use of CBCT eliminates superimposition of surrounding anatomical structures for better diagnosis and surgical approach.[59] These advances have significantly improved the art and science of PAS and have increased the feasibility and predictability of this procedure to save natural dentition.

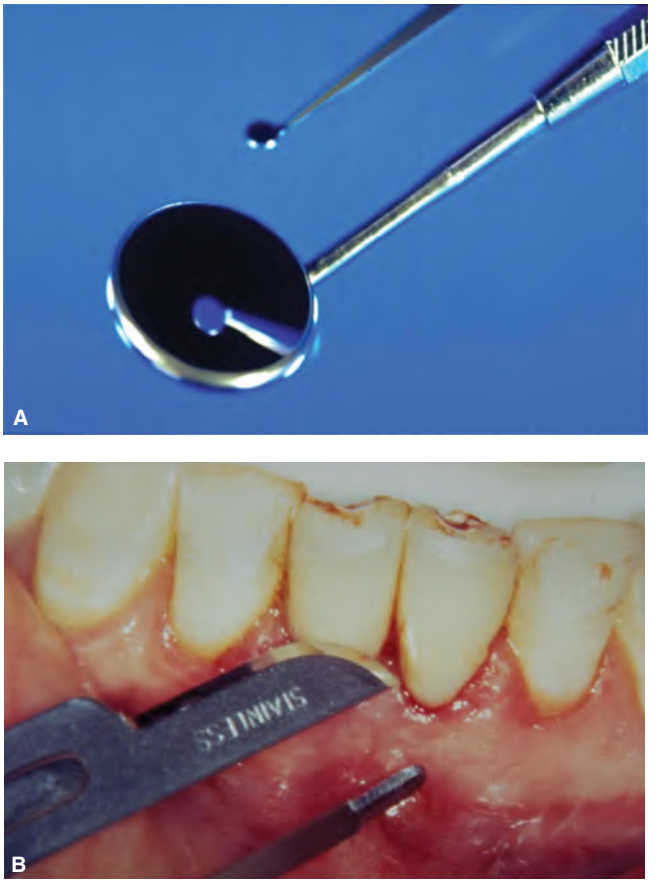

FIGURE 24-16 ***A.*** Comparison of standard intraoral mirror and micro mirror, as well as ***B.*** A 15C blade and a micro blade used for endodontic microsurgery.

PROCEDURES INVOLVED IN PERIAPICAL SURGERY

The procedures involved in PAS are anesthesia, homeostasis, flap design, incision and reflection, apical access, periapical curettage, root-end resection, root-end cavity preparation, root-end filling, flap replacement and suturing, post-operative care as well as instructions, and suture removal and evaluation.

Anesthesia

Giving local anesthesia during PAS has three purposes. They include (1) complete comfort for the patient, (2) hemostasis during the surgical procedure, and (3) prolonged pain control following surgery. Achieving profound local anesthesia and establishing effective hemostasis depend on the selection of local anesthetic solution and proper use of vasoconstrictor medications, utilizing proper injection technique, rate of injection, and the use of supplemental injection when needed. Proper use of local anesthetic with a hemostatic agent not only causes patient comfort, but also improves the ability of the operator to perform PAS more effectively.[45] For more details, see Chapter 18, "Management of pain, fear, and anxiety in the endodontic patient."

Local Anesthetic in Surgical Endodontics

Based on their chemical structures, local anesthetic medications are classified as esters or amides. The esters include Procaine, Tetracaine, Propoxycaine, and Chloroprocaine. The amides include local anesthetics such as Lidocaine, Mepivacaine, Prilocaine, and Etidocaine. Lidocaine is currently the most commonly used local anesthetic. The reasons for its popularity are (1) its rapid onset, (2) its production of profound anesthesia, (3) its prolonged action, (4) its low toxicity and antigenicity, (5) its high diffusion rate, and (6) its different concentrations of vasoconstrictors.

Choice of Vasoconstrictor

The type and concentration of vasoconstrictor in the local anesthetic have an effect on both the duration of anesthesia and the quality of homeostasis at the surgical site. Vasoconstrictor agents used in dentistry include epinephrine, levonordefrin, and norepinephrine.[60] Because the use of norepinephrine causes marked tissue ischemia, it should not be used in surgical endodontics for the purposes of hemostasis.[60] Epinephrine is the most effective and most widely used vasoconstrictor material in dental anesthetics.[61] Infiltration injection of 1.8 ml of 2% lidocaine with 1:100,000 epinephrine produces about a threefold increase in the plasma concentration of epinephrine with little or no significant systemic cardiovascular effects. It appears that cardiac effects of local anesthetics containing epinephrine are minimal and that they can be safely used in stable cardiovascular disease patients.

A randomized double-blind controlled clinical trial reported that homeostasis was judged significantly better in

1:100,000 epinephrine containing local anesthetics compared to 1:200,000 epinephrine containing local anesthetics.[62] Buckley et al. demonstrated that the use of lidocaine containing 1:50,000 epinephrine produced more than a 50% improvement in homeostasis compared to 2% lidocaine containing 1:100,000 epinephrine in patients undergoing periodontal surgery.[63] The use of a local anesthetic containing 1:50,000 epinephrine has been advocated for local infiltration in the surgical field. Clinical trials indicate that injection of local anesthetic containing 1:50,000 epinephrine produces a transient tachycardia that returns to normal within 4 minutes.[64]

Systemic Effects of Vasoconstrictors in Local Anesthetics

Because a high concentration of vasoconstrictor containing anesthetic such as 1:50,000 epinephrine is preferred to obtain effective vasoconstriction for lasting homeostasis (Figure 24-17), there is a concern related to its effects on systemic circulation.[58,65,66] Although the systemic effects such as pulse rate and blood pressure are minimal in response to the amount of epinephrine used in surgical procedures, one study clearly demonstrates that the plasma level of epinephrine is elevated when a high dose of epinephrine is utilized.[66] It appears that virtually all of the adverse effects associated with epinephrine are dose-dependent.[58] The cardiovascular effects are usually minimal and short-lived and are well-tolerated by the majority of patients except in patients with severe cardiovascular disorders or patients who have had cardiovascular surgery. The use of 1:50,000 epinephrine with 2% lidocaine is recommended for local anesthesia in the majority of cases of PAS.[58] The selection of an appropriate anesthetic agent and concentration of vasopressor should always be based on the medical status of the patient and the discretion of the operator. Consultation with physicians is recommended in patients with moderate to severe medical conditions. These recommendations should be obtained in writing and documented in the patient records.

Flap Design, Soft Tissue Elevation, and Tissue Retraction

Comprehensive knowledge of head and neck anatomy is essential when planning for a PAS. A thorough understanding of head and neck anatomy is necessary to avoid unwanted complications during surgical endodontics. Knowledge related to the anatomy of soft tissue and bony landmarks can assist the operator to reduce the risk for postoperative morbidity. Periapical, panoramic, and CBCT radiographs can provide evidence of important hard tissue structures. However, the details of soft tissues are not seen by traditional radiography. Serman[67] described the course of the inferior alveolar canal once it emerged from the mental foramen in cases where two foramens are present, which requires greater attention from the clinician. Naitoh et al.[68] studied the presence of the accessory mental foramina using CBCT and found it to be present in 7% of the population. There are fewer anatomic considerations when maxillary teeth require PAS. However, when a palatal approach is considered, the location of greater palatine foramen where the greater palatine artery and nerve pass through should be considered very carefully. Severing the palatal artery is life-threatening and should be avoided. Tomaszewska[69] performed a morphometric analysis of the routes of the greater palatine canal and a systematic review of the literature related to the anatomy of this canal. The results indicate that the greater palatine foramen has four different opening directions: inferior-anterior-medial (82.1%), inferior-anterior-lateral (4.0%), anterior (7.6%), and vertical (5.3%). Yu et al.,[70] using a surgical microscope, reported the distance from the cementoenamel junction (CEJ) to the branch of greater palatine artery to be 9.04 ± 2.93 mm (canine), 11.12 ± 1.89 mm (first premolar), 13.51 ± 2.08 mm (second premolar), 13.76 ± 2.86 mm (first molar), and 13.91 ± 2.20 mm (second molar).

Muscle attachments to the jaw bones can pose problems during surgical flap dissection. If attachment levels are close to the apex of a tooth or if the tooth is very long, full reflection may be difficult. A shallow vestibule may also

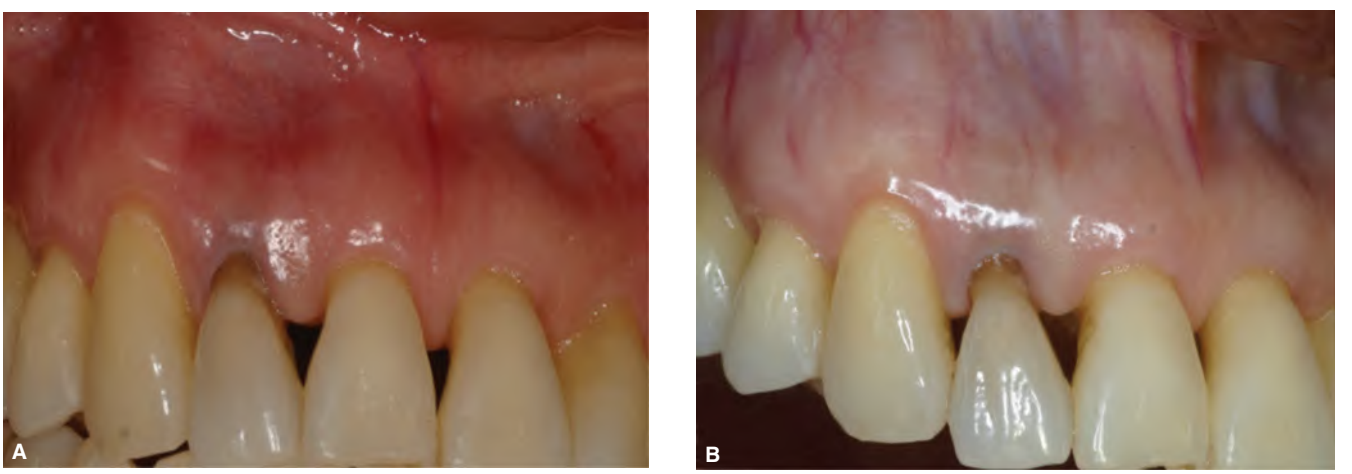

FIGURE 24-17 ***A.*** Gingival tissues before injection of a local anesthetic. ***B.*** Gingival tissues after injection of a local anesthetic containing 1:50,000 epinephrine.

complicate reflection and therefore limit surgical access.[71] It is important to predict and be prepared for these challenges prior to beginning PAS.

Prior to initiating an incision, the surgeon should evaluate clinically and radiographically the location and size of the periapical lesion and the number of the teeth needed to be included in the field of operation. After initial incision and during flap reflection, there are important principles to follow for achieving the best healing and reducing postsurgical patient morbidity. An operating microscope can be used during incision and reflection to allow for better handling of soft tissue.[72] The use of a dental operating microscope will provide superior illumination as well as magnification to aid the operator during endodontic microsurgery.[72]

The factors that influence the healing of the mucoperiosteal tissues during surgical endodontics include flap design, tissue handling, wound closure, and post-surgical care. Here is a list of surgical do's and don'ts during flap design and reflection:

Don't place incisions over a bony defect. A suture during primary closure should be placed over sound bone structure whenever possible.[71–73] Five millimeters of space should separate the bony defect from the incision line.[74]

Don't place an incision over the root eminence. Root eminences, as well as the flutes between these protrusions, can be visualized and palpated before the first incision is made.[73]

Don't traumatize the flap during retraction. The flap will have a tendency to slip free from the retractor. Pinching the flap with the retractor should be avoided. Any unnecessary trauma to the soft tissue should be avoided to decrease postoperative morbidity. The vascular bed within the flap[71,75] can be damaged by the sharp edges of instruments.

Do create a passive or tension-free flap during wound closure. Larger flaps may be required to achieve a tension-free flap.[76] Keep soft tissues hydrated during PAS. Soft tissue during surgery should not be allowed to dehydrate. A dry flap runs the risk of shrinkage.[77] Evaluate soft tissue morphology, smile line, and periodontium before making any incision.

*E*valuate hard tissue structures before incising the soft tissues. Try to use a single pass to incise the soft tissue to the desired depth. Create incision edges that are consistently perpendicular to the tissue's surface. Secure the suture knot away from the incision line to prevent delayed healing.

Flap Design

Flap design is one of the most important steps in PAS. There are many different flap designs available to choose for apical surgery. The proper selection can make a significant impact on the outcomes of the surgery. The primary goal of raising a flap is to provide access to the surgical site while considering the aesthetics of the soft tissue. The surgeon should evaluate the surgical area clinically and radiographically to determine the size of the lesion, the number of teeth to be included, and the closure of the surgical wound. If the lesion involves more than one tooth, or a larger lesion is expected, the flap should be designed accordingly. A small flap makes retraction difficult, more traumatic, and can delay healing. Large flaps heal as well as small flaps.

A larger flap is more desirable than a smaller flap since the larger flap prevents tear and trauma to the flap and provides more access and visibility to the surgical site. A vertical incision is usually required for PAS. It provides better access and visibility during surgery. Vertical incisions should always be placed at the line angles, and the papillae should be included in the flap for wound closure.

Aesthetic consideration during flap design is an important part of today's surgical endodontics. All treatment planning must have a proven scientific basis to provide successful outcomes. A review of current endodontic literature reveals disagreement related to the indications and contraindications for various flap designs. In the past, different flap designs were introduced in order to gain access to the surgical site. Yet very few of these designs took aesthetics into consideration, especially in such aesthetically sensitive areas as the maxillary anterior region. Crestal bone loss and gingival recession are detrimental to gingival health and aesthetics (Figure 24-18). Human studies have shown an average of 0.5 to 0.62 mm crestal bone loss after raising a full thickness flap.[78–82] Harrison and Jurosky,[77] as well as Chindia and Valdehaug,[83] did not find any difference in the healing of intrasulcular and submarginal incisions. Since Chindia and Valdehaug[83] measured the clinical attachment level (CAL) preoperatively and did not report CAL postoperatively and only referred to changes in pocket depths, the results of their study are questionable. The results of these two studies differ considerably from those of Kramper et al.[84] who clinically and histologically evaluated the healing of three common surgical flap designs. They found that intrasulcular wounds showed no epithelial closure at two days post-operation and continued to show persistent inflammation up to 156 days later.[84] More importantly, these authors reported the loss of

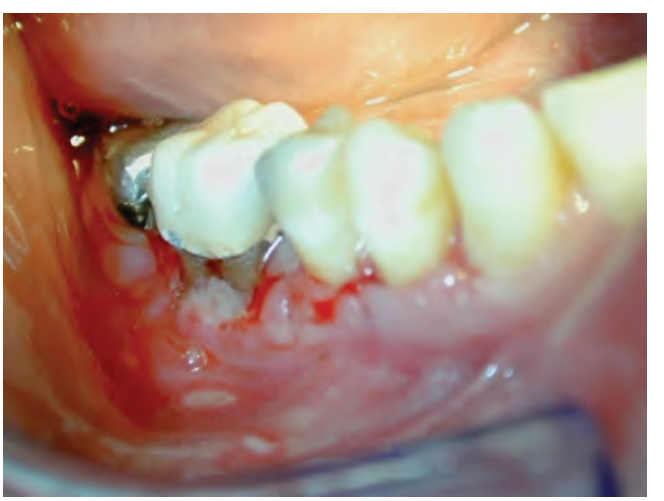

FIGURE 24-18 Severe crestal bone loss and gingival recession after a periapical surgery.

alveolar bone of approximately 0.5 to 1 mm with intrasulcular incisions. Grung[82] also found gingival recession of 0.5 mm (on average) three months after raising full mucoperiosteal flaps.

Current surgical techniques used in PAS have been mimicking the results of periodontal flap surgery and wound healing. Use of such evidence may be misleading when used for surgical endodontics. Wound healing in surgical endodontics is, by primary intention, different from periodontal surgical wound healing. The Luebke-Ochsenbein flap design may be used when sufficient attached gingival and keratinized mucosa exist (Figure 24-19). However, it cannot be used in areas where there is insufficient attached and keratinized gingiva, short roots, large lesions, sinus tracts coronal to the horizontal incision, or anatomical obstructions such as the frenum. The incision in this design can cross the osseous defects created during osteotomy that can result in an unpredictable outcome. In addition, sharp corners in this flap design where the vertical and horizontal incisions meet may result in necrosis and possible scarring of soft tissues. The following factors should be considered for flap designs during PAS:

- Tissue biotypes.
- Location of lip line.
- Presence of restorations, crown margins, and presence of a pontic.
- Access to the surgical site.
- Mucogingival consideration such as gingival recession, dehiscence, or fenestration.
- Periodontal considerations.
- Thickness of the soft tissue.
- Depth of vestibule, muscle attachment, and frenum location.

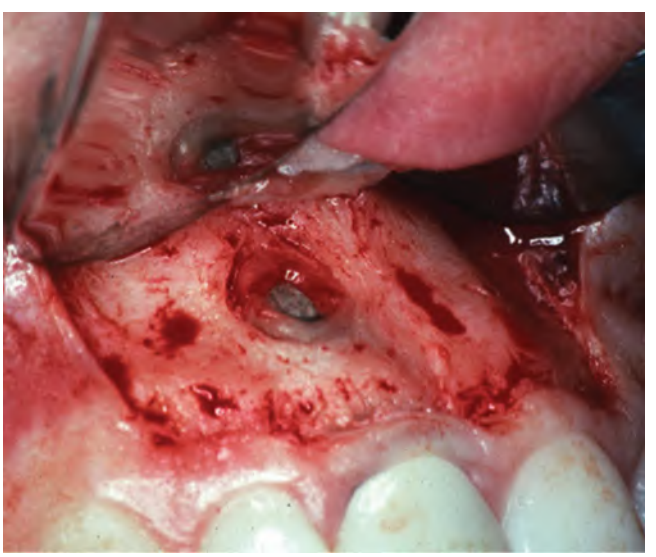

FIGURE 24-19 The Ochsenbein-Luebke flap design is used to prevent esthetic issues. It is made in the attached gingiva with two accompanying vertical incisions to reach the apex of the offending tooth.

- Location and extent of the lesion.
- Number of teeth involved in the surgery.
- Length of the involved tooth or root.
- Proximity to vital structures.

An important factor during evaluation for flap design is the determination of the tissue biotypes (Figure 24-20). The flap design is different for a thin biotype (thin triangular periodontium and papilla) versus a thick biotype (square tooth with thick and flat periodontium and wide papilla). Tooth shape, crestal bone height, and interdental papilla should also be considered as these features influence gingival shape. Tarnow et al.[85] described the relationship between crestal bone height and the interdental contact point. They examined 288 human papillae and discovered that if the distance between the contact point and the crestal bone was 5 mm or less, the papilla usually fills the embrasure space 100% of the time. At distances of 6 mm and 7 mm, the percentage decreases to 56% and 27%, respectively.[85] Velvart et al. warned that during reflection, mobilized papilla that is thin, narrow, and high may not have sufficient blood supply and may increase the risk of necrosis and subsequent decreased papilla height after healing.[86] A well-designed flap had the following characteristics:

1. *It can be easily performed by the operator.*
2. *It can be easily reflected by the operator.*
3. *It provides adequate access to the lesion.*
4. *It does not compromise the aesthetic.*

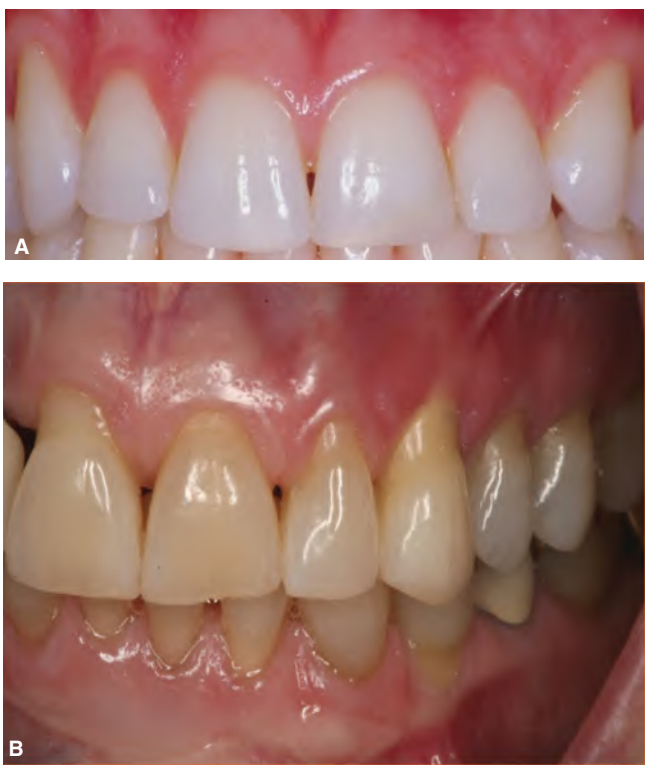

FIGURE 24-20 ***A.*** A thin biotype (thin triangular periodontium and papilla). ***B.*** A thick biotype (square tooth with thick and flat periodontium and wide papilla).

5. *It does not compromise blood flow to the flap or the remaining gingival margin or papilla.*
6. *It does not impinge on important anatomical structures.*

Flap Designs

Soft tissue flaps used in surgical endodontics can be categorized as full mucoperiosteal flaps and submarginal flaps.

Full Mucoperiosteal Flaps

Full mucoperiosteal flap requires the reflection of the entire soft tissue overlying the cortical plate. This flap usually has a horizontal incision and one or two vertical incisions (Figure 24-21). The horizontal incision is an intrasulcular one that needs to incise the medial area and separate the buccal and lingual papilla to prevent double papilla formation. The greatest disadvantage of the horizontal incision is that it may cause slight shrinkage along the gingival crest that leads to recession.[73]

The vertical incision in these flaps is made between root eminences. The vertical incision in these flaps should not be made over eminences because of the presence of thin mucosa at this location. The main advantage of this flap is the preservation of supraperiosteal vessels within the flap and therefore retention of the blood flow to the flap. Depending on the number of vertical incisions and their angulations, these flaps are categorized as: triangular, rectangular, or trapezoidal.

A triangular flap has a single vertical-releasing incision and a horizontal incision (Figure 24-22). This flap allows relatively good access and visibility, precludes incising over a bony defect, and has a lower tendency for a hemorrhage. Advantages of the triangular flap include minimally disrupted vasculature, that results in sufficient blood flow to the free flap.[58] The limited incisions lead to lower morbidity. The triangular flap is easy to perform and provides sufficient reflection to the coronal and middle thirds of the root.[87]

A disadvantage of the triangular flap is an insufficient reflection to the apical third of the root. If this occurs, the flap may have to be extended both mesially and distally to expose a larger area. Another disadvantage of this flap is the high risk of gingival recession due to the fully released papilla.[87] This is the main disadvantage of sulcular flaps and is due to tissue shrinkage.[86] Moiseiwitsch[53] criticized the profession for shying away from periapical surgery in the mandibular premolar region. He pointed out the common practice of placing the vertical incision for a triangular flap at the mesial line angle of the canine so the mental nerve would be stretched within the flap to achieve adequate access for surgery on a tooth posterior to mental foramen. He suggests placing the vertical incision distal to the surgical site so that good access is achieved without stretching the content of the mental foramen. One complication of this method is compromising blood supply to the soft tissue of this region. On the other hand, Kim[39] suggests placing a vertical incision mesial to the first premolar when performing a surgery on the mandibular first molar. The reason is to avoid the mental foramen and also to avoid the muscle attachment at the second premolar, which heals poorly if damaged.

The rectangular flap is advantageous in the anterior area (especially when the roots are long) and provides better access than the triangular flap (Figure 24-23).[39] Since the vertical incisions are parallel to the larger vessels, the blood supply is minimally disrupted. They provide better access in the case of long roots with shallow vestibules, and visualization is improved.[87] Kim[39] argues against making the flap wider in the base (i.e., trapezoidal flap design) due to inadequate supporting evidence and also due to the cutting of the blood vessels obliquely rather than parallel to them. Mormann and Ciancio[88] have shown that the circulation is disturbed when the ratio of the length to the base of the flap is more than 2:1.

Limited Mucoperiosteal Flaps (Submarginal Flaps)

The horizontal and vertical incisions in the submarginal (limited mucoperiosteal) flaps do not involve gingival margins and papilla. The horizontal incision in these flaps is made in the attached gingiva, and the vertical incisions are made in

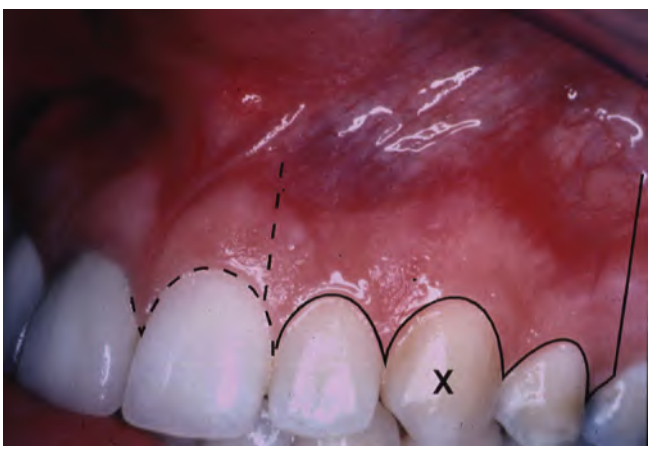

FIGURE 24-21 A full mucoperiosteal flap usually has a horizontal incision and one or two vertical incisions. The vertical incisions should be made at least one tooth away from the apex of the target tooth.

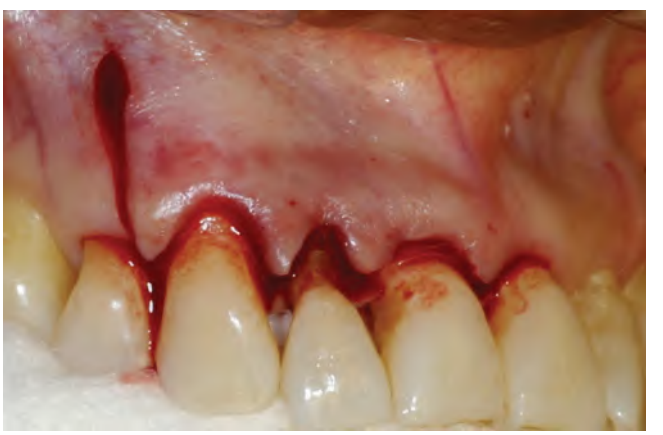

FIGURE 24-22 A full mucoperiosteal triangular flap has a single vertical-releasing incision and a horizontal incision. The vertical incisions are made one tooth away from the apex of the right lateral incisor tooth.

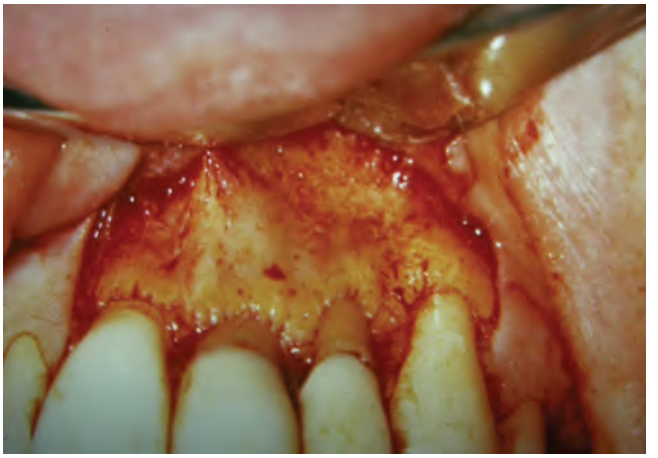

FIGURE 24-23 A full mucoperiosteal rectangular flap is raised to perform a periapical surgery on maxillary central and lateral incisors with long roots.

both alveolar mucosa and attached gingiva (Figure 24-19). In this design, there is a need to maintain at least 2 to 3 mm of attached gingiva. This action prevents mucogingival complications and achieves an adequate wound closure.

This design was developed by Ochsenbein and Luebke in 1974 and is commonly used when crowns and bridges are present. This flap is created by placing scalloped incisions in the attached gingiva about 1 mm away from the mucogingival junction and 2 mm apical to the depth of the sulcus followed by two vertical-releasing incisions to provide adequate access to the apical site. The placement of incisions in the attached gingiva aids in blood supply to the area and also allows for rapid wound healing.[89] This design allows blood supply to the gingival margins from the crestal bone and through the papilla from the lingual side. Since the attached gingiva around crown margins are not incised, there is reduced risk of recession.[73,87] The scalloped design allows for exact positioning of the wound edges. This design is preferred in aesthetic locations with porcelain restorations. Postoperative soft tissue contraction is minimized by this design, and aesthetics are usually preserved.

The limitation of this flap design is its requirement for at least 2 mm of attached gingiva coronal to the incision line.[90] It also requires ideal periodontal health and bone support. When periodontal pocket depths are excessive or inadequate attached gingiva is present, using this flap can lead to complications such as dehiscence or fenestrations. Furthermore, there is no access to the surface of the root beneath the band of attached gingiva. Moreover, an unpredictable scar line is a common occurrence with this design (Figure 24-24).[91]

A variation of this design was developed by Vreeland and Tidwell.[92] In this variation, a split thickness flap is first incised for the first 2 mm until it reaches the bone, followed by full thickness reflection. This technique allows for less exact positioning of the wound edges.[92] However, the complications are similar to those of the previous design.

To prevent scarring by submarginal flap designs, Velvart[89] developed papilla-based incisions (Figure 24-25). He states

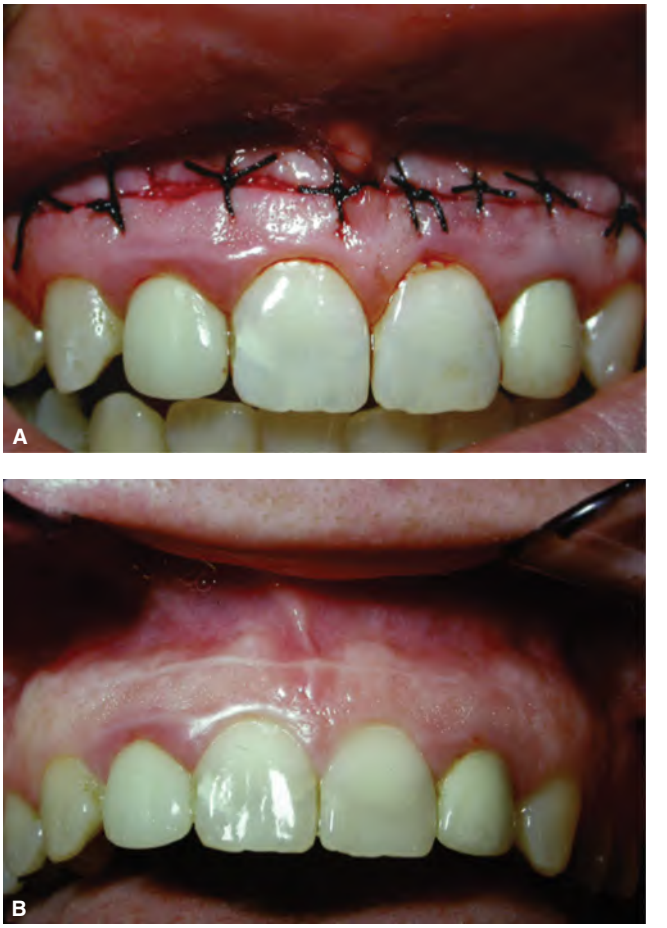

FIGURE 24-24 ***A.*** An Ochsenbein-Luebke flap after suture placement. ***B.*** Scar formation three months after the surgery.

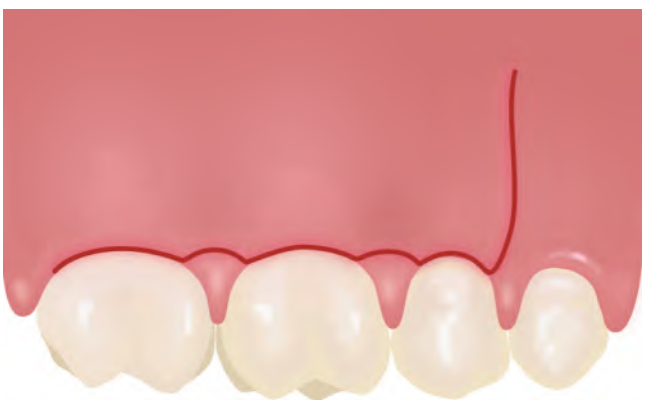

FIGURE 24-25 A submarginal flap is used to preserve the papilla and prevent gingival recession.

that maintaining the integrity of papilla during the surgical procedure is significant for functional, phonetic, and aesthetic reasons. Recession is a significant risk when incisions are placed in the crown margins. Velvart[89] has shown a mean of 0.05 ± 0.39 mm of papillary height one month after surgery. After three months, virtually no recession was evident when a papilla-based incision was made. In contrast, the adjacent papilla with a sulcular flap incision had 1.25-mm recession.

Velvart[89] recommends atraumatic handling of soft tissue as a required note for success of this flap design. This technique involves two incisions at the base of the papilla: one a shallow incision to a depth of 1.5 mm through epithelium and connective tissue and a second incision directed to the crestal bone. This method prevents thinning of the coronal aspect of the flap, which is important for healing. Velvart[89] states that the papilla-based incisions not only prevent recession of papilla, but also cause minimal scarring. Von Arx et al.[93,94] showed the loss of papillary height to be greatest with a sulcular incision compared to papilla-based or submarginal incisions. Sargolzaie et al.,[95] in a randomized controlled trial, showed significant decrease in gingival margin height using a full thickness flap compared to a papilla-based technique at a one-month recall. However, no statistical difference was present in bleeding on probing, attachment loss, probing depth, or the gingival index between the two approaches.

Sabeti et al.[96] described a modified papillary horizontal incision to preserve the papillae and enhance the aesthetic outcome of the surgical endodontics (Figure 24-26). The purpose of this technique is to produce an endodontic surgical flap design that underscores the importance of soft tissue preservation, repair, and reconstruction. For this technique, using a microsurgical blade, horizontal papillary incisions are made at a right angle to the papillae at the level of the CEJ extending two teeth beyond the target tooth mesially and distally, thus creating a butt joint at the papillae. The papillary incisions are connected later, extending the incision intrasulcularly at the labial aspects of all involved teeth in the procedure (Figure 24-26). Depending on the amount of access needed, either a pouch design or one or two vertical incisions will be placed at the line angles of the flap (Figure 24-26). Vertical incisions should be placed at the most distal (most distant proximal) line angle of the tooth in order to use the adjacent unincised papilla for suturing (Figure 24-26). The incision usually extends two teeth beyond the target tooth mesially and distally. This allows sufficient access and visibility. Holding the microsurgical blade at a 45-degree angle, a beveled vertical incision is made to prevent scarring during healing by attaching connective tissue to connective tissue and epithelium to epithelium.[97]

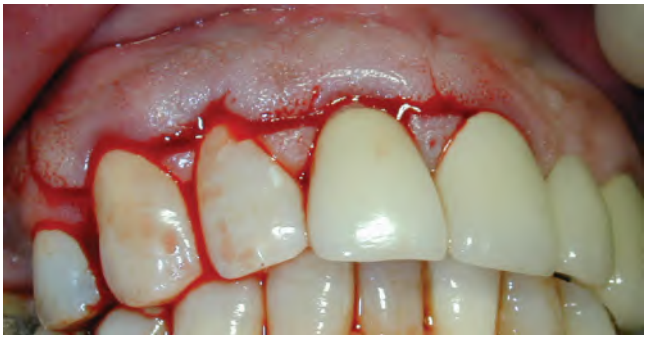

FIGURE 24-26 A modified submarginal flap is used to preserve the papillae and enhance the aesthetic outcome of the surgical endodontics.

The vertical incision is made in the form of a c-shape (curved) and will extend at least 4 mm apical to the extension of the bony defect. Then the incision will end 2 to 3 mm horizontally towards the defect (cutback) to prevent scar formation. The c-shape vertical incisions are composed of a horizontal component at the coronal part, an internally curved component at the mid-part, and a cutback component at the apical part within the mucosa. The advantage of the c-shape incision is that it gives more access and better flexibility for repositioning of the flap than straight vertical incisions. The horizontal component improves tissue adaptation at closure. The internally curved and cutback components provide flap flexibility and reduce tension by increasing the length of the incision. Using blunt dissection, a mucoperiosteal flap is carefully reflected away from the neck of the teeth and available bone to expose the bony margin of the defect.

The advantages of the modified horizontal papillary incisions design are:

- The incision preserves papillae and leaves the marginal gingiva minimally disturbed.
- Wound healing may be enhanced as a result of reduced disruption of vascularity.
- Scar formation is prevented or minimized. Good access and visibility are provided as a result of flap flexibility.
- The flaps may be incised and reflected without much difficulty.
- Repositioning of the flap is easy and precise.
- Patient compliance and ability to maintain good oral hygiene are improved because the marginal gingiva are minimally disturbed, and the patient can brush more comfortably. Root coverage may be achieved.

Flap Elevation and Reflection

Flap elevation is the process of separating the soft tissues (gingiva, mucosa, and periosteum) from the surface of the alveolar bone. This process should begin in the vertical incision a few millimeters apical to the junction of the horizontal and vertical incisions. The periosteal elevator of choice (Figure 24-27) should be used to gently elevate the periosteum and its superficial tissues from the cortical bony plate (Figure 24-28A). Once these tissues have been lifted from the cortical plate, and the periosteal elevator can be inserted between them and the bone, the elevator is then directed coronally. This allows the marginal and interdental gingiva to be separated from the underlying bone and the opposing incisional wound edge without direct application of dissectional forces. This technique allows for all of the direct reflective forces to be applied to the periosteum and the bone. This approach to flap reflection is referred to as "undermining elevation."[71]

This undermining elevation should continue until the attached gingival tissues (marginal and interdental) have been lifted from the underlying bone to the full extent of the horizontal incision (Figure 24-28B). After reflection of these tissues, soft tissue elevation is continued in an apical

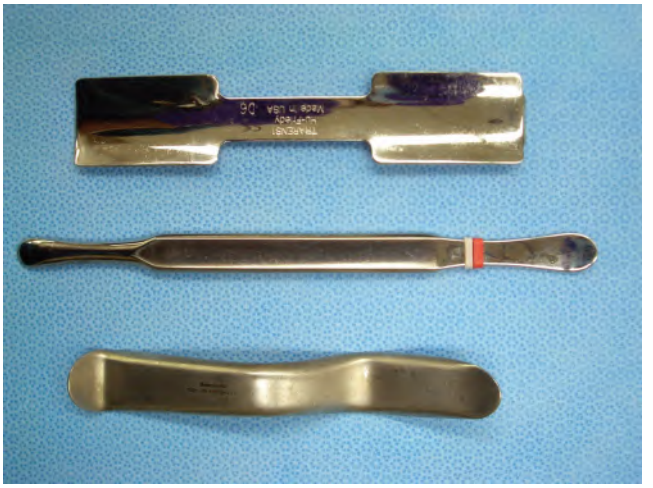

FIGURE 24-27 Examples of endodontic tissue retractors (Top—Arens Tissue Retractor; Middle—Seldin retractor; Bottom—University of Minnesota retractor).

direction, lifting the alveolar mucosa, along with the underlying periosteum, from the cortical bone until adequate surgical access to the intended surgical area has been achieved. Small bleeding tissue tags will often be noted on the exposed surface of the cortical bone once the flap is fully reflected (Figure 24-28C), especially in the interradicular depressed areas. Bleeding from these tissue tags will stop in a few minutes and they should not be damaged or removed. Research evidence strongly suggests that these bleeding tissue tags are cortical retained periosteal tissues and may play an important role in healing and reattachment of the flap to the cortical bone.[98]

Flap retraction is the process of holding in position the reflected soft tissues. Proper retraction depends on adequate extension of the flap incisions and proper reflection of the mucoperiosteum. It is necessary to provide both visual and operative access to the periapical and radicular tissues. The tissue retractor must always rest on solid cortical bone with light but firm pressure. In this way, it acts as a passive mechanical barrier to the reflected soft tissues. If the retractor inadvertently rests on the soft tissue at the base of the flap, trauma to the alveolar mucosa may result in delayed healing and increased post-surgical morbidity.

Selection of the proper size and shape of the retractor (Figure 24-27) is important in minimizing soft tissue trauma. If the retractor is too large, it may traumatize the surrounding tissue. If the retractor is too small, flapped tissue falls over the retractor and impairs the surgeon's access. This results not only in increased soft tissue trauma, but also in extending the length of the surgical procedure. An axiomatic principle of surgical endodontics is that the longer the flap is retracted, the greater the postsurgical morbidity. This is a logical conclusion based on the probability that blood flow to the flapped tissues is impeded during flap retraction. In time, this will result in hypoxia and acidosis with a resulting delay in wound healing.[71]

Regardless of whether the retraction time is short or long, the periosteal surface of the flap should be irrigated frequently with saline (0.9% sodium chloride) solution. Saline should be used rather than water because the latter is hypotonic to tissue fluids. It is not necessary to irrigate the superficial surface of the flap because the stratified squamous epithelium prevents dehydration from this surface. Limited mucoperiosteal flaps are more susceptible to dehydration and may require more frequent irrigation than full mucoperiosteal flaps.[71]

Local Hemostatic Agents

Obtaining local hemostasis during surgical endodontics is essential to successful management of this procedure. Hemostasis during PAS is achieved primarily by the epinephrine present in local anesthetic. The local hemostatic agents used during surgical endodontics are good adjuncts for hemostasis and are intended to control bleeding from small blood vessels. Many hemostatic agents have been advocated for use during PAS. Their mechanism of action, ability to control bleeding, and effect on healing vary considerably. They generally assist coagulation by inducing rapid development of a blood clot.[65] The most commonly used hemostatic agents in PAS are: bone wax, epinephrine pellets, ferric sulfate, calcium sulfate, collagen-based materials, Surgicel, Gelfoam, aluminum chloride, and HemCon.

Bone Wax

Bone wax was introduced as a hemostatic agent by Horsley in 1892.[99] It is a non-resorbable material composed of approximately 88% highly purified bees wax and 12% isopropyl palmitate, which acts as a softening and conditioning agent.[99] The function of bone wax as a hemostatic agent is

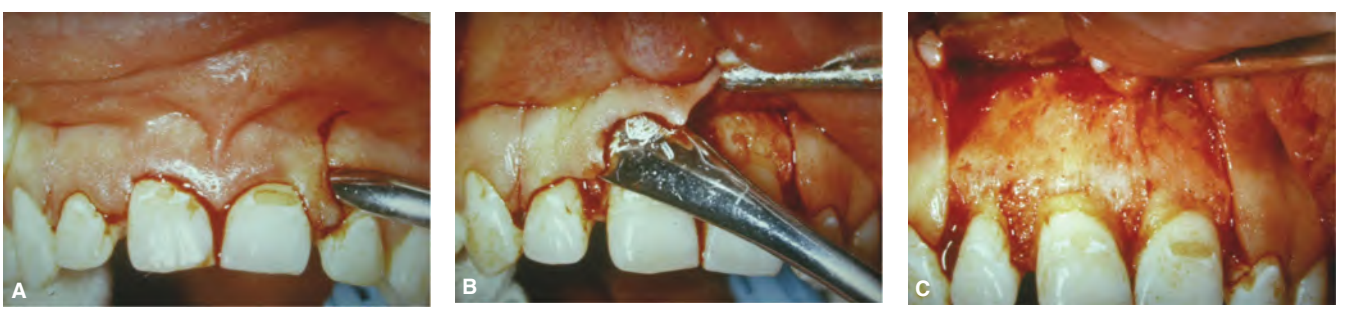

FIGURE 24-28 ***A.*** Initial flap elevation. ***B.*** Partially reflected flap. ***C.*** Completely reflected flap.

purely mechanical. It induces a tamponade effect when it is packed firmly against the bone surface. Bone wax does not affect the blood clotting mechanism.[58]

When using bone wax for the purpose of hemostasis, the entire bony crypt is usually filled with the material under firm pressure. The excess is then carefully removed to expose only the apex of the tooth. Following root-end filling, the bone wax should be completely removed.[58] Studies have reported that remnants of the bone wax in the bony crypt cause persistent inflammation, foreign body reaction, sinus tract formation, and delayed healing.[100–104] Because of these adverse effects, the use of bone wax is not recommended when other alternatives are available.[38,65,101,105]

Epinephrine Pellets

Vasoconstrictors have been recommended as topical agents for hemorrhage control during PAS. Many clinicians use epinephrine as the primary hemostatic agent during periapical surgery.[38,58] Before placing an epinephrine pellet in the bony crypt, granulation tissue should be removed from the periapical tissue. This action ensures direct contact of epinephrine with the bone. A combination of chemical and mechanical hemostatic actions by use of epinephrine and application of pressure has a profound effect on hemostasis.[58] Cotton pellets containing racemic epinephrine HCl (Racellet #3; Pascal Co, Bellevue, WA, U.S.A.) are commercially available (Figure 24-29). Each pellet contains an average of 0.55 mg of racemic epinephrine hydrochloride and usually provides good local hemostasis during periapical surgery.[106] However, two concerns arise with the use of epinephrine-impregnated cotton pellets for topical hemostasis: (1) systemic cardiovascular effects and (2) the retention of cotton fibers in the wound.

Because cancellous bone is highly vascular and contains a large amount of capillary distribution, direct application of epinephrine to the bone during periapical surgery may produce marked cardiovascular changes.[107] Wood et al.[108] compared the heart rate of patients after local application of intraosseous injection with that of patients who had infiltration injection. Significant increase in the heart rate was identified in those patients who had intraosseous injection compared with those who had infiltration injection after two minutes.[108] Besner reported that the pulse rate of patients did not change when one Racellet pellet containing an average of 1.15 mg of epinephrine HCl was used during periapical surgery.[65] Vickers et al.[106] measured blood pressure and pulse rate changes in 39 patients when #3 Racellet pellets were left in the bony crypt for two to four minutes. Based on their results, they concluded that there was no evidence for significant cardiovascular changes when #3 Racellet pellets were compared with saline-saturated pellet controls.[106] These findings were later confirmed by Vy et al.[109] who showed that placement of 2.25% racemic epinephrine in CollaCote collagen had little or no effect on the blood pressure and pulse rate of patients during surgical endodontics.[109] Kim and Kratchman claim that topical application of epinephrine has a local vasoconstrictive effect on the capillaries and has little or no systemic effect.[58] Based on the available evidence in the literature, it appears that the systemic cardiovascular effect of cotton pellets containing epinephrine is minimal.

The retention of cotton fibers present in the cotton pellets saturated with epinephrine used during PAS is a critical issue.[37,110] Gutmann and Harrison[37] stated that loose cotton fibers left in the surgical site may affect the root-end seal by becoming trapped between the root-end cavity preparation and the root-end filling material. They also note that cotton fibers may serve as foreign bodies in the surgical site and may delay wound healing. To address this concern, each pellet applied during periapical surgery must be accounted for and the bony crypt should be lightly curetted to remove any embedded cotton fibers before wound closure.[105] The retention of cotton fibers in the crypt can be eliminated by use of CollaCote saturated with 10 drops of 2.25% racemic epinephrine with cotton pellets.[109]

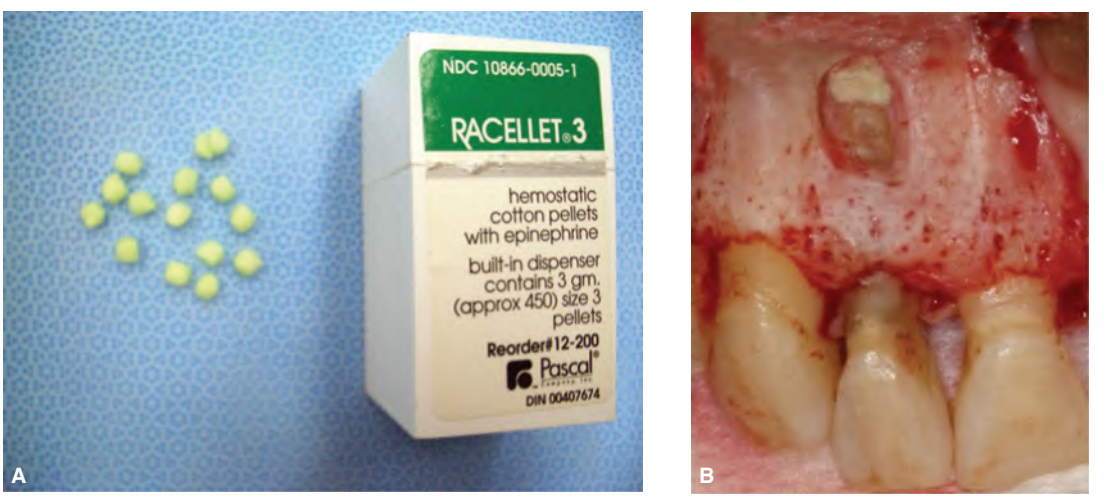

FIGURE 24-29 ***A.*** Cotton pellets containing racemic epinephrine HCl are commercially available. ***B.*** Topical application of these pellets has a local haemostatic action.

Ferric Sulfate

Ferric sulfate is a chemical that has been used as a hemostatic agent since it was first introduced as Monsel's solution (20% ferric subsulfate) in 1857.[58] It is a necrotizing agent with a pH of 0.8 to 1.6.[101] Ferric sulfate causes agglutination of blood proteins and plugging of the capillary orifices. When it comes in contact with vital tissues, it immediately causes color change. Because of its low pH, this material is very cytotoxic and must be used with caution. Complete removal of coagulum produced after its use is recommended.[58,111]

Lemon et al. reported a significant adverse effect on osseous healing when ferric sulfate was left in standardized osseous defects created in rabbit mandibles. They reported impaired healing, a foreign body reaction, or, in some cases, abscess formation when ferric sulfate was left in the rabbit mandibles (Figure 24-30A).[112] In a follow-up study using the same model, the authors reported no significant difference in healing between the experimental and untreated control groups when the surgical sites were curetted and irrigated with saline following a five-minute application of ferric sulfate (Figure 24-30B).[111] Therefore, application of ferric sulfate as a hemostatic agent is safe as long as the coagulum is completely removed from the surgical site.[58,101,111,112]

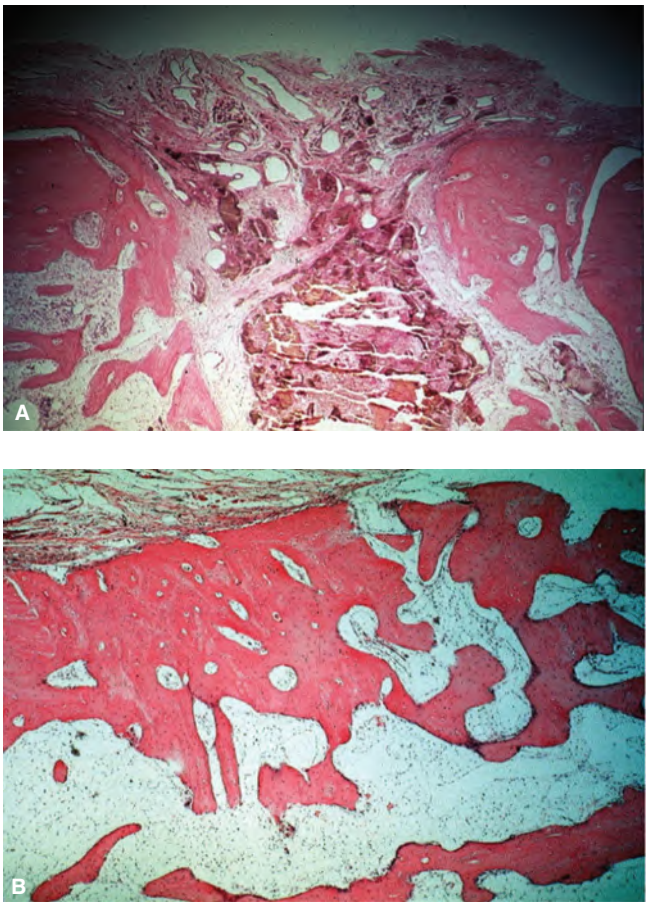

FIGURE 24-30 ***A.*** Impaired healing, a foreign body reaction, and abscess formation are observed when ferric sulfate was left in the rabbit mandibles.[111] ***B.*** No adverse effects are noted when the surgical sites are curetted and irrigated with saline following an application of ferric sulfate.[112]

Calcium Sulfate

Calcium sulfate, also known as plaster of Paris, has been used as a substitute bone graft material to fill bone defects since the late 1800s.[105] It has been primarily used in dentistry to fill large surgical bone defects, as a barrier material in guided tissue regenerative procedures, and repair of furcation defects.[65] Calcium sulfate has been recommended as a hemostatic material during periapical surgery. It serves as a physical barrier and plugs vascular channels.[58,113] It is a resorbable material and after placing it in the bony crypt, using wet cotton pellets, and packing it against the crypt walls, it can be carved away to allow access to the root-end.[65, 113,114] After completion of root-end filling, the residual calcium sulfate can be removed or left in the bony crypt.

Collagen-based Materials

The collagen used for surgical hemostasis is obtained from bovine sources and is supplied in sheets and sponge pads.[65] Studies have shown that osseous regeneration in the presence of collagen usually takes place uneventfully and without a foreign body reaction or infection.[115] A study that examined the effects of hemostatic agents on peripheral nerve function in rats showed that bovine collagen is the most suitable hemostatic agent compared to bone wax, gelatin sponge, and Surgicel.[116]

Surgicel

Surgicel is a material that resembles surgical gauze and is prepared by the oxidation of regenerated alpha cellulose (oxycellulose).[100,101] It primarily acts as a physical barrier and, after coming in contact with blood, becomes a sticky mass that serves as an artificial coagulum.[100,101,105] In an animal study that compared the osseous response of three different hemostatic agents (i.e., bone wax, Surgicel, and Gelfoam), when left *in situ*, Surgicel retarded the rate of healing and caused the most intense inflammatory reaction between the test materials.[100] Because of the difficulty in removing all of the Surgicel from bony wounds, the small fragments left behind result in a prolonged inflammatory response and foreign body reaction.[100]

Gelfoam

Gelfoam is a gelatin-based sponge that is water insoluble and biologically resorbable.[58,100,101] It stimulates the intrinsic clotting pathway by promoting platelet disintegration and the subsequent release of thromboplastin and thrombin.[58,100,101] Ibarrola et al. compared the response of rat tibias to bone wax, Surgicel, and Gelfoam histologically.[100] At the end of the 120-day experiment period, Gelfoam usually resorbed completely and the bone defects healed adequately.

Aluminum Chloride

Expasyl (Kerr Corp., Orange, CA, U.S.A.), a paste containing aluminum chloride and kaolin has been advocated for gingival retraction to ensure separation of the marginal gingiva and drying of the sulcus before impression-taking and insertion of

restorations.[117,118] Several studies have shown that aluminum chloride elicits inflammatory tissue reactions.[119,120] Von Arx et al.[117] compared the three different hemostatic agents, bone wax, Expasyl, and ferric sulfate (Stasis), in a rabbit calvarium model for periapical surgery. Expasyl was found more effective than Stasis in controlling the bleeding. Expasyl in combination with Stasis proved to be the most effective test materials to control the bleeding within the bony defects. However, in that same study, aluminum chloride demonstrated a foreign body reaction and the presence of giant cells and other inflammatory cells after three weeks and 12 weeks post-operatively. New bone formation was minimal and delayed in contrast to the control sites. To prevent these reactions, it is currently recommended to curette and freshen the walls of the bony crypt with a round bur and rinse the surgical site with saline before wound closure when using aluminum chloride as a hemostatic agent in periapical surgery.[117]

HemCon

HemCon dental dressing (Patterson Dental, St Paul, MN, U.S.A.) has been recently suggested as a potential hemostatic agent. This material not only is hemostatic, but also has bactericidal properties; an additional benefit when compared to other hemostatic agents.[121] It is manufactured from freeze-dried chitosan and molds to form a highly electropositive sponge-like material that binds to negatively-charged red blood cells. As such, it results in the formation of a viscous clot that facilitates hemostasis.

Osteotomy

By definition, osteotomy is the removal of cortical and cancellous bone covering the root end(s) at the site of surgery (Figure 24-31). The location and size of entry to the bony crypt where the root-end resection and preparation as well as root-end filling are performed is an important phase of PAS. Complete radiographic examinations are essential components of any aspect of root canal treatment.[122] Periapical radiographs have limited value for the diagnosis and extent of periapical lesions and their relationship to vital structures. The use of CBCT is essential to determine the location and size of periapical lesions, their relationship to the vital structures, and thickness of cortical bone plates.[69,70]

Proper osteotomy provides access to the periapical aspects of a tooth. A thorough understanding of anatomy and bone physiology is necessary to avoid mechanical trauma and injury and prevent unwanted complications during and after surgical endodontics. Since bone is a vital tissue, it has to be removed gently and effectively with minimal heat generation. Heat generation during osteotomy is affected by bur size and design, speed of cutting, amount of penetration, amount of pressure during cutting, and irrigation as well as aspiration of generated debris.[123-133]

Effect of Heat, Bur Design, and Speed

Heat generation during osteotomy can cause irreversible damage to an already vulnerable bone that has had a reduction of blood flow into it due to the use of vasoconstrictor. Eriksson et al.[123] reported that the threshold for irreversible damage to the bone is between 44°C and 47°C for one minute. During osteotomy, the use of saline coolant is imperative to lower the temperature of the cut tissues. Another contributing factor to the generation of heat during osteotomy is the method of bone removal. Matthews and Hirsch[124] conducted cadaver studies and examined the effect of incremental and constant drilling. They found incremental drilling resulted in effective temperature reduction and reduction of generated debris.[124] Based on these results, osteotomy should be performed with light stroking motions and copious saline irrigation to maximize cutting efficiency and minimize heat generation.

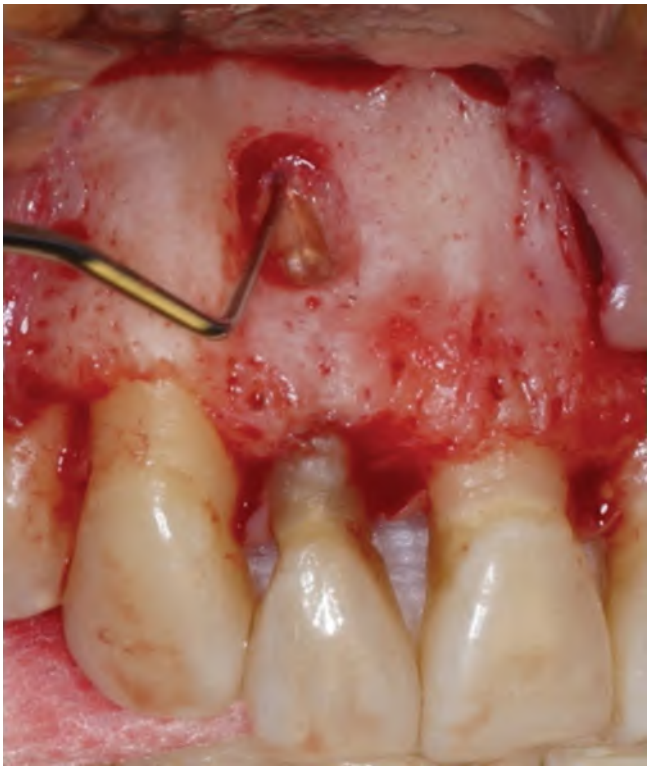

FIGURE 24-31 A complete osteotomy has been performed to carry out periapical surgery on the maxillary right lateral incisor.

Another factor that might affect osteotomy is the speed of the bur during this procedure. The influence of drilling speed, axial drilling force, and feed rate have been extensively studied in relation to bone drilling temperature without much consensus.[125] Vaughan and Peyton[126] found that the increased drill speed during cavity preparation resulted in increased temperature. However, on the contrary, a cadaver study conducted by Matthews and Hirsch[124] found no significant change in temperature during drilling from 345 rpm to 2900 rpm. Nam et al.[127] found that increasing the speed (600 rpm and 1200 rpm) or the force (500 g and 1000 g) affects temperature in the bone.

Several histological studies in the 1960s compared the effect of high speed and low speed handpieces and reported less injury or no injury to the bone with the use of a high-speed handpiece. Despite this inconsistency in the literature,

there have been case reports of surgical emphysema causing subcutaneous emphysema of the face, which may be fatal from the use of high-speed dental handpieces during surgical procedures.[128] The shape of the bur, type of bur, and drill specifications such as rake angle and clearance angle all play a role in the cutting efficiency, and, therefore, heat generation. Kalidindi[129] found that with increasing drill diameter, the temperature increases exponentially, resulting in an increase in overall friction and heat.

The clearance angle or the angle by which the flank of the drill clears the material during drilling is critical as this may cause frictional heat.[129] It is generally recommended that a clearance angle of 15° be used for better cutting efficiency and less heat generation.[130] Moss[131] and Boyne[132] compared different bur types and shapes and reported a No. 6 or 8 round bur produces smoother cut surfaces, less inflammation, and shorter healing times compared to using a fissure or diamond bur. Moss reports that a No. 6 round bur causes a smaller zone of necrosis than a fissure bur.[131] According to Calderwood et al.,[133] diamond burs are the least effective burs for bone removal and cause delayed healing.

Osteotomy Size

A debate exists regarding the size of the osteotomy during PAS. Boyne[132] examined nine patients with 21 periapical defects in the anterior region with at least one cortical plate intact. Lesions were divided into two groups: 5 to 8 mm and 9 to 12 mm. He found complete bone regeneration in smaller defects compared to the larger defects, which contained herniation with fibrous tissue formation. Hjorting-Hansen and Andreason[134] conducted a similar study on dogs where they found complete healing of defects that were about 5 mm or less when one cortical plate was intact. However, when both plates were removed, incomplete healing occurred with formation of fibrous tissue. These results suggest that smaller-sized defects have better chances for complete healing. Microsurgical techniques currently used in surgical endodontics allow the operator to create a significantly smaller-sized osteotomy compared to the traditional technique of periapical surgery. Rubinstein and Kim state that small lesions heal faster than large lesions.[135]

Osteotomy Procedure

A sharp round bur is usually used to remove the bone until the apex is located (Figure 24-32). Using a light brushing motion for removal of the bone is recommended. CBCT is extremely helpful to determine the location of the apex, inclination of roots, thickness of bony plates, and dimension and extension of the periapical lesion. When the lesion has perforated the cortical plate buccally or lingually, a thin bone covers the root, or a fenestration exists, osteotomy can be achieved easily. However, in cases where intact cortical bone is present, the procedure is more complicated and requires removal of the intact cortical plate to expose the apical portion of the tooth.

Placing a radio-opaque material at the site of osteotomy assists in localizing the root(s). The root is generally darker and has a yellowish color, lacks bleeding when probed, and has smooth and hard texture with surrounding periodontal ligament.[136] Use of an operating microscope along with 1% methylene blue dye can assist in separating root from the bone.

Periapical Curettage

The goal of periapical curettage is the removal of pathological soft tissue surrounding the apex (Figure 24-33). This procedure allows optimal access and visibility to the

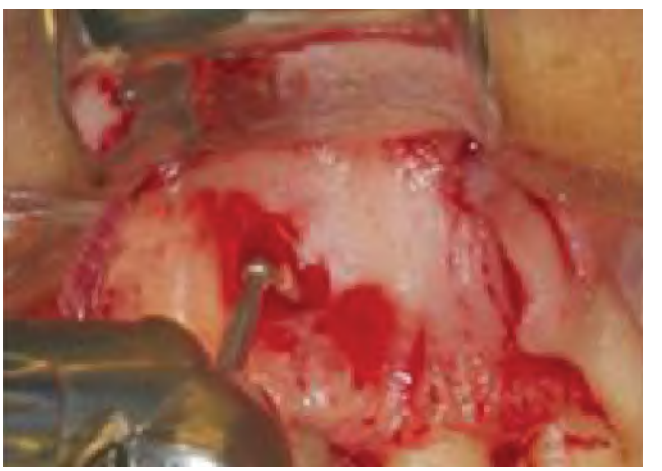

FIGURE 24-32 A sharp round bur in a high-speed handpiece is used to remove the bone. This action should be accompanied with continuous spraying of sterile water.

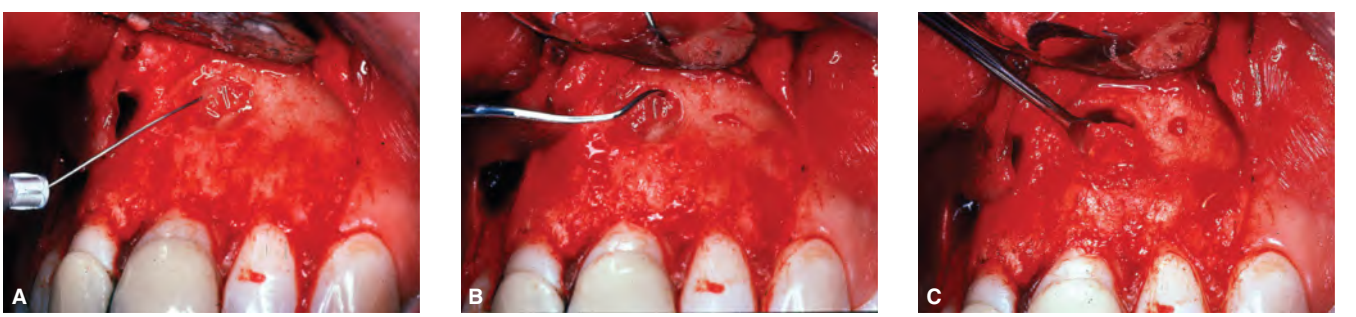

FIGURE 24-33 ***A.*** Injection of local anesthetic into the periapical lesion. ***B.*** Peeling away the lesion with an explorer. ***C.*** Removal of periapical lesion with a curette.

surgical site. Removal of a periapical lesion reduces bleeding and provides a biopsy specimen for histological examination. The soft tissue should be carefully peeled away with a suitable curette. The cavity should be examined under magnification to ensure a clean bony crypt and no tissue tags. Injection of a local anesthetic facilitates removal of the lesion and reduces pain and discomfort during the rest of the operation.[137,138] If the Schneiderian membrane is involved in the soft tissue lesion, careful removal of the lesion is recommended to avoid sinus penetration and perforation.[139] Total removal of the lesion is not necessary to ensure successful outcomes.

Suitable instruments to remove soft tissue include Lucas 86 bone curettes, Columbia 11/12 periodontal curettes, and 13/14 periodontal curettes. A sample of soft tissue should be submitted for biopsy.

Root-end Resection

The purpose of root-end resection is to remove anatomic variations, correct operator errors, evaluate and create an apical seal, and reduce fenestration of root apices. Root-end resection involves beveling the apical portion of the root end (Figure 24-34). Root-end resection removes the untreated apical portion of the root and enables the surgeon to determine the cause of failure. It also provides a flat surface that allows the operator to create a root-end cavity preparation and place a root-end filling material.

There is no consensus regarding the amount of root resection during PAS. Gilheany et al.[140] suggest removal of at least the apical 2 mm of a root to minimize bacterial leakage. Morfis et al.[141] in anatomical studies of the root apices have shown the presence of accessory foramina in all groups of teeth. Kim and Kratchman[58] have suggested that at least 3 mm of the root end must be resected to include 98% of the apical ramifications and 93% of lateral canals. It is not recommended to remove more than 3 mm of the root end, thus preserving 7–9 mm of the root for strength and stability.[58]

Another significant factor to proper root-end resection is the angle of beveled root. One of the major benefits of microsurgery is minimization of the angle of bevel necessary for visualization. In the traditional technique, a bevel of 45° to 60° was necessary with the large surgical instruments. Kim and Kratchman[58] believe that this procedure can expose many tubules, cause greater loss of buccal bone, and weaken the root. However, with the modern microsurgical technique, they recommend little or no beveling of the root during root-end resection.[58]

It is crucial to analyze the anatomy of the root outline after root-end resection. According to Kim and Kratchman[58] this is one of the most important steps for microsurgery and critical for the success of the treatment. Carr[142] attributes failed surgical cases to the missed lingual canal or inadequate cleaning of the isthmus. Observing the shape and the outline of the root as well as identifying isthmuses under the microscope with methylene blue dye can assist in avoiding this misstep. Under high magnification, any cracks, canal aberrations, missed canals, or isthmuses can be identified after a complete root-end resection.[143]

Root-end Cavity Preparation

Conventional root-end cavity preparation technique utilized rotary burs in a micro-handpiece. This technique had many shortcomings such as difficulty of access (especially with limited working space), risk of perforation of the root, and insufficient depth of root-end cavity preparation to place an adequate root-end filling material. In addition, a large number of infected isthmus spaces could not be cleaned and filled.[144] In 1957, the first ultrasonic instrument in endodontics was introduced by Richman. Since then, Carr introduced retro-shaped ultrasonic tip design.[142] One of the most significant advances in periapical surgery was the introduction of ultrasonic micro-surgical tips for the root-end preparation. The use of ultrasonic tips is currently considered the standard of practice in surgical endodontics.

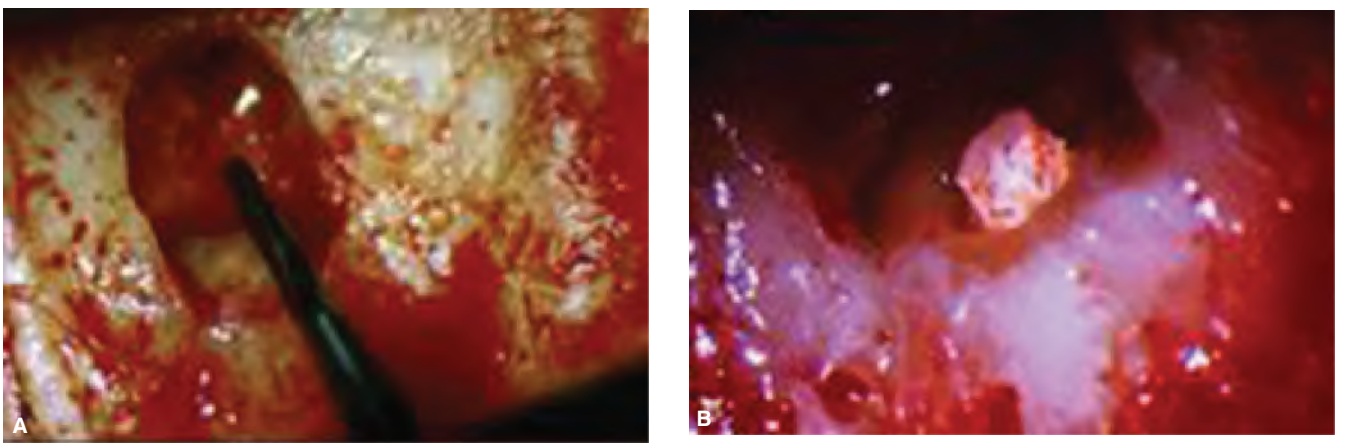

FIGURE 24-34 ***A.*** Root-end resection is performed using a fissure bur in a high-speed handpiece with continuous spray of sterile water. ***B.*** The entire apex should be resected and be visible for inspection.

Several studies reported the advantages of using ultrasonic tips over conventional instruments. There are several ultrasonic tips designed for root-end cavity preparation during surgical endodontics (Figure 24-35).[145] In a scanning electron microscope (SEM) study, Wuchenich et al.[146] compared ultrasonic and conventional bur root-end cavity preparations in cadavers. They found that the ultrasonic tips produced more parallel walls and cleaner surfaces compared to bur preparations (Figure 24-36). In another SEM study, Gorman et al.[147] reported that the root-end cavities prepared with ultrasonic instruments showed significantly less smear layer compared with those made by conventional burs. In a prospective randomized controlled treatment of 399 patients, the overall success rate in teeth whose roots were prepared with ultrasonic tips was 80.5%. This dropped to 70.9% when the roots were prepared with conventional burs.[148] This clinical study showed a significantly better outcome when ultrasonic tips were utilized to prepare deeper and cleaner preparations and address anatomical difficulties.[148]

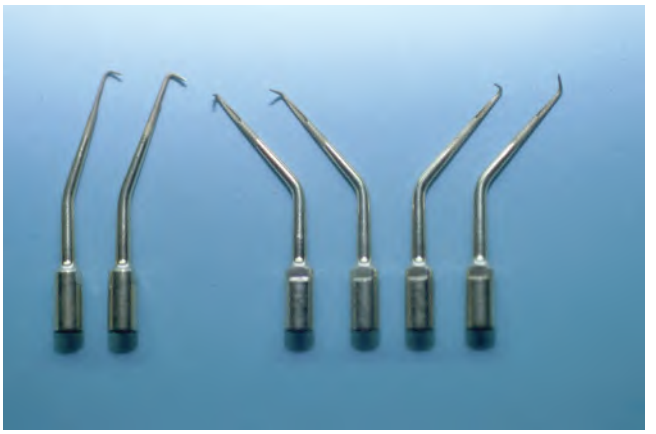

FIGURE 24-35 Currently, a variety of ultrasonic tips are available to prepare root-end cavities for various roots with different characteristics.

However, despite many advantages, ultrasonic tips can cause root crack formation and apical leakage.[149] As found in an *in vitro* study by Layton et al.,[150] root-end cavities prepared by ultrasonic tips can increase the number of micro-fractures significantly compared to roots that only had root-end resections. The study suggested that lower power settings may reduce the number of micro-fractures. Contrary to these findings and other investigations, Gray et al.[151] conducted a study in cadavers and found an insignificant number of cracks with ultrasonic preparations. This difference might be due to the presence of periodontal ligament in cadaver teeth that absorbs vibrations during root-end preparations with ultrasonic tips.

In most of these studies, stainless steel ultrasonic tips were used during root-end preparations. Recently, many diamond-coated tips have entered the market along with the belief that they cut more quickly and efficiently and reduce contact time with the tooth. Although the microfractures may occur as a result of the use of ultrasonic tips during root-end preparations, there is a lack of evidence correlating the presence of the fractures with success rates of surgical endodontics. Thus, the advantages of ultrasonic techniques outweigh the unknown clinical significance of the occurrence of microfractures.[148]

Currently, a variety of tips is available to accommodate virtually all access situations. When used, the tips are placed at the root-end parallel to the long axis of the root. A Class I cavity preparation is made with ultrasonic tips to a minimum depth of 3 mm into the canal (Figure 24-37). More complicated root anatomy may require other types of preparations.

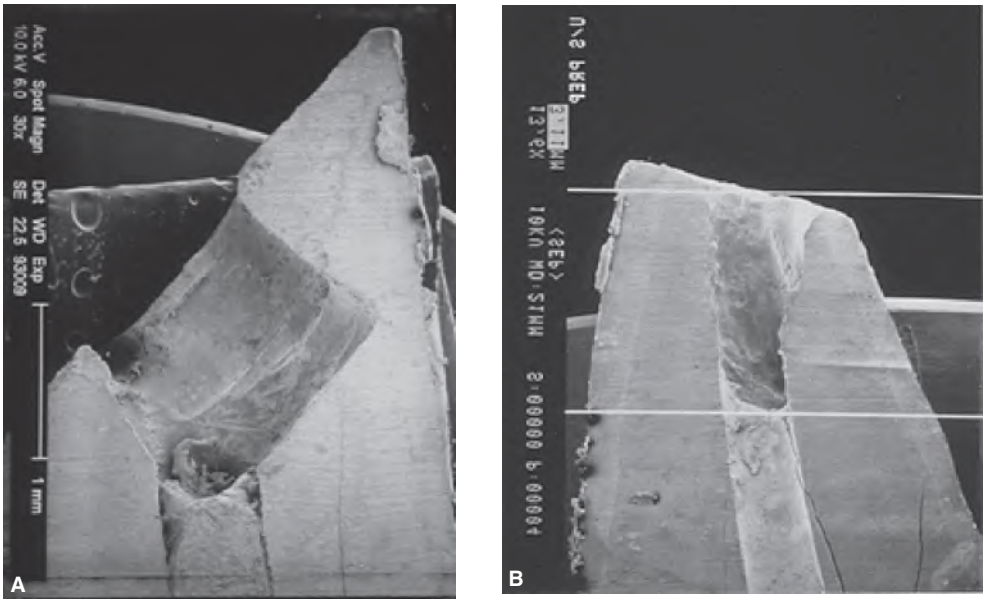

FIGURE 24-36 ***A.*** SEM image of a root-end cavity prepared by a small inverted bur. ***B.*** SEM image of a root-end cavity preparation by an ultrasonic bur.

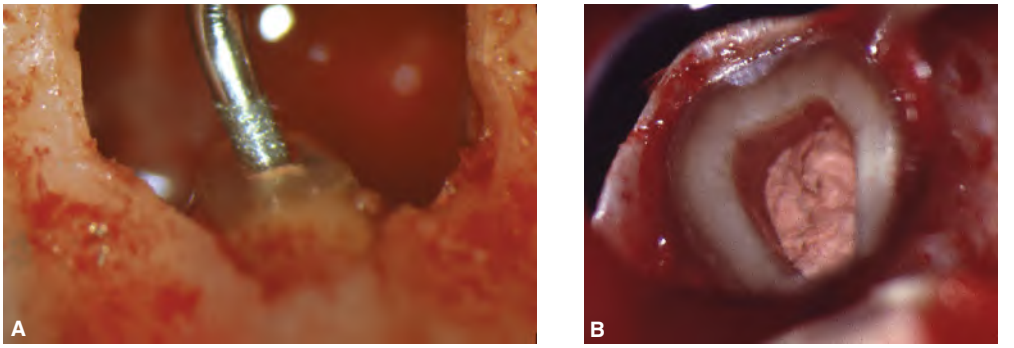

FIGURE 24-37 ***A.*** An ultrasonic tip is used to make a root end cavity preparation. ***B.*** A Class I root cavity preparation is prepared with the ultrasonic tip to a depth of 3 mm into the canal.

The ultrasonic tips offer the advantages of control and ease of use and permit uniform depth of preparation.[152] In addition, the ultrasonic tips produce smaller apical preparations, allow easier preparation of an isthmus, follow the direction of the canals, clean the canal surfaces better than burs, and create less fatigue for the operator.[151,152]

Root-end Filling Materials

The purpose of a root-end filling is to establish a seal between the root canal space and the periapical tissues. An ideal root-end filling material should be: 1) able to prevent leakage of bacteria and their by-products into the periapical tissues, 2) non-toxic, 3) non carcinogenic, 4) biocompatible with the host tissues, 5) insoluble in tissue fluids, 6) dimensionally stable, 7) unaffected by moisture during setting, 8) easy to use, 9) radiopaque, 10) nonstaining, and 11) bioinductive (promote cementogenesis).[153] Numerous materials have been introduced as root-end filling materials including gutta-percha, amalgam, polycarboxylate cements, zinc phosphate cements, zinc oxide eugenol paste, IRM cement, EBA cement, Cavit, glass ionomers, composite resins, gold foil and leaf, silver points, cyanoacrylates, polyHEMA and hydrogen, Diaket root canal sealer, titanium screws, and Teflon.[37] The suitability of root-end filling materials has been tested by their leakage assessment, marginal adaptation, cytotoxicity, and usage in experimental animals and humans. Although a plethora of materials is available, no material has been found that fulfills all or most of the properties for ideal retrograde filling material.

In 1993, Mineral Trioxide Aggregate (MTA) was developed as a new root-end filling material by Torabinejad et al. at Loma Linda University, CA, U.S.A. The main ingredients of MTA are tricalcium silicate, tricalcium aluminate, tricalcium oxide, and silicate oxide.[154] MTA has been evaluated extensively for microleakage (dye penetration, fluid filtration, and bacterial leakage), marginal adaptation (SEM), and biocompatibility (cytotoxicity, tissue implantation, and *in vivo* animal histology). The sealing ability of MTA has been shown to be superior to that of Super-EBA and was not adversely affected by blood contamination.[155] Its marginal adaptation was shown to be better than that of amalgam, IRM, or Super-EBA.[156] MTA has also been shown to be less cytotoxic than amalgam, IRM, or Super-EBA.[40] Its pH, when set, is 12.5 and its setting time is three hours and 45 minutes, which is much longer than that of amalgam, IRM, or SuperEBA. The compressive strength of MTA is reported to be 40 MPa immediately after setting, which is less than that of amalgam, IRM, and SuperEBA, but increases to 70 MPa after 21 days, that is comparable to that of amalgam, IRM, and SuperEBA.[154] Unlike other root-end filling materials that do not tolerate moisture, MTA requires moisture to set. Hydration of MTA powder results in a colloidal gel that solidifies into a hard structure.[157] Several studies reported low or no solubility of MTA. However, a long-term study showed increased solubility over a 78-day period.[158] Although some of the studies showed conflicting results, an investigation confirmed that MTA has limited antimicrobial effect against some facultative bacteria.[159] Animal usage tests in which MTA and other commonly used root-end filling material were compared have resulted in less observed inflammation and better healing with MTA. Histological sections in these studies have demonstrated the regeneration of new cementum over the MTA root-end filling,[160,161] a phenomenon not observed with other commonly used root-end filling materials (Figure 24-38). Recent clinical studies have shown significantly better outcomes when MTA was used compared with other commonly used root-end filling materials.[95,162]

Root-end Filling Placement

The method for placement of the root-end filling material will vary depending on the type of filling material used. Zinc oxide-eugenol cements (IRM and Super-EBA) are best mixed to a thick clay-like consistency, shaped into a small cone, and attached to the back side of a spoon excavator or the tip of a plastic instrument or Hollenback carver, and placed into the root-end preparation.

MTA is a unique root-end filling material with physical properties much different than those of other materials. It is a very fine, gray-colored powder that is mixed with a sterile liquid, such as saline or local anesthetic

FIGURE 24-38 Complete periapical regeneration of tissues including formation of cellular cementum *(C)* adjacent to the MTA are observed when MTA is used as a root-end filling material in dogs.

solution, on a sterile glass slab. It cannot be mixed to a clay-like consistency, similar to IRM or Super-EBA, because, as more powder is added to the liquid, the mix becomes dry and crumbly. If the mix is too wet, it is runny and very difficult to handle due to its lack of form. The surgical area must be kept very dry during its placement and care must be taken not to wash out the filling material by irrigation prior to closure of the soft tissue. The setting time of MTA is 2.5 to 3 hours. Properly mixed, MTA should be free of excess moisture—firm but not crumbly. It can be delivered to the root-end preparation by various carriers (Figure 24-39).

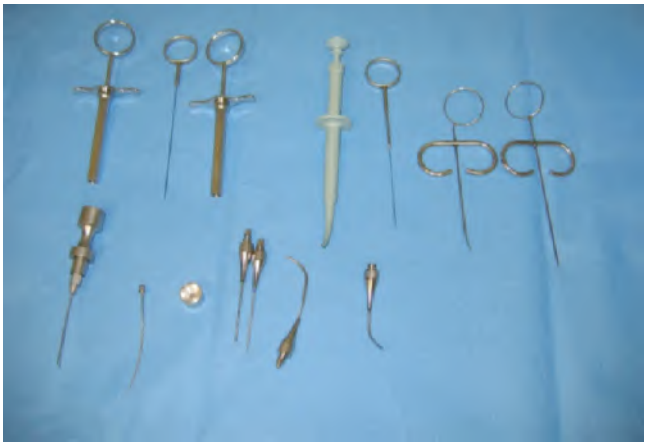

FIGURE 24-39 Different carriers for placement of MTA into root-end preparations.

Wound Closure

After final inspection of the root-end filling and removal of all visible excess filling material and any surgical packing, a radiograph should be taken to evaluate the placement of the root-end filling and to check for the presence of any root fragments or excess root-end filling material. A thorough examination of the underside of the flap and in the depth of the fold between the mucoperiosteum and the alveolar bone should be done prior to repositioning the flap in order to remove any debris or foreign material that may be present. The final steps in periapical surgical procedures are wound closure and soft tissue stabilization.

Repositioning and Compression

The elevated mucoperiosteal tissue should be gently replaced to its original position with the incision lines approximated as closely as possible. The type of flap design will affect the ease of repositioning, with full mucoperiosteal flaps generally providing less resistance to repositioning than limited mucoperiosteal flaps. Using surgical gauze, slightly moistened with sterile saline, gentle but firm pressure should be applied to the flapped tissue for two to three minutes (five minutes for palatal tissue) prior to suturing (Figure 24-40). Tissue compression, both before and after suturing, not only enhances intravascular clotting in the severed blood vessels, but also approximates the wound margins, especially the dissectional wound. This reduces the possibility of a blood clot forming between the flap and the alveolar bone. Coagulum formation under the flap delays healing. There are many factors that may promote, compromise, or delay wound healing.[163]

Healing

Although wound closure is performed at the end of PAS, it is no less important than other steps of this procedure. It is essential that the operator has a good scientific and technical aspect of wound healing so that the tissues are managed for predictable outcomes. Soft tissue management is a crucial part of a clinician's skill, and it not only ensures a successful endodontic outcome, but it also produces aesthetic results.

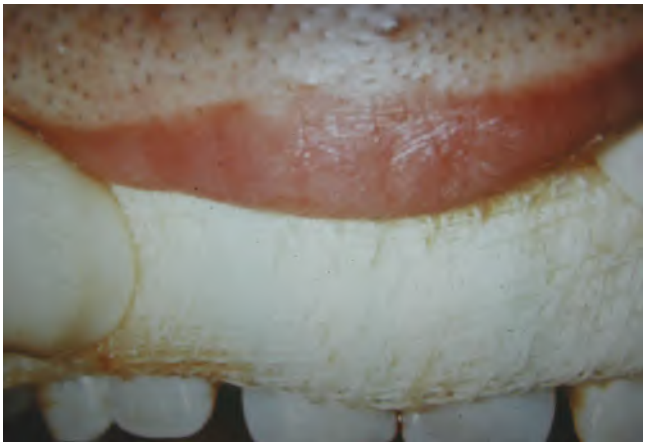

FIGURE 24-40 A large piece of gauze sponge moistened with saline is applied to flap after suturing.

Different types of wounds have different rates of healing. Selvig and Torabinejad[164] compared wound healing in cats after flap surgery. They reported that the fastest healing was observed in the region of the free gingiva (seven days), whereas healing of the attached gingiva (14 days) and the alveolar mucosa (28 days) took considerably longer. If healing occurs more quickly in some areas than others, it can be anticipated where complications might arise. The findings by Selvig and Torabinejad[164] help to explain why dehiscence and healing by secondary intention occur more frequently in the alveolar mucosa.

Once the thin blood clot beneath the flap is replaced with parallel fibers for collagen adhesion, the sutures should be removed. This process takes place in two to four days. At three days, the incisional wound is covered with two to three cell layers. Therefore, the earliest reasonable time to remove sutures is four days after surgery.[164]

Suturing

Sutures do not perform miracles on their own; flap designs and handling of the soft tissues are important factors, too. The soft tissue flaps should remain in position after suturing even when the lips and tongue move (Figure 24-41). The purpose of placing sutures is to reapproximate the wound edges and stabilize the flap accurately in the desired position. This process should ensure primary intention healing with formation of a thin fibrin clot with little or no movement.

Sutures are used to assist in the initial attachment. They can be removed once the blood clot has been replaced by collagen fibers. If sutures remain for an extended period of time, a peri-suture epithelial sleeve can develop in as little as three days. The entire suture track can be enrobed in seven days.[165] Sutures should not be incorporated into healing, but should facilitate initial attachment.

An ideal suture should support the incision while it heals, conform to the tissues, not harbor bacteria, cause minimal tissue reaction, and dissolve when it is no longer needed. Sutures are available in many different materials, the most common being synthetic fibers (nylon, polyester, polyglactin, and polyglycolic acid), collagen, gut, and silk. Sutures are classified as absorbable or nonabsorbable, by size according to the manufacturer's minimum diameter, and by physical design as monofilament, multifilament, twisted, or braided.

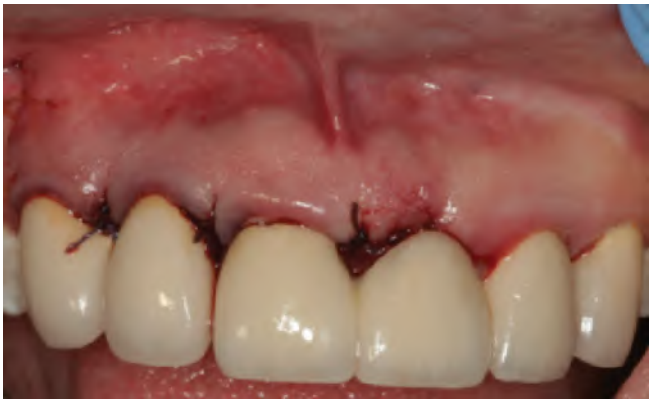

FIGURE 24-41 The soft tissue flap has remained in proper position and location after suturing and pulling the lips.

The classification of suture size is complicated by the existence of two standards, the United States Pharmacopeia (USP) and the European Pharmacopeia (EP). The USP size is designated by two Arabic numerals (one of which is always a zero) separated by a hyphen (3-0, 4-0, 5-0, etc.). The higher the first number, the smaller the diameter of the suture material. The EP system is a number that represents the manufacturer's minimum diameter tolerance in millimeters (1 = 0.10 mm, 1.5 = 0.15 mm, etc.) of the suture.

Monofilament sutures are advocated by multiple authors. The smooth sides of a monofilament suture decrease the inflammatory reaction and limit tissue adhesion to the suture. Multifilament sutures allow for the attachment of tissue while producing a wicking action that allows harmful bacteria to invade the suture track.[166,167] Silk sutures are not recommended because they tend to accumulate plaque and allow rapid bacterial colonization.[168]

SUTURING TECHNIQUES

There is a wide variety of suturing techniques designed to accomplish the goals of closure and stabilization of flaps involving oral mucoperiosteal tissues. All suturing techniques should be evaluated on the basis of their ability to accomplish these goals. Several authors have compared the effects of continuous and interrupted suture techniques. Their findings indicate that the interrupted suturing technique provides for better flap adaptation than the continuous technique and, therefore, is the recommended and most commonly used technique for surgical endodontics.[169,170]

Sutures are holding mechanisms and should not pull or stretch the tissue as a tear in the flap margin may result. Sutures that close an incision too tightly compromise circulation and increase chances for the sutures to tear loose upon swelling.

Before placing sutures, bleeding should be controlled to prevent the formation of a hematoma under the flap. A hematoma will prevent the direct apposition of the flap to the bone and can act as a culture medium for bacterial growth. The suturing techniques that are most conducive to rapid surgical wound healing are the single interrupted suture, the interrupted loop (interdental) suture, the vertical mattress suture, and the single sling suture.

SINGLE INTERRUPTED SUTURE

The single interrupted suture is used primarily for closure and stabilization of vertical releasing and relaxing incisions in full mucoperiosteal flaps and horizontal incisions in limited mucoperiosteal flap designs (Figure 24-42). The initial needle penetration should be through the independent (movable) tissue. The point of needle entry should be from the buccal or facial and 2 to 3 mm from the incision margin

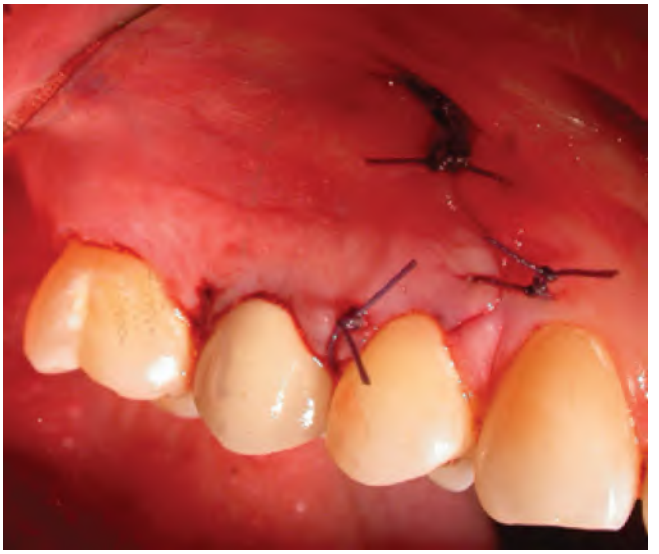

FIGURE 24-42 The single interrupted sutures have been used for closure and stabilization of horizontal and vertical incisions of a full mucoperiosteal flap on a maxillary second premolar.

in order to provide sufficient tissue to minimize suture tear-out. The needle should then enter under the surface of the mucoperiosteum of the dependent (immovable) tissue and penetrate through the mucoperiosteum at a point 2 to 3 mm from the incision margin. In order to accomplish this, it is often necessary to elevate the attached mucoperiosteum from the underlying bone for a distance of a few millimeters at the point of needle insertion. It is important that the periosteum is included with the tissue bite, otherwise the suture will most likely tear out of the fragile attached gingiva.

After the suture needle has been passed through the mucoperiosteum on both sides of the incision, the suture material should be drawn through the tissue until the end opposite the needle is approximately one to two inches from the tissue. The suture should be tied with a secure knot. A surgical knot is the most effective and least likely to slip. The surgeon's knot is best tied by wrapping double loops or throws of the long end (end with the needle attached) of the suture around the needle holder. By then grasping the short end of the suture with the needle holder and slipping the throws off, the first half of the surgical knot is tied. After adjusting the tissue tension, the second half of the knot is tied by repeating the same process, only wrapping the loops of suture around the needle holder in the opposite direction from the first tie, like a square knot. Suture knots should be placed to the side of the incision. Suture knots collect food, plaque, and bacteria, thus resulting in localized infection and a delay in healing when placed directly over the incision.

INTERRUPTED LOOP (INTERDENTAL) SUTURE

The interrupted loop, or interdental suture, is used primarily to secure and stabilize the horizontal component of full mucoperiosteal flaps. The surgical needle is inserted through the buccal or facial interdental papillae, then through the lingual interdental papillae, and then back through the interdental embrasure. It is tied on the buccal or facial surface of the attached gingiva. This suture technique highly predisposes the fragile interdental tissue to inflammation and retarded healing, resulting in a loss of the outer gingival epithelium, with possible blunting or formation of double papillae.

A modification of the interrupted loop suture described above is as follows. After the surgical needle has been passed through the buccal and lingual papillary gingiva, the suture is passed over the interdental contact and secured with a surgeon's knot. This modification eliminates the presence of suture material in the interdental embrasure, thus reducing post surgical inflammation to this delicate tissue. In clinical situations where the horizontal component of a full mucoperiosteal flap involves teeth with full coverage crowns, this modification allows for a slight incisal or occlusal repositioning of the mucoperiosteal flap. This can be accomplished by placing slight tension on the suture over the interdental contact and may compensate for a loss of gingival height resulting from the sulcular incision.

VERTICAL MATTRESS SUTURE

The vertical mattress suture has the advantage that it does not require needle penetration or suture material being passed through tissue involved in the incisional wound (Figure 24-43). The surgical needle enters and exits the flapped mucoperiosteum some distance apical from the incision line. The suture is then passed through the interdental embrasure, directed to the lingual of the adjacent tooth and passed back through the opposite interdental embrasure. The needle then enters and exits the flapped mucoperiosteum again and is passed through the embrasure, lingual to the tooth and through the opposite interdental embrasure to be tied on the buccal surface with a surgeon's knot. This suture technique also provides for the opportunity to return the flapped tissue

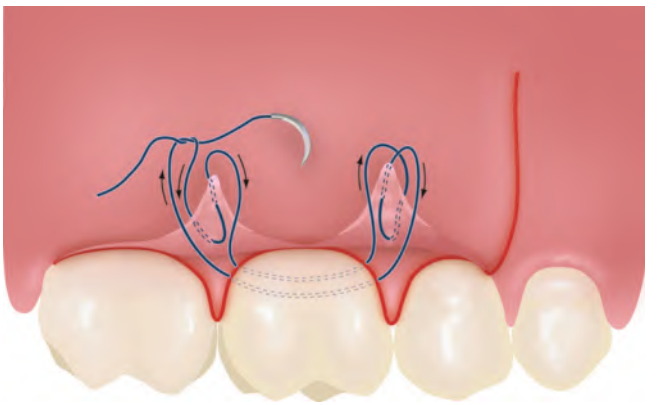

FIGURE 24-43 A vertical mattress suture avoids penetration of tissues in the incisional wound and it supports the interdental papilla in a coronal direction.

to a slightly incisal or occlusal position from its original in order to compensate for a loss of gingival height.

SINGLE SLING SUTURE

The single sling suture is similar to the vertical mattress suture (Figure 24-42). The surgical needle is passed through the attached gingiva of the flap, through the interdental embrasure but not through the lingual soft tissue. It is then directed lingual to the tooth and passed through the opposite interdental embrasure and over the incisal or occlusal margin of the flap. The needle is then passed through the attached gingiva of the flap, from the buccal or facial, back through the embrasure, passed lingual to the tooth, through the opposite embrasure, passed over the flap margin, and tied with a surgeon's knot (Figure 44A and B). This suture technique is particularly effective for achieving the maximum incisal or occlusal level when repositioning the flap. Because the lingual anchor is the lingual surface of the tooth and not the fragile lingual tissue, tension can be placed on the flapped tissue to adjust the height of the flap margin.

POST-OPERATIVE CARE AND INSTRUCTIONS

All patients who undergo PAS should receive both oral and written postoperative care and instructions. The patients should be informed of the possibility of postoperative swelling and discomfort. To reduce postoperative swelling and discomfort, the patient should use an ice pack with moderate pressure on the outside of the face (20 min on, 5 min off) for a few hours after the surgery. Mild analgesics such as ibuprofen should be prescribed for relief of post-operative pain. Some oozing of blood is expected after PAS. To reduce minor bleeding, the patient should place a moistened gauze pad over the area and put finger pressure on it for 15 minutes. The patients should be warned against lifting their lips and should check the surgical site. This may dislodge and untie the sutures. Twenty-four hours after surgery the patients can use warm salt water after each meal. Gentle brushing is recommended 24 hours after the surgery. Drinking lots of fluid and eating soft foods that contain high levels of protein are recommended. Patients who smoke should refrain from smoking for at least three days after the surgery. Excessive bleeding, pain, swelling, or a high temperature should be reported to the operator immediately. Sutures should be removed three to seven days after PAS.

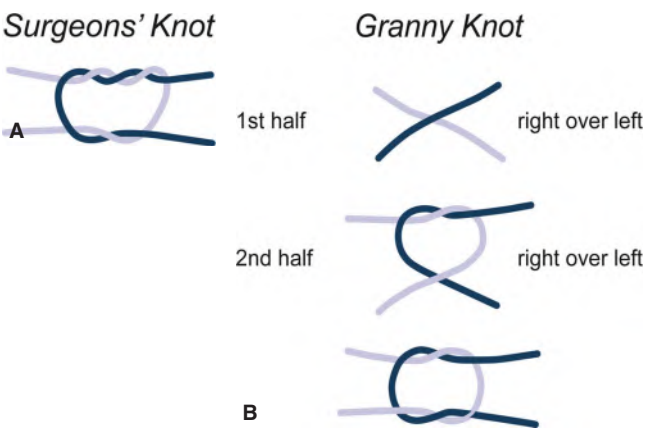

FIGURE 24-44 ***A.*** Surgeon's knot is a modified square knot in which the first overhand knot is doubled; the doubling prevents slippage especially when flaps are under tension. ***B.*** Granny knots result when a throw (complete twist of two strands) is followed by another throw in the same sequence. Granny knots result in knot slippage with less security than a surgeon's knot.

HEALING

Soft and hard tissues are manipulated during periapical surgery. Depending on the location and procedure involved, incisional, dissectional, and/or excision wounds are created during PAS.

Soft Tissue Healing

The soft tissue healing consists of clotting, inflammation, epithelialization, and connective tissue healing, as well as maturation and remodeling.[77] The main event during clotting is the conversion of fibrinogen to fibrin. The clot formation should be limited between the edges of the soft tissue incisions. The clot forming between the soft tissue wound and underlying hard tissues should be thin and minimal. Pressing the soft tissue flap against the underlying hard tissues with a wet piece of gauze usually prevents thick clot formation. Failure of the clot to form results in leakage of blood into the wound site and causes delayed wound healing. Acute and chronic inflammatory reactions occur after surgery and are part of the healing process.[77]

The soft tissue healing consists of formation of the epithelial bridge between the edges of the soft tissue incisions and the underlying connective tissue. Surface epithelial cells migrate on the fibrin surface until they make contact with their counterparts from the opposite border of the wound, forming an epithelial bridge.

The connective tissue under the epithelial layer and in contact with the underlying hard tissues contains all of the normal elements found in any connective tissue in the body. As healing progresses, there is a decrease in the amount of inflammation and maturation of connective tissue.[77,98]

Hard Tissue Healing

Harrison and Jurosky examined the healing of osseous incisional wounds in monkeys.[171] Their findings show that the hard tissue healing starts by the proliferation of endosteal cells into the wound coagulum. At about 12 to 14 days, woven bone appears at the site of surgery. The healing process occurs from inside to outside, ending in the formation of mature lamellar bone.

OUTCOME OF SURGICAL ENDODONTICS

Mead et al.[172] searched (electronically and manually) the literature related to the success and failure of periapical surgery since 1970 and assigned levels of evidence to these studies. This search located 79 clinical studies. Their research found seven low-level RCTs (LOE-2), four cohort studies (LOE-4), and 12 case control studies (LOE-3). Fifty-six studies were classified as LOE-4 case series. They reported that the majority of frequently quoted "success and failure" studies were case series. Rubinstein and Kim,[173] in a prospective study, examined the one-year healing rate of surgical endodontics performed under the surgical operating microscope (SOM) in conjunction with microsurgical techniques and reported a 96.8% success rate. A follow-up of the same cases showed a 91.5% success rate after five to seven years.[174] Maddalone and Gagliani[175] studied the outcome of periapical surgery using microsurgical technology and ultrasonic root-end preparation and reported a similarly high success rate. Torabinejad and Rubenstein[9] in 2004 discussed important advances in surgical endodontics and the impact of microsurgical techniques and new root-end filling materials on retaining teeth that may otherwise be extracted. Sechrist et al.[176] examined the success rate of surgical endodontics in 25 patients whose root-end cavities had been filled with MTA. Based on their findings, they reported that 93% of these cases were functional and asymptomatic. Kim and Kratchman[58] published an article in 2006 discussing the advantages of modern microsurgical techniques such as ease of distinguishing root apex from bone, the ability of the operator to make a smaller osteotomy, better hemostasis control, and ergonomic benefits to the operator. In this article, the authors state that "on the basis of published research, [they] believe that endodontic microsurgery with MTA is a predictable procedure to save teeth."

In a systematic review in 2009, Torabinejad et al.[7] compared the clinical and radiographic outcomes of nonsurgical retreatment with those of surgical endodontics. They reported a significantly higher success rate for traditional surgical endodontics at two to four years (77.8%) compared with nonsurgical retreatment for the same follow-up period (70.9%; $P < .05$). At four to six years, however, this relationship was reversed, with nonsurgical retreatment showing a higher success rate (83.0%) compared with surgical endodontics (71.8%; $P < .05$).

In a meta-analysis of the literature, Seltzer et al.[177] compared the outcomes of traditional surgical endodontics to current endodontic microsurgery techniques and materials and reported a 94% success rate with endodontic microsurgery compared to a 59% success rate when using older techniques and materials. In a recent systematic review, Torabinejad et al. compared the outcomes of tooth retention through endodontic microsurgery to tooth replacement using a single implant.[178] Their findings show that teeth treated by microsurgical technique had survival rates of 94% at two to four years and 88% at four to six years, indicating that microsurgically-treated teeth tend to be lost at low rates over time (Table 24-2). The outcomes of surgical endodontics have significantly improved because of the use of the microscope and new root-end filling materials such as MTA and other bioceramics.

TABLE 24-2 Survival Rates of Microsurgery Between Two to Four and Four to Six Years[178]

Author (Year)	Survival rate (%)
Chong et al. (2003)	95
Taschieri et al. (2008)	94
Taschieri et al. (2008)	93
Taschieri et al. (2011)	91
Overall % (2–4 years)	*94*
Von Arex et al. (2012)	87
Overall % (4–6 years)	*88*

CORRECTIVE SURGERY

Corrective surgery can be defined as the surgical procedure required to repair defects that occur in root or furcation areas as a result of therapeutic misadventures or pathologic processes. These defects are located in areas of the root other than the apex and are not amenable to nonsurgical repair. When a periodontal pocket is associated with a defect located in the cervical one-half of the root, it will be necessary to reflect a mucoperiosteal flap to ensure proper repair of the root defect and correction of the periodontal defect. Furcation defects are generally repaired nonsurgically, but there are situations where a periodontal component is present and can only be corrected via a surgical approach. Therapeutic misadventures requiring corrective procedures include, but are not limited to, perforations that occur during complicated endodontic access, root canal location and preparation procedures, or as a result of misdirection of burs during the preparation of post space. Pathological processes that may cause some of these defects include caries, periodontal lesions, external resorption, and perforating internal resorption. Traumatic injuries that result in fractures of the root may require a surgical approach to treatment in order to retain the remaining root or combination of root and crown structures.

Corrective surgical procedures include periapical surgery where access is gained through a flap procedure to repair the root and periodontal defects. Root resection or tooth-root resection is also a possibility when a defect can only be corrected if an entire root of a multi-rooted tooth is removed and the remaining crown of the tooth is re-contoured and retained.

In mandibular molars, a hemisection procedure may be required to remove the entire crown and root on the side of the tooth where the defect is present. A tooth replantation or transplantation procedure is only a treatment option when all other nonsurgical and surgical procedures have already been attempted and failed or have been deemed impossible to perform. In these cases, the tooth is extracted, the defect is repaired, and the tooth is repositioned back into its original socket.

PERFORATION REPAIR

Perforations in the floor of the pulp chamber in multi-rooted teeth may occur during endodontic access preparation, post space preparation, or in conjunction with extensive caries or resorption lesions. The multi-rooted teeth with the greatest potential for furcation perforations are the maxillary and mandibular molars. When a perforation occurs in this area of the tooth, the initial attempt at repair should be from an internal, nonsurgical approach (Figure 24-45). Corrective surgery is reserved for those teeth where nonsurgical repair is not a treatment option or the attempted nonsurgical repair has failed. When surgery is necessary, a buccal mucoperiosteal flap is reflected, the furcation bony defect is curetted to remove any pathologic tissue, and the perforation site is repaired.

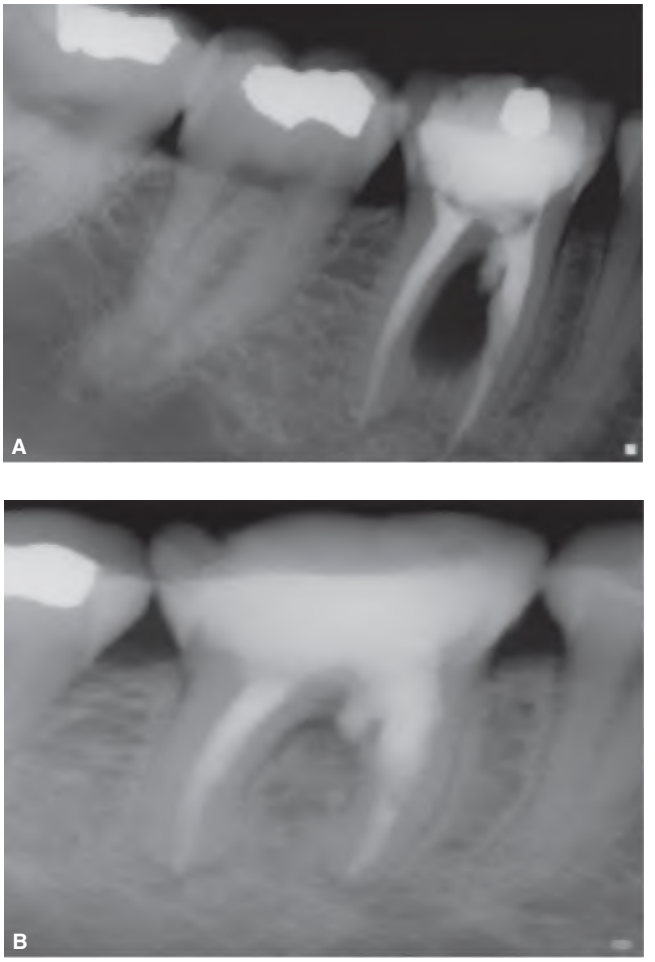

FIGURE 24-45 ***A.*** A strip perforation that occurred during cleaning and shaping was repaired internally with MTA. ***B.*** Radiograph taken 9 months later shows no evidence of pathosis in the repaired area. (Courtesy of Dr. Albert Goerig, Olympia, Washington, U.S.A.)

In an animal study, Dean et al.[179] reported that when the perforation defect was repaired surgically, the associated bone defect was filled with decalcified freeze-dried bone, and the window in the bone was covered with a periodontal membrane, the percentage of osseous healing was 75% to 100%. In those cases where the perforation was repaired, the bone defect was allowed to fill with blood, and the bone window was covered with a periodontal membrane, the percentage of osseous healing was 92% to 100%. When only the perforation was repaired and the other two procedures were not done, the osseous healing was only 52% to 70%. In the remaining three groups, the same blood clot, membrane, and osseous fill were performed on the bone defect, but none of the perforations in these three groups was repaired. The amount of osseous repair for these three groups ranged from 0% to 15%. The results suggest that, in this particular model, repair and sealing of the perforation site were necessary to achieve significant osseous healing.

Strip perforations in the cervical one-third of the root occur most frequently in the thin distal aspects of the mesial roots of mandibular molars and the mesiobuccal roots of maxillary molars (Figure 24-46A). Nonsurgical repair should be the first treatment option in these cases. If surgical repair is deemed necessary, the perforation must be accessed and visualized through a window created in the buccal bone (Figure 24-46B–D). In many cases, surgical repair will be very difficult, if not impossible, and the bony window may be so large that a periodontal defect is created. If neither the nonsurgical nor surgical options is feasible, other possible treatment options include root resection, hemisection, tooth replantation, or extraction followed by the placement of a bridge or osseointegrated implant.

If an external cervical root defect is present as a result of root caries or cervical root resorption, the treatment approach may be different from that proposed for a perforation defect in the same area. If the carious or resorptive defect does not penetrate into the pulp canal space, a surgical approach to treatment is the first choice. A limited envelope soft tissue flap is generally adequate to visualize, cleanse, and repair the affected area. If the defect is in a cosmetic area, an appropriate composite resin or glass ionomer material will be the material of choice. MTA is the material of choice if the defect is in an area where cosmetics are not an issue. Periodontal pocketing is often associated with these cervical defects, so crown lengthening or vertical root extrusion may also have to be performed in order for the area to be properly restored and cleansed.

Perforations occurring at the mid-root level are usually the result of burs being misdirected or over-thinning of the root canal walls during post space preparation. If possible, these defects should be immediately sealed via an internal approach. If surgery is necessary (Figure 24-47), the same

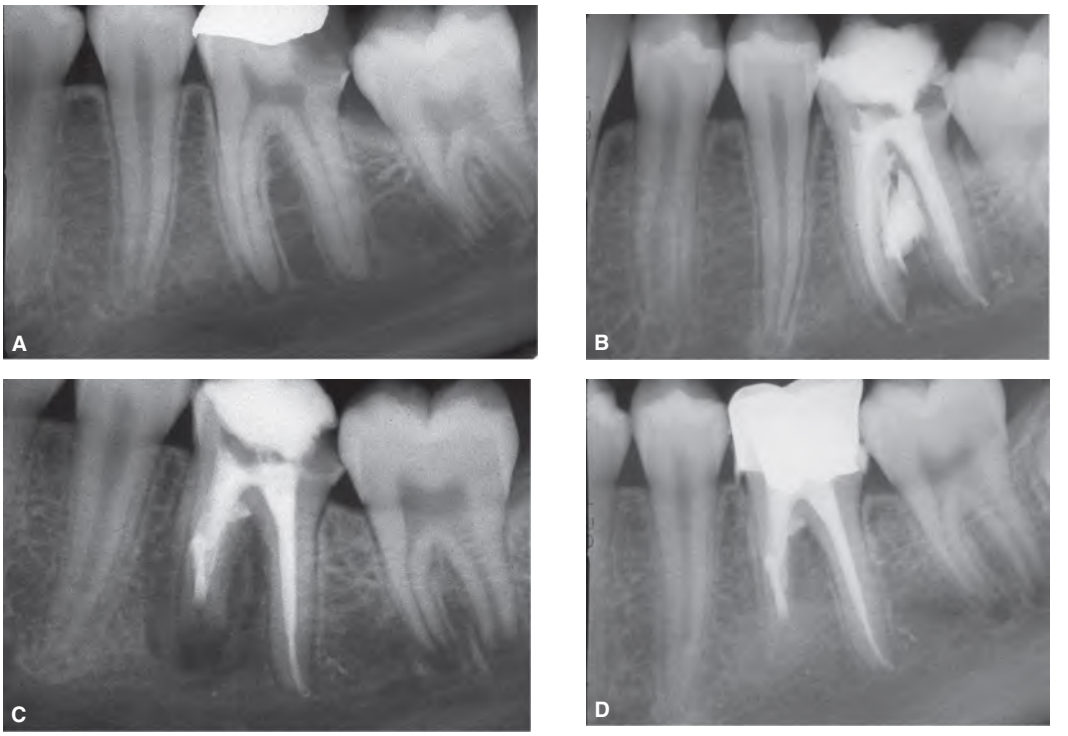

FIGURE 24-46 ***A.*** A preoperative radiograph of the first mandibular molar shows presence of a thin distal walls of the mesial roots. ***B.*** A severe strip perforation occurs during cleaning and shaping of the root canals along with extrusion of root canal filling materials into the periapical tissues. ***C.*** The perforation was repaired with MTA. ***D.*** A radiograph taken one year later shows healing of the repaired area.

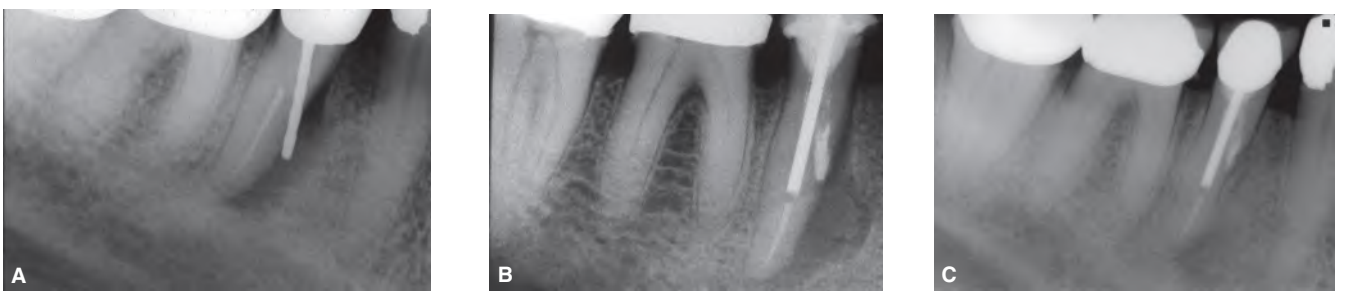

FIGURE 24-47 ***A.*** An off-center post has perforated the root and caused a bony lesion. ***B.*** Internal and external perforation repairs with MTA. ***C.*** Three years later, full resolution of the bony lesion is evident. (Courtesy of Dr. N. Chivian, West Orange, New Jersey, U.S.A.)

steps are followed as for the other areas that require surgical repair. If there is cortical bone covering the root surface coronal to the level of the mid-root repair site, there will be less chance of creating a chronic periodontal problem. A perforation located in the apical third of the root is treated surgically by removing that portion of the root apical to the perforation site and, if necessary, placement of a root-end filling.

The management of external root resorption lesions located apical to the cervical area will depend on whether the defect communicates or does not communicate with the pulp canal space. Those lesions that do not communicate with the pulp canal space will need to be corrected surgically in order to arrest the active resorptive process. The location of the resorptive defect on the root surface will be a key factor in determining if a successful surgical repair can be accomplished. If the lesion is located on the distal or the lingual surface of the root, it may be impossible to visualize and correct the defect from a surgical approach. In these situations, tooth replantation for a single-rooted tooth, root resection, or hemisection may be the only treatment choices if an attempt is to be made to retain the tooth. Otherwise, extraction may be the only option.

When these external resorptive defects are approached surgically, a full thickness mucoperiosteal flap is reflected, the bony window over the defect site is enlarged for visualization, the resorptive defect is curetted, and the site is repaired with MTA. If there is no bone covering the root surface coronal to the defect, a periodontal regenerative procedure may also be performed in conjunction with the corrective surgical procedure.

When an external root resorptive defect communicates with the pulp canal space or an internal resorptive defect communicates with the external root surface, a combination nonsurgical and surgical approach to treatment may be required. The first step in either of these situations is to cleanse and shape the root canal system and place an internal dressing of calcium hydroxide. At a subsequent visit, the calcium hydroxide is removed, the root canal is dried, and that portion of the root canal system apical to the perforating defect is obturated with an appropriate obturating material. The external portion of perforation defect is then packed with a matrix/barrier material, and the repair site is repaired internally. If it is impossible to adequately dry the root canal system so that root canal obturation can be performed or the external resorptive process remains active, then a combination treatment must be performed.

With a combination treatment, a mucoperiosteal soft tissue flap is reflected, the pathologic defect on the root surface is curetted, the root canal space can then be dried, and the portion of the root canal space apical to the defect can be obturated. The perforation defect on the root surface and into the root canal space is then repaired with an appropriate repair material. Another technique that has been suggested is to place a tight-fitting gutta-percha cone into the root canal space without sealer, repair the external defect, let the repair material set, and then remove the gutta-percha cone and proceed with the internal root canal obturation procedure. The inherent danger with this latter technique is that the compacting forces required to perform the obturation procedure may displace the external repair material. Once the soft tissue flap is sutured, the access opening is closed with a temporary sealing material. Any additional nonsurgical treatment can be completed at a subsequent visit after the soft tissue flap has healed. If there is an extensive bone loss over the external root surface, a periodontal regenerative procedure may also be required. If the defect is found to be so extensive that the root cannot be retained, one of the alternative treatment options noted above must be considered.

ROOT RESECTION

Root resection is the removal of one or more roots from a multi-rooted tooth (Figure 24-48). It is usually performed in maxillary molars, but can also be performed for mandibular molars. The indications for this procedure are presence of severe bone loss in a periodontally-involved root, untreatable roots with broken instrument, perforations, caries, resorption, vertical root fracture, and calcified canals in a multi-rooted tooth. The contraindications for root amputation are absence of sufficient bony support for the remaining root or roots as well as fused roots. After raising a sulcular flap without a vertical-releasing incision, root resection is performed by making a horizontal cut to separate the root from the crown. The crown remains intact and the remaining stump is gradually shaved to the buccal aspect of the root, resulting in good anatomic contour and hygiene.

HEMISECTION

Hemisection is the surgical division of a multi-rooted tooth into two segments (Figure 24-49). It is usually performed in mandibular molars, but can also be performed for maxillary molars. The indications and contraindications for this procedure are very similar to those for root resection. In mandibular molars, the tooth is divided buccolingually through the bifurcation. In maxillary molars, the tooth is sectioned mesiodistally through the furcation. After raising a sulcular flap without a vertical-releasing incision, hemisection is usually

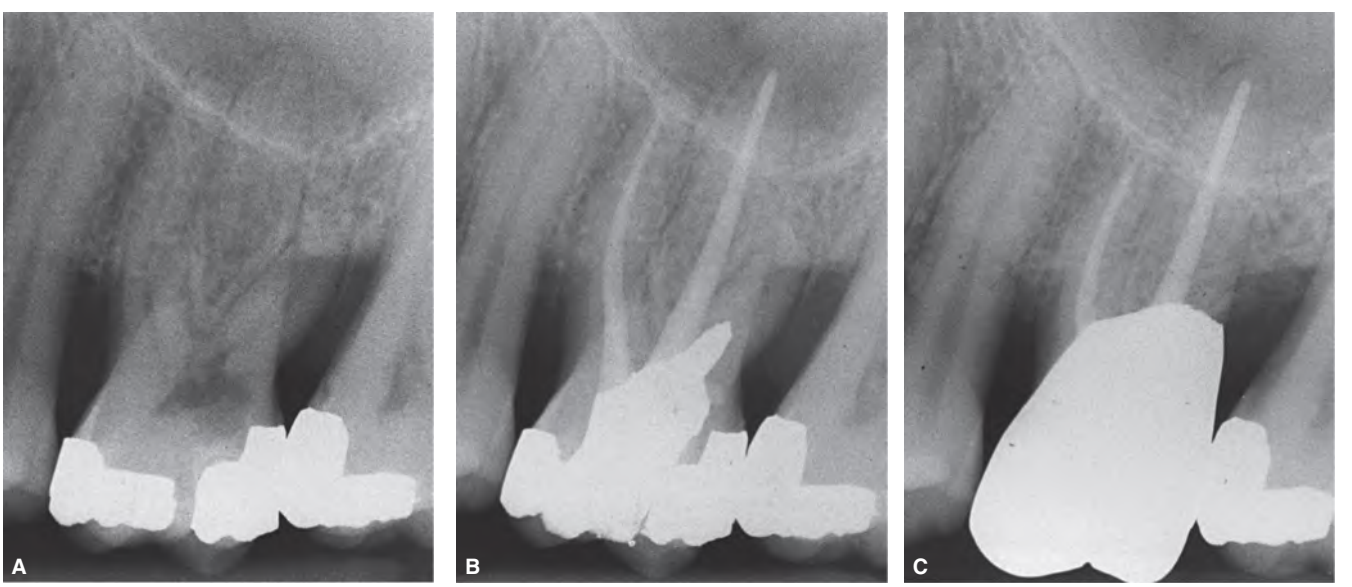

FIGURE 24-48 ***A.*** A severe bone loss around the distobuccal root of the first maxillary molar was noted. ***B.*** Root canal treatment was followed by an amalgam core extending 4 mm into the distobuccal canal. ***C.*** The root was resected and a crown subsequently placed.

carried out by making a vertical cut through the crown into the furcation. This action results in complete separation of the tooth in two segments. The unsalvageable root and its coronal segment are then removed (Figure 24-49).

BICUSPIDIZATION

This procedure is usually performed on a mandibular molar, making two bicuspids out of a molar that has severe bone loss or destruction in its furcation area (Figure 24-50). The indications for bicuspidization of mandibular molars are the presence of irreparable furcation perforation, furcation pathosis from a periodontal abscess, presence of buccolingual cervical caries, or a fracture in the furcation. The contraindications for bicuspidization are the presence of deep furcation, unrestorable crowns, absence of adequate bony support for roots as well as fused roots and presence of fused roots. After raising a buccolingual sulcular flap without a vertical-releasing incision, bicuspidization is performed by making a vertical cut through the crown into the furcation using a fissure bur. This procedure results in complete separation of the roots and creation of two separate segments similar to two premolars after restoration.

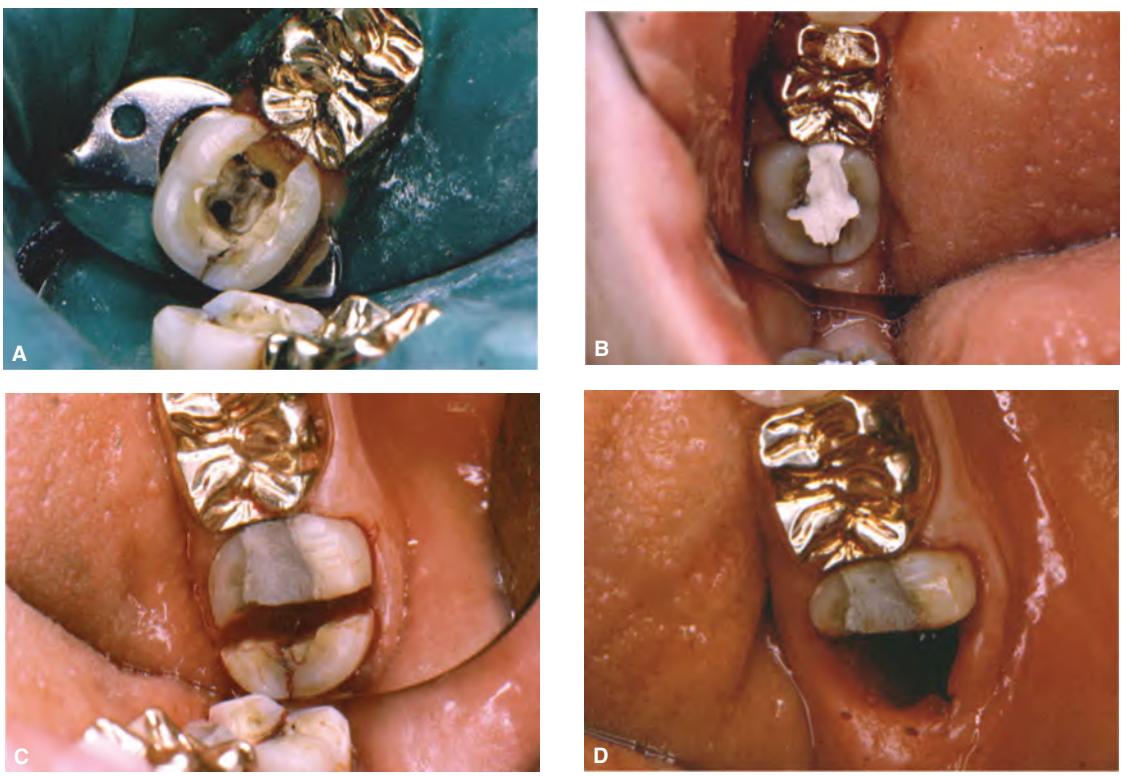

FIGURE 24-49 ***A.*** A mandibular second molar has a fracture across the distal marginal ridge extending to the midroot level. ***B.*** An amalgam core has been placed. ***C.*** A hemisection procedure has been completed and the distal root is ready for extraction. ***D.*** The mesial half of the tooth has been retained as a bicuspid to form an occlusal stop to prevent the extrusion of the opposing maxillary molar.

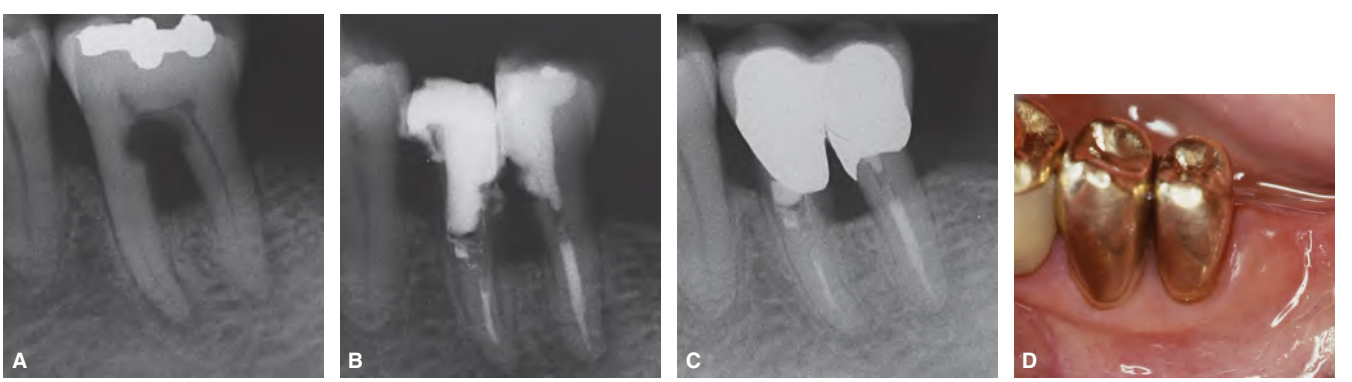

FIGURE 24-50 ***A.*** Caries in the furcation and furcal bone loss are evident in the mandibular first molar. There is adequate bony support for both roots. ***B.*** Root canal treatment and bur separation through furcation and crown. ***C.*** Restoration with a porcelain fused to metal crown splinting the two roots. ***D.*** Good gingival response at 30-month recall with no probing defects.

PROGNOSIS

Prognosis of teeth that have had root amputation, hemi section for bicuspidization varies according to the situation. If these procedures are performed correctly and the tooth is restored properly, the major factor affecting success is the patient's oral hygiene. The patient must be willing and able to perform extra procedures to prevent plaque accumulation. Failure to do so will result in the failure of the procedure.

TOOTH REPLANTATION

Tooth replantation involves extracting a tooth, correcting its deficiencies outside of the mouth, and replanting it into its original socket (Figure 24-51)[181]. The indication for this procedure is when an apical surgery is contraindicated as a result of patient medical conditions, proximity of anatomic structures such as the mental foramen or mandibular canal to the surgical site, and/or presence of thick bone. When properly performed this procedure is very successful. Torabinejad et al. compared the survival rates of replanted teeth with those of implant-supported single crowns.[180] Meta-analysis of current data revealed a weighted mean survival rate of 88% (95% CI, 81%–94%) for intentionally replanted teeth. Root resorption occurred in 11% of these cases. For more details, see Chapter 25.

TRANSPLANTATION

Transplantation is defined by extraction of an erupted, embedded, or impacted tooth from one part of the mouth into

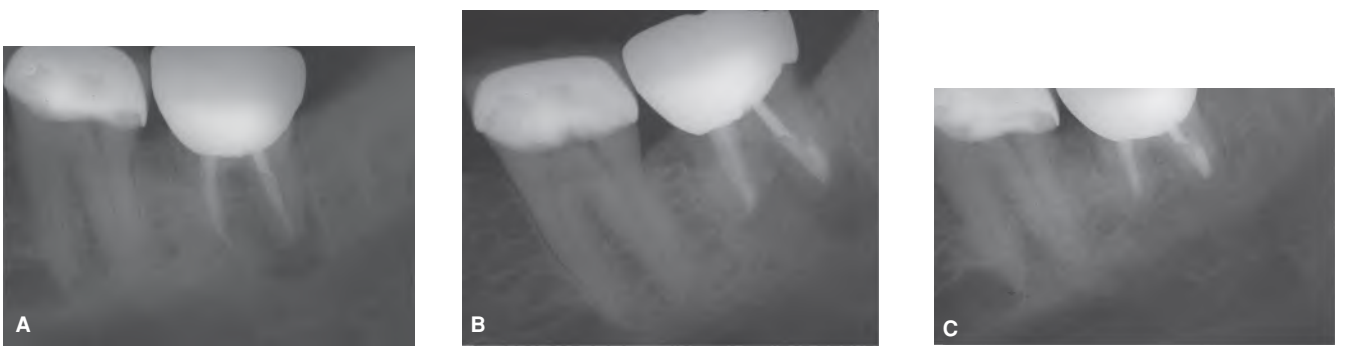

FIGURE 24-51 ***A.*** Preoperative radiograph of a second mandibular molar associated with symptomatic apical periodontitis. ***B.*** Postoperative radiograph of the tooth after replantation procedure. ***C.*** A follow-up radiograph two years after the procedure shows complete resolution of the periapical pathosis.

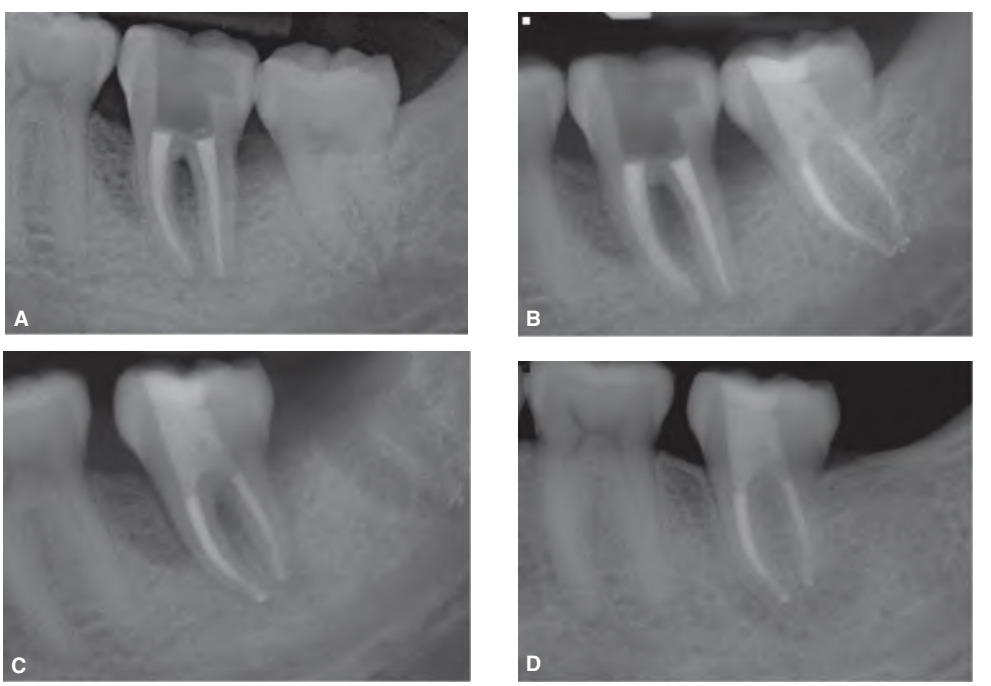

FIGURE 24-52 ***A.*** Preoperative radiograph of a second mandibular molar shows presence of symptomatic apical periodontitis and sever bone loss. ***B.*** Postoperative radiograph of the tooth after root canal treatment of the third molar. ***C.*** After auto transplantation of the third molar into the socket of second molar. ***D.*** A radiograph taken 46 months later shows complete healing of the periapical tissues around this tooth.

an extraction site or surgically prepared recipient site within the same individual (Figure 24-52).[181] Transplantation of a tooth has long been an acceptable treatment and is indicated for either a nonsalvageable or missing tooth.[182–184] Transplantation is contraindicated if the transplanted tooth is badly broken down, has inadequate bony support, is difficult to extract, and does not fit the extraction site. After extracting the unsalvageable tooth and preparing the socket for transplantation of a new tooth, the new tooth is extracted atraumatically with minimal damage to the periodontium. Its roots are resected, and Class I cavity preparations are prepared and filled with a root-end filling material outside of the mouth. The tooth is then replanted into its new socket. During the latter procedures, the tooth must be kept in moist gauze to prevent dehydration and necrosis of the periodontal ligament. When appropriately indicated and performed, autotransplanted teeth have a good prognosis.[184] Ankylosis and resorption are the most common causes of this procedure. Torabinejad et al.[181] compared the survival rates of autotransplanted teeth with those of implant-supported single crowns.[181] Meta-analysis of available data showed a weighted mean survival rate of 89% (95% CI, 81%–94%) for autotransplanted teeth.

For updated information, the readers are referred to Torabinejad and Rubinstein[185] and Rubinstein, Fayad, and Torabinejad.[186]

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Chapter 25

Intentional Replantation of Endodontically Treated Teeth

Stephen P. Niemczyk

The advent of the surgical operating microscope (SOM) heralded a new era in periapical surgery. In conjunction with innovative root-end preparation devices and root-end filling materials with enhanced biocompatibility, the specialty experienced a *renaissance* of possibilities previously unimagined. Difficult scenarios were somewhat less daunting, and the degree of precision offered more optimistic prognoses in most cases. Indeed, contemporary studies[1,2] demonstrate a sharp contrast of success rates when compared to those procedures performed a few decades earlier with traditional armamentarium and methods.[3] However, there remain instances where an *in vivo* approach would be deemed contraindicated. Anatomical considerations with regard to root angulations and positions, cortical plate thicknesses, and arch position may compromise the access and reflection of the selected site and jeopardize the surgical management of the root end. Preexisting conditions, either natural or iatrogenically induced, might exacerbate the situation; in this case a conventional surgical approach would be ill advised. Even with local factors notwithstanding, the physical or psychological health of the patient may contraindicate a protracted surgical intervention. It is in these clinical circumstances that the technique of intentional replantation (IR) represents a viable treatment solution.

References to the splinting and adornments of teeth can be found in the ancient Mayan and Egyptian cultures; however, the earliest recorded instructions detailing the extraction and replantation of teeth may be attributed to Abul Kasim (known as Albucasis in the West), the renowned Arabic surgeon of the tenth century. His treatise *Al-Tasrif* (*The Method*) was later translated into Latin as *De chirurgia*, distinguishing him as the first Oral Surgeon of note.[4] Several hundred years later the renowned French dentist, Pierre Fauchard, completed his epic work, *Le chirurgien dentist; ou, traité des dents* (The Surgeon-Dentist; or, Treatise on the Teeth), in which he describes the replantation and transplantation of teeth from one individual to another.[5] Intra-operative treatment of the tooth was both imaginative and innovative. Thomas Berdmore,[6] advocated filling the cavity of the extracted tooth with lead or gold prior to its reinsertion into the socket, and the English barber-surgeon, John Hunter,[7] proposed boiling the tooth to facilitate the removal of disease. This father of modern surgery was also a proponent of harvesting "scion" teeth (immature teeth from young individuals) for replantation in his adult patients, often having several "donors" in attendance. The teeth would be removed from sequential individuals until one, demonstrating the proper fit, could be inserted into the recipients extraction site! Although his attempts were more often than not unsuccessful, his intuitions and insights are prophetic when considered in the light of modern day medicine.

A more scientific examination of the procedure would occur almost two centuries later when assessing the role of the periodontal ligament (PDL) in re-attachment. Fredel and Scheff[8,9] noted, in their dog studies, that the absorptive (resorptive) process did not occur in teeth protected by the PDL (referred to as the periosteum). They surmised that the PDL was essential for the reunion of the tooth within its respective alveolus. In 1955, Hammer[10] reported that a viable PDL was instrumental in the re-attachment and reintegration of replanted teeth, with "an average 10 year life span expected when the replantation was accomplished in a technically flawless manner." In spite of these discoveries, the procedure was still considered either a by-product of a traumatic event[11] or a treatment option of last resort[12,13] due to the overwhelming incidence of ankyloses and root resorption. However, emerging technologies and a significant variation of the technique have minimized the two major complications of the replantation: tooth fracture and physiologic dehydration.

In its simplest terms, the IR could be thought of as a "Controlled Avulsion"; the forces exerted on the tooth, both in magnitude and direction, are more precisely regulated, and an extra-oral environment is created to maintain PDL viability. Although the level of evidence for this procedure in humans consists primarily of case reports,[14–16] controlled studies outlining the cause and effect on hard and soft tissues during the extraction and replantation are well documented in animals.[17,18] Extrapolation of these and other retrospective studies in the literature serve as treatment guidelines when establishing protocols for this procedure. Careful case selection and strict adherence to modified handling and replantation protocols will minimize the most prevalent complications of the IR: root fractures and resorptions. Finally, as with conventional microendosurgery, the IR is not meant to substitute for the nonsurgical treatment of infected canal spaces. Without adequate debridement and obturation to reduce the etiology, periapical healing may be delayed or inhibited.[19] In instances where conventional endodontic therapy has been rendered incomplete, or improvement of the existing intra-canal condition via retreatment is not attempted first, diminished success rates have been demonstrated.[20–23]

INDICATIONS AND CONTRA-INDICATIONS: TRADITIONAL AND CONTEMPORARY

Several investigators[12,24,25] set forth guidelines for IR, the majority of which were also indications for more traditional surgical corrections at that time.

Indications:

1. Unsuccessful endodontic therapy where endodontic retreatment is not feasible.
2. Failure to negotiate the canal completely, either because of calcifications or curvatures, or failure to adequately debride and obturate the canal spaces.
3. Presence of separated instruments or other iatrogenically introduced foreign objects (Figure 25-1).
4. Presence of a large post/core or other intentionally inserted prosthetic that cannot be easily removed.
5. Iatrogenic perforation or root resorption invading the canal space.
6. Gross overfill or root anatomy that is not conducive to conventional obturation methods (immature/blunderbuss apices).
7. Deciduous teeth. Here, IR is performed as a space maintenance operation (Figure 25-2).
8. Root fracture. Visual inspection can be performed, and the tooth replanted, if no fracture can be detected.
9. Presence of a large area of rarefaction, where root resection is not feasible.
10. Anatomical constraints and proximity to vital structures. This may include root apices that approximate the mandibular nerve or the maxillary sinus, and natural skeletal formations that would inhibit surgical access.
11. Patient declines an *in vivo* surgical intervention.

Several of these factors could conceivably overlap into indications for a surgical correction; the direction of the treatment would often be dependent on the skill of the operator, the degree of the correction, and the willingness of the patient.

Contra-indications:

1. Periodontal disease with marked mobility and/or furcation involvement.
2. Labial or inter-septal bone is missing.
3. Root curvatures that would precipitate fractures during the extraction.

The availability of the microscope and specialized root end instruments has dramatically expanded the realm of treatment possibilities. Therefore, a more valid listing of the indications[26] is reflected in the scenarios that are incontrovertible when contemplating an *in vivo* surgical approach, two of which persist even decades later:

1. Second/third molar root end surgery. The inclinations of the root apices, particularly of mandibular molar teeth, are often more lingual, requiring a deeper and more extensive osteotomy to uncover them.
2. Limited access due to anatomical structures, particularly skeletal. Overlying external oblique ridges and malar processes (Figure 25-3) can create retraction and access issues. Palatal approaches to the root apex in patients with a shallow vault could risk a vascular accident if misdiagnosed.
3. Medically compromised patient unable to safely endure a prolonged or involved *in vivo* approach. This is especially significant for those who are at risk due to cardiovascular disease or pulmonary compromise.
4. Patient noncompliance and refusal of a conventional surgical procedure.
5. Risk/presence of neural injury (Paresthesia).

These conditions establish boundaries for feasibility and safety from both the patient's perspective and the surgeon's preoperative assessment. Likewise, a pragmatic guide to the true contra-indications would include:

1. Severe periodontal involvement. The tooth presents with more than 2+ mobility and/or is depressible into the socket.
2. Extensive caries that is clinically detectable at or below the osseous crest.
3. Extensive loss of cortical/septal bone.
4. Difficult or impossible nonsurgical extraction.

Maintenance of the bone and other supporting/attachment structures are of paramount importance to the success of the IR procedure. Compromising the soft tissue response because of chronic periodontitis can potentially inhibit reattachment to the socket. If the level of hard tissue is diminished secondary to a crown lengthening or surgical extraction window, critical support post extraction is lost, and the replanted tooth may require more involved prosthetics to secure retention.

Case Selection and Armamentarium

Critical assessment of root anatomy of the proposed tooth is essential to affect delivery from the socket without overt stresses that could precipitate a partial or complete fracture. This is the overwhelming complication of this procedure, and is a significant condition in the informed consent. The initial appraisal is performed with radiographs from several angles, but is limited by its two-dimensional representation. A far more accurate appraisal is accomplished by obtaining a cone beam computerized tomography (CBCT).[27,28] Not only are root length and widths more accurately measured, but the degree and direction of curvatures present are immediately apparent and appreciated. For more details, see Chapter 9C.

The "Virtual Extraction"™ can be planned with regard to application of force and delivery direction, with a preview of the possible obstructions that could hinder an efficient and successful removal of the root from the socket (Figure 25-4).

Factors to consider when planning the extraction are:

1. ***Tooth (root) length/width***. The longer the root, the greater the force required to begin the luxation motion and sever the PDL. This is also true with root forms

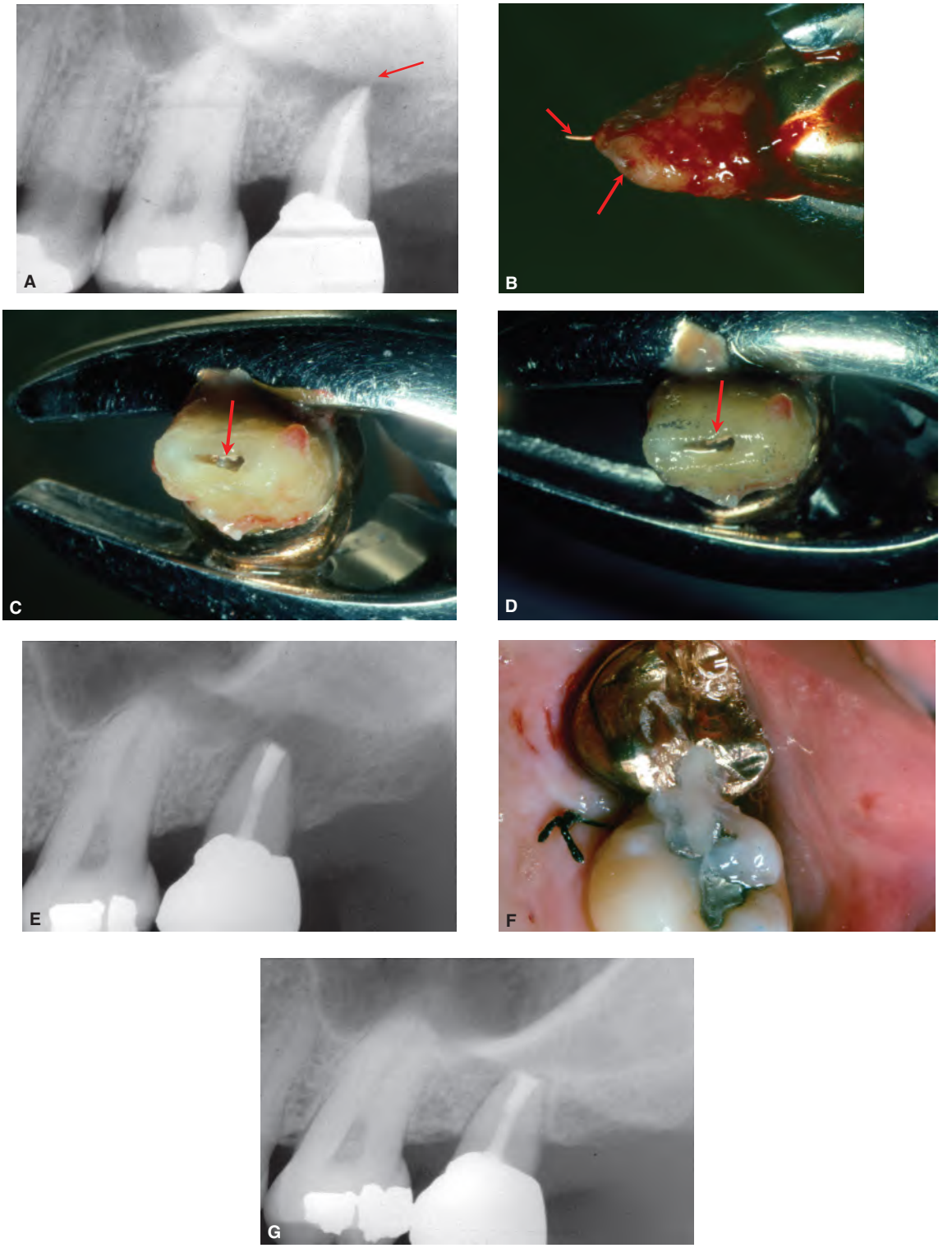
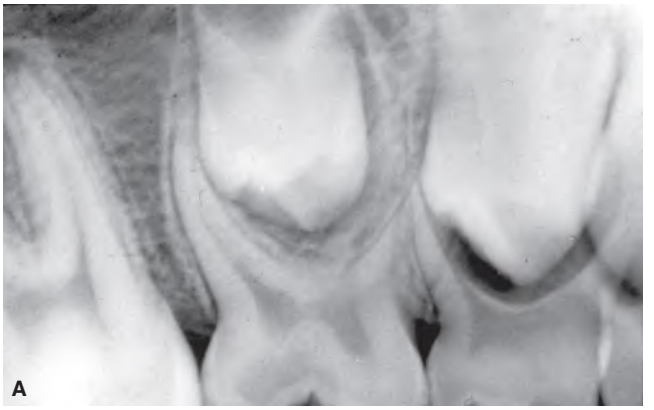
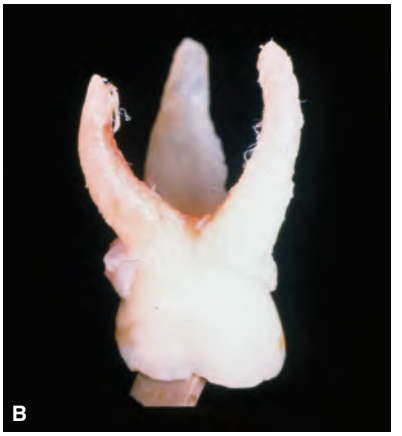


FIGURE 25-1 Intentional replantation of a maxillary left second molar with canal obstruction. ***A.*** Preoperative radiograph. A titanium carrier (Thermafil, Tulsa Dental, OK) was used to obturate the canal space, and was not retrievable. There is evidence of a small overextension of filling material (arrow), and a large periapical radiolucency. ***B.*** Extracted tooth in the forceps prior to placement in the basin. Note the overextended gutta-percha point and the titanium carrier protruding from the apical foramen (arrows). ***C.*** The root end after resection. Some of the gutta-percha has been removed from around the carrier, disclosing a large isthmus between the two canals. This "ribbon" configuration began in the pulp chamber and continued to the apical foramen (arrow). ***D.*** Root-end preparation was achieved using a small (#1) round diamond bur to sever the titanium carrier in the canal space (arrow). A conventional carbide would cause excessive vibration on the titanium, possibly fracturing the tooth. ***E.*** The tooth immediately after replantation. ***F.*** The tooth was stabilized with an acid-etched composite splint between the adjacent marginal ridges. A 4/0 silk suture coapts the mesial papilla. The splint will be removed in 14 days. ***G.*** Four-year recall radiograph showing complete healing. The tooth was asymptomatic and functional.





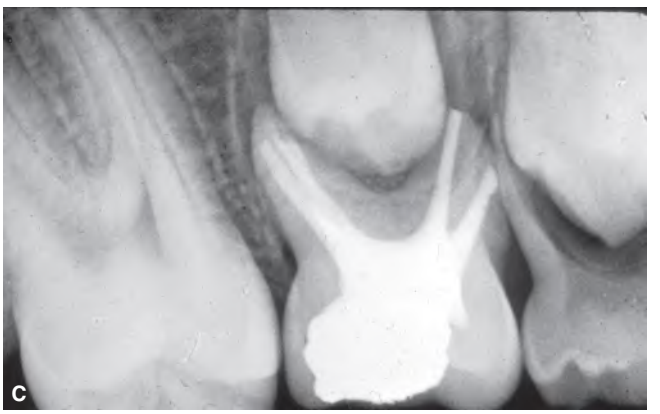
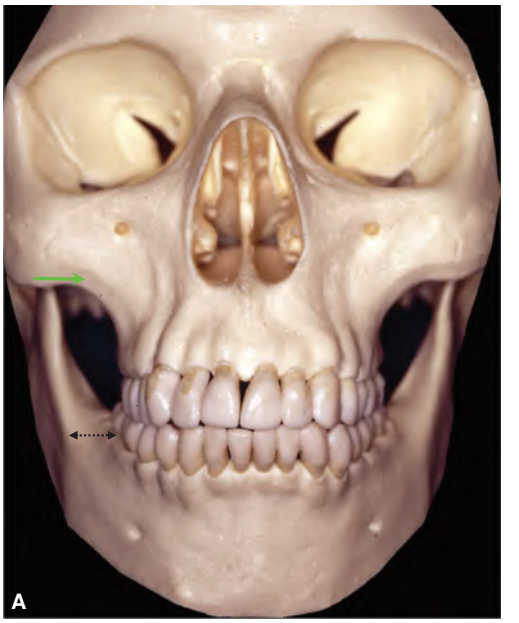


FIGURE 25-2 Intentional replantation for space maintenance in a maxillary right second deciduous molar. ***A.*** A large carious lesion compromised the pulp. The tooth is required to maintain the space for the developing permanent successor. Patient behavior prevented normal restorative treatment. ***B.*** The excess curvatures will be resected away to ease the replantation. The tooth will be accessed, and the pulp chamber and canals filled with Zinc Oxide Eugenol cement. ***C.*** The tooth has been replanted. Root resorption is common, and any materials introduced into the canal system should allow for that event. (Courtesy of Dr. Israel B. Bender, Philadelphia, PA, U.S.A.)



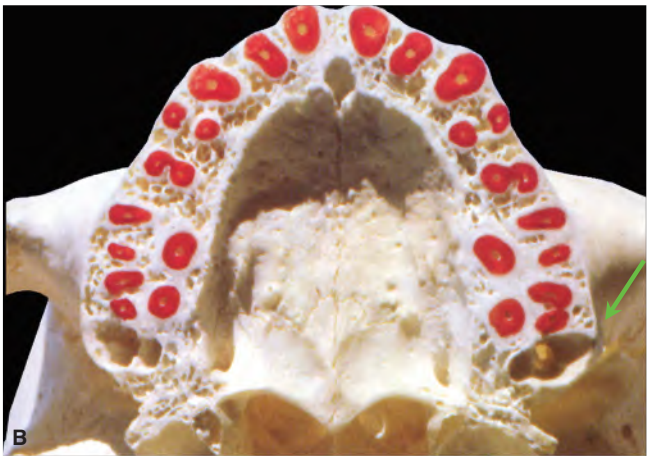


FIGURE 25-3 ***A,B.*** Adequate access to root end structures can be inhibited or prohibited by the overlying skeletal anatomy. In the mandible, the external oblique ridges (red arrows) effectively thicken the width of the buccal cortical plates from the distal root of the first molar to the third molar. This dramatically increases not only the extent of the osteotomy required to reach the root-end, but also the depth the bur must extend to complete the resection operation (black arrow). In the maxilla, the malar process of the zygomatic arch, combined with the restriction of the overlying soft tissues, presents a considerable challenge to a buccal surgical access in the second and third molar sites (green arrows).

that are wider buccal-lingually than mesial-distally. The areas of the root surface most prone to resorption postoperatively are the convex surfaces subject to these luxation motions.[29] Minimizing these forces is the key to reducing this postoperative complication. Conversely, roots that are long and thin have a greater chance of fracturing during the luxation phase of the procedure.

2. ***Root curvatures***. Although there are no "arrow straight" roots, if the curvature is gentle and, in multi-rooted teeth, if they all curve in the same direction, then a single path of draw can be directed from the socket. If the curvature is too severe, or abruptly dilacerated (Figure 25-5), the chances of separating the root at the apex of the

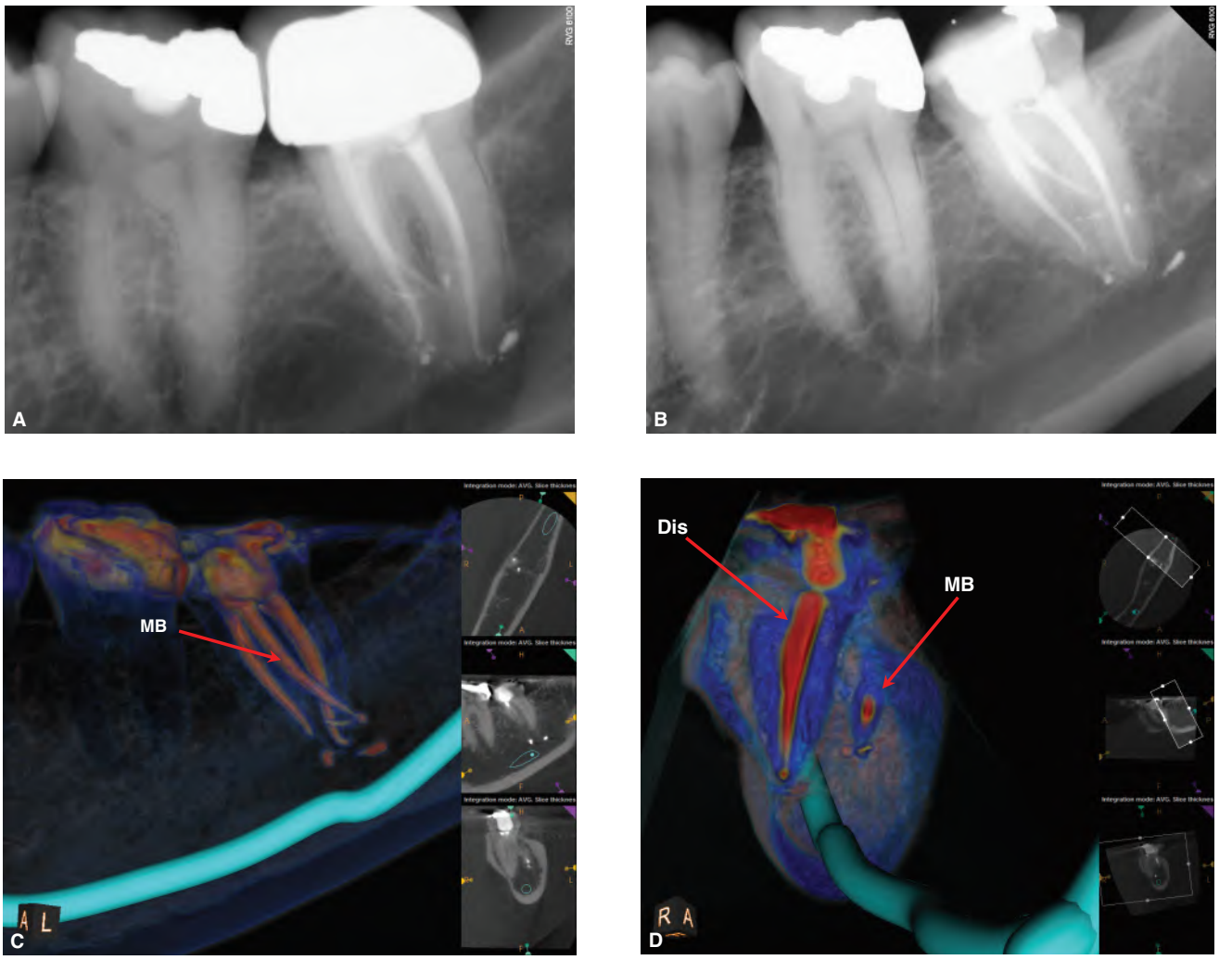


FIGURE 25-4 The inability to accurately assess the clinical environment with conventional radiographs is dramatically demonstrated in this case series. The patient presents with the chief complaint of sensitivity to biting, the onset being the completion of the endodontic treatment. A clinical examination revealed no evidence of deep periodontal probing or vertical fracture, and percussion and palpation findings were within normal limits. ***A.*** Postoperative radiograph from 2011 reveals the presence of several small extrusions of sealer from the apices of the mandibular left second molar. ***B.*** Radiograph taken prior to the patient receiving a new crown in 2014. The extruded material has not resorbed. ***C.*** The MB root is a separate root, coursing more distal and buccal than the main root form. There is no overt periapical pathology, but small extrusions of material are present. The material is not in the mandibular canal. Any foreign body is ordinarily removed via curettage, but the overlying bone thickness measures 11.5 mm to the distal root apex (nearly one-half inch). ***D.*** The excavation would be further complicated by the location of the MB root centered in the proposed osteotomy, itself 5.5 mm below the surface of the cortical plate. Access to the periapical extrusions could be realized during an IR procedure, but the atypical root anatomy presents a considerable challenge to an atraumatic extraction, and needs to be addressed during the informed consent.

curvature are high and should be anticipated. This effect is magnified in multi-rooted teeth, where divergent roots, especially in maxillary molars, hamper the safe extraction. Convergent root forms present a similar dilemma, but if the inter-radicular bone is absent, it may not pose a problem. Even if there is bone between the roots, it is a relatively small amount in terms of area. It may be spontaneously fractured during the extraction and delivered with the tooth without consequence.

3. ***Periradicular lesions and mobility***. It would seem advantageous to have some mobility associated with the selected tooth. However, if the mobility is due to absence of adequate cortical plate(s), then the tooth may be too periodontally compromised to be considered for replantation. Periapical lesions will oftentimes be attached to the extracted root, eliminating the need for periapical curettage. Radiolucent areas alongside the root surface, especially when accompanied by deep, discreet probing depths, are highly suspicious of vertical root fracture, and should be examined postextraction with great diligence until proven otherwise.

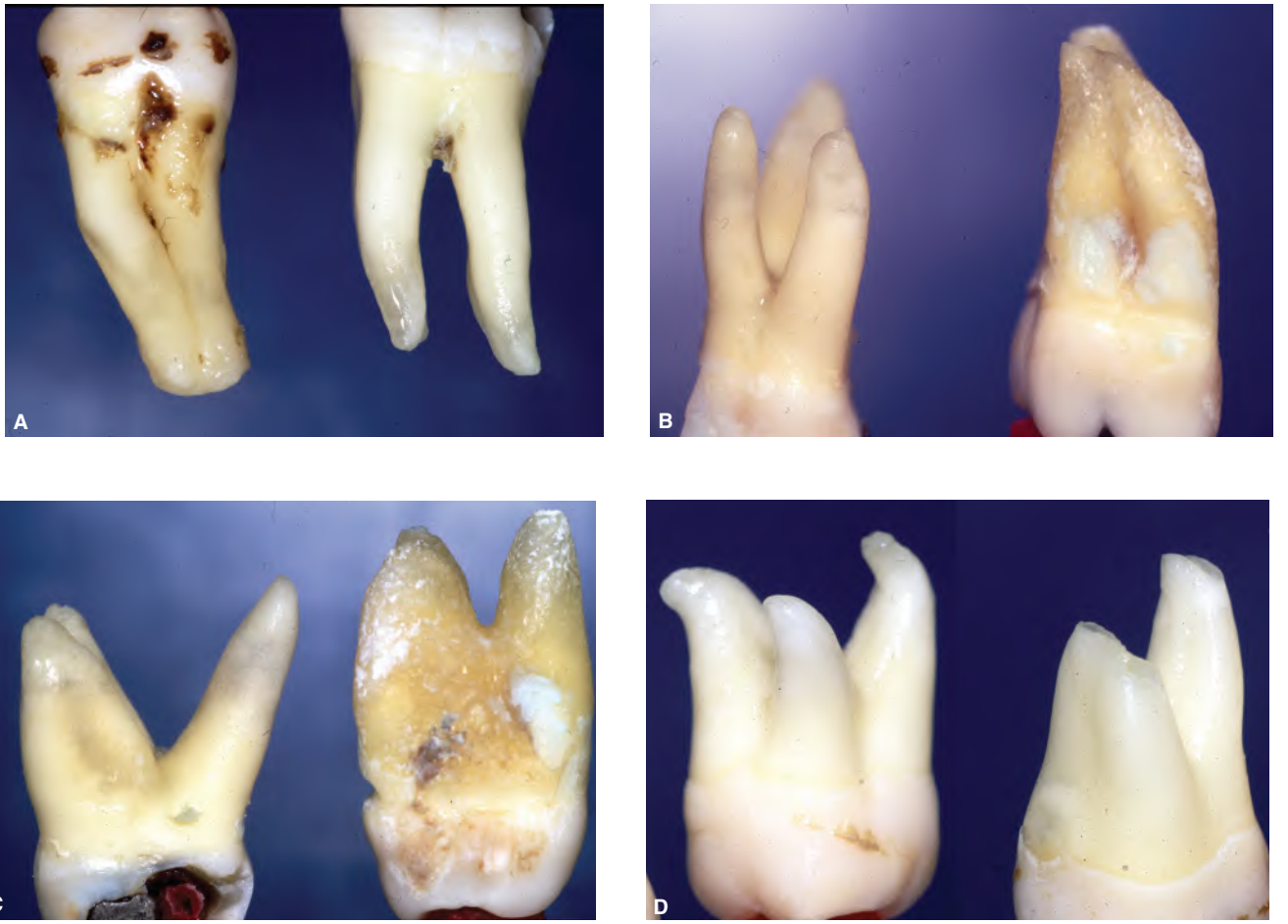


FIGURE 25-5 Root configurations and curvatures play a significant role in the success of the extraction. ***A,B.*** When the roots are fused (left tooth in A, right tooth in B), the extraction may be less complicated. Surface invaginations and concavities provide a positive index for replantation and resistance to exarticulation. The exception to this is when there is a partial fusion, with the apical segments joined, but the furcation separation is intact. In those instances, if there is significant bone through the furcation, conservative extraction without hemisecting the two roots may be impossible. ***C.*** When the root curvatures are mild, and have a similar orientation and direction, the tooth can be delivered along the appropriate path of draw. However, if the roots are widely divergent (left tooth), then fracture of either the root(s), cortical plate(s) or both is likely to occur. ***D.*** When the root-ends are severely dilacerated, fractures often occur at the angle of the dilacerations. The level of the fracture frequently coincides with the planned level of the resection (right tooth). Nevertheless, the orphaned root tip and any associated pathology must be delicately and completely harvested from the socket. Failure to do so may result in persistent periapical inflammation and incomplete replantation. Postoperative stabilization of multi-rooted teeth is generally a minor concern, even for teeth with fused root forms.

The armamentarium for IR is simple and readily available (Figures 25-6 and 25-7):

1. ***Emesis basin***. Once the tooth is delivered from the socket, it will be submerged in a basin containing a tissue culture solution. This basin should be deep enough to accommodate the forceps containing the tooth, while being compact enough so as to not be unwieldy. The stainless steel emesis basin satisfies the requirements of strength, utility, and sterility.
2. ***Nonairstream high-speed handpiece***. The Impact Air™ (Palisades Dental, South Hackensack, NJ, U.S.A.) has a liquid only stream that bathes the bur when activated. The drive air is exhausted through the back of the handpiece, minimizing the overspray during the resection of the root under the fluid surface in the basin. There are similarly configured handpieces from other manufacturers, and they are equally suitable if they satisfy the criteria outlined above.
3. ***Forceps***. The traditional extraction forceps, of the pattern appropriate for the selected tooth, were until recently the only instruments available for removal of the tooth. In 2003, the Physics Forceps™ (Golden Dental Solutions, Detroit, MI, U.S.A.) (Figure 25-7) was introduced and allows for a less traumatic delivery of the tooth. It utilizes first-class lever mechanics, with one jaw functioning as a bumper-fulcrum and the other as an elevating arm. Once correctly positioned, minimal effort is required to elevate the tooth 2–3 mm from the socket. The tooth is then delivered from the socket with a different transfer forceps. If executed properly, there is minimal damage to the PDL, and the cortical plate remains intact.

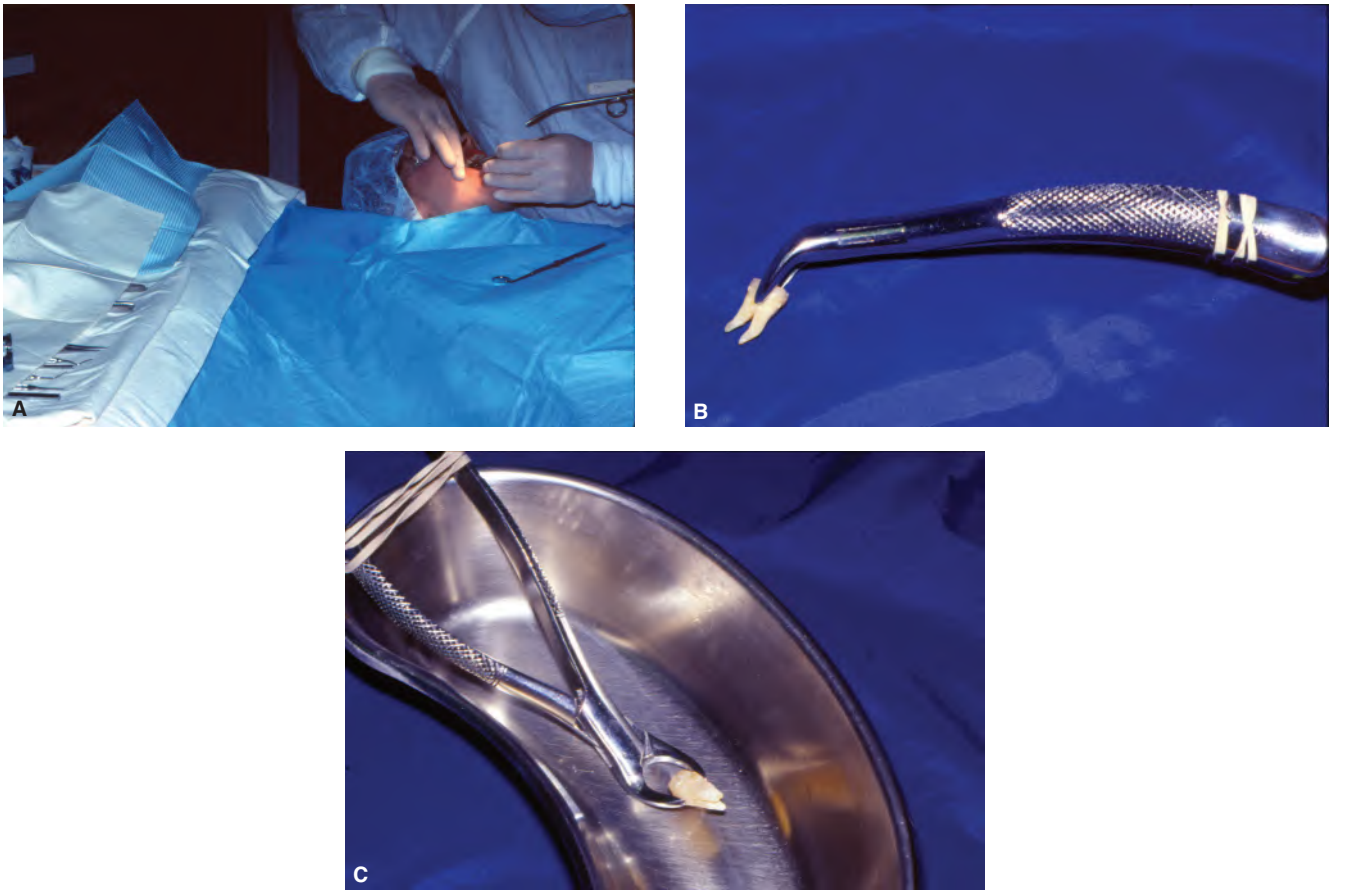


FIGURE 25-6 Preparation for IR procedure. ***A.*** A sterile field is secured from the extraction site side of the jaw to the adjacent mobile surgical table. This will safeguard the asepsis of the tooth if it dislodges from the transfer forceps during delivery to and from the operating table. ***B.*** Once the tooth is mobile enough to be removed from the socket, a transfer forceps (the pattern will be dictated by the tooth location) is gently adapted to deliver the tooth from the extraction site. The beaks of the forceps are coronal to the attachment tissues, and a sterile ligature band is wound around the handles to exert a mild closing pressure on the crown of the tooth. This makeshift hemostat allows the forceps to secure the tooth without the clinician having to actively squeeze the handles together. ***C.*** The forceps with the tooth is placed in the emesis basin containing HBSS, fully submerging the tooth beneath the surface of the fluid. Once safely in the solution, the tooth can be repositioned in the forceps beaks for improved access and manipulation. The basin can also be repositioned and tilted slightly for surgeon's comfort and ease of access, then stabilized with in this new position with sterile towels.

Modified IR Technique

The keystone to any replantation, intentional or posttraumatic, is the maintenance of the PDL cell vitality on the root surface. Both the extra-alveolar time period and the manner in which the tooth is stored or transported are critical in the preservation and regeneration of these cells.[30] If the PDL becomes desiccated secondary to dehydration, or the structure of the cells is distorted while in the incorrect storage environment, ankyloses and root resorptions are a likely scenario.[31-34] Traditionally, isotonic saline was considered the fluid of choice for the hydration of the root surface during replantations,[35-37] either dripped onto the root surface or soaking the gauze containing the extracted tooth. The rationale was the osmolality comparable to that of the PDL cells. It also prevented the air drying of the root surfaces. However, because saline lacks any nutritional benefits, it fails to sustain the healthy metabolism of the periodontal fibroblasts,[38] with marked reduction in cell viability after prolonged storage.[39] For the aforementioned reasons, the recommendation was to complete the *ex vivo* surgical procedures as rapidly as possible, replacing the tooth in the alveolus within 5–10 minutes. Unless the surgical staff was well orchestrated in the procedure, the time constraints could induce procedural mishaps and anxiety for both the surgeon and patient.

In 1983, an animal study[40] compared post replantation resorptions in monkey teeth when stored in either milk or saliva. It was found that teeth stored for up to six hours in milk demonstrated healing patterns similar to teeth that were replanted immediately (little or no ankyloses or resorptions). This, and a previous study by the same investigators,[41] inspired a search by the chapter's author to find a more physiologic replacement for saline in the IR procedure. Several laboratory tissue culture solutions were considered, selecting the sterile Hank's Balanced Salt Solution (HBSS) with phenol red indicator (Life Technologies, A Thermo Fisher Scientific Brand, Grand Island, NY, U.S.A.). This solution contains the essential metabolites to support cell survival including D-glucose, calcium chloride, and magnesium

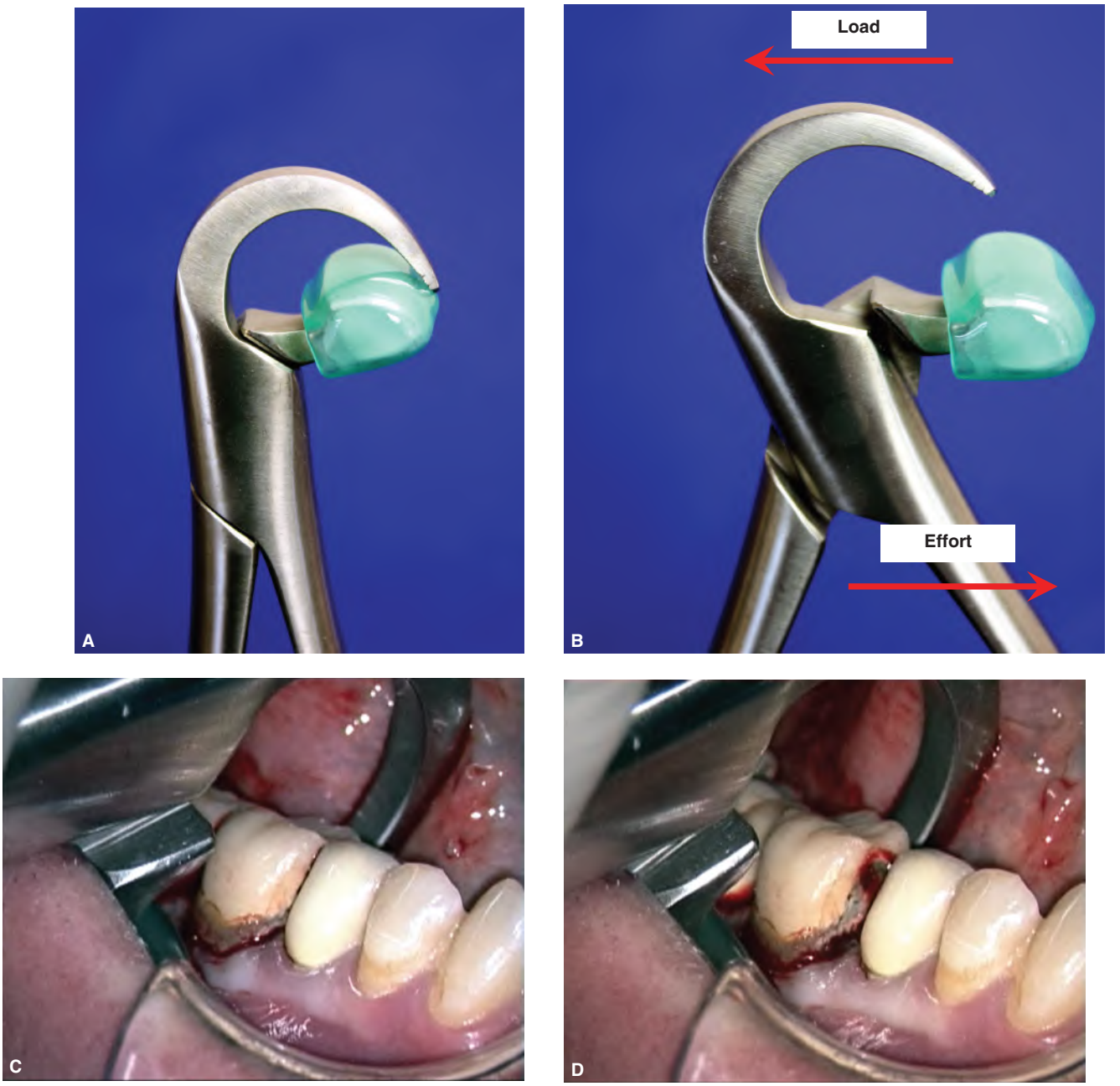


FIGURE 25-7 The Physics Forceps is available in a wide range of patterns and applications, specific for tooth and jaw location. The device employs first class lever mechanics, where the fulcrum is between the effort (handles) and the load (tooth surface). ***A.*** The assembled forceps. ***B.*** The fulcrum, or bumper, is covered with a disposable silicone bumper guard. When the force is applied in one direction, the beak exerts an elevating force in the opposite direction, extracting the tooth. ***C.*** Clinically, the bumper is placed at the mucogingival junction of the tooth to be extracted, and the beak engaged on the opposite side of the tooth above the periodontal attachment by closing the handles. The handles of the forceps are then rotated a few degrees until resistance is felt; the rotation stops, but the pressure is statically maintained. Shortly, the fibers of the PDL begin to separate, and a small hemorrhage may form in the sulcus. ***D.*** The handles are then rotated a few more degrees, until the tooth has moved 1–3 mm in the socket. The forceps are disengaged, and the tooth can be delivered from the socket into the emesis basin.

sulphate anhydrous. It demonstrates both the ideal pH (7.4) and osmolality range (280 mosmol kg^{-1}), and can maintain this preservation environment for up to 24 hours with replenishment.[42-44] (Note: the phenol indicator is used to signal depletion of the metabolites in solution; the abbreviated time frame of the procedure does not warrant this feature, so HBSS without the phenol red is the preferred solution.) Continuous immersion of the extracted tooth in this solution supported the safe expansion of the extra-oral treatment window. This allowed for a more careful scrutiny of the root surfaces and, by extension, greater precision in the diagnosis and root end treatment. In a clinical teaching setting, this "time buffer" enables the clinician to carefully execute the procedure and develop the proper skill set.

This modification to the traditional IR procedure was implemented in the private practice setting, and the results were debuted in 1993 as part of a microsurgical course. It was subsequently presented in 1994 at an international specialty conference in Philadelphia.[45] Since then, dozens of articles and case reports have been generated using this tissue culture solution for IR, with remarkable degrees of success. Subsequent research comparing HBSS to other media for storage and transport has confirmed its superiority with regard to biocompatibility, cost, and availability.[46-48] The following section will detail the new protocols using HBSS.

The technique consists of six stages (Figure 25-8):

1. Prophylaxis
2. Anesthesia
3. Luxation/Delivery
4. Evaluation
5. Root-End Resection/Filling
6. Replantation

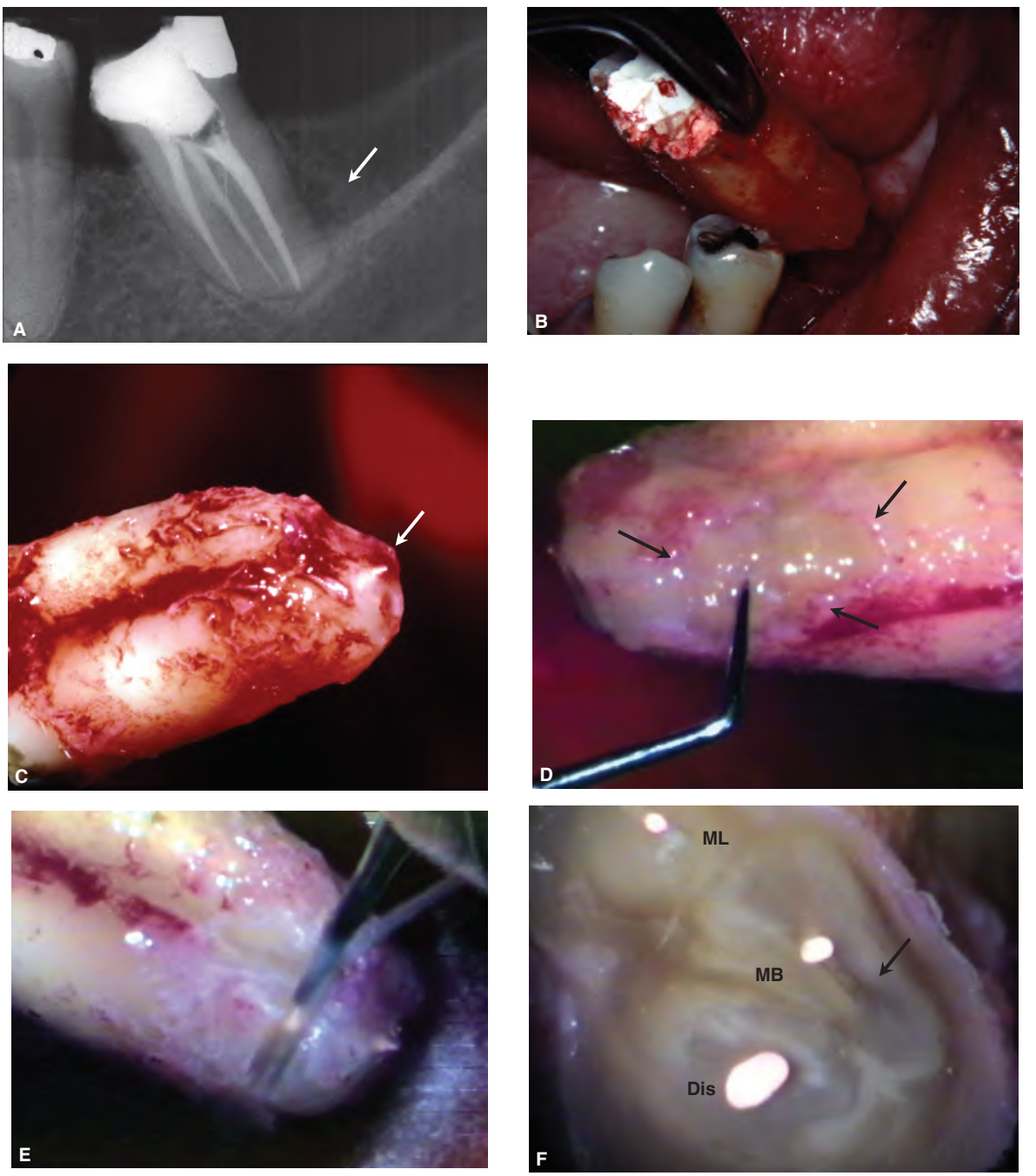
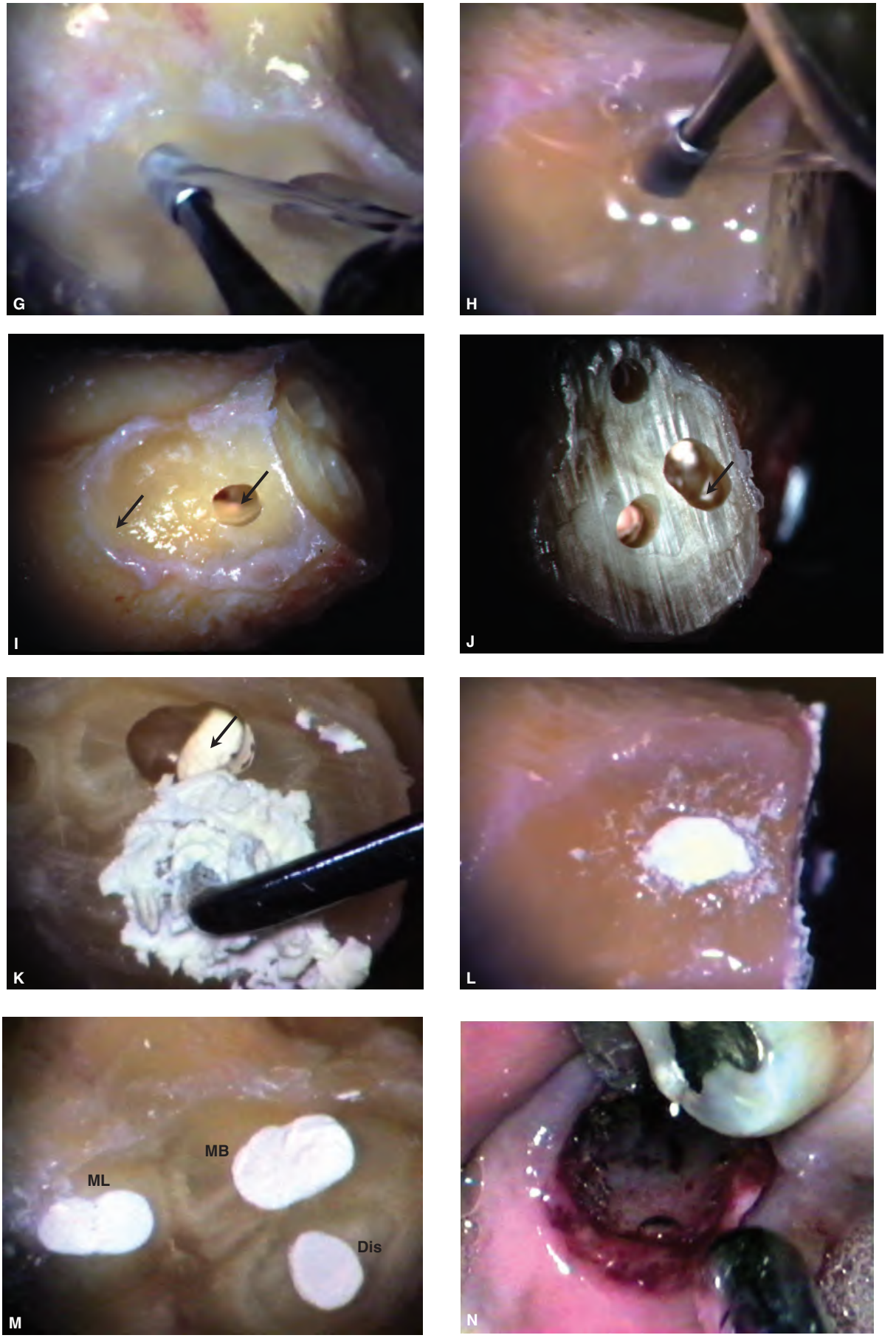


FIGURE 25-8 (*Continued on next page*)



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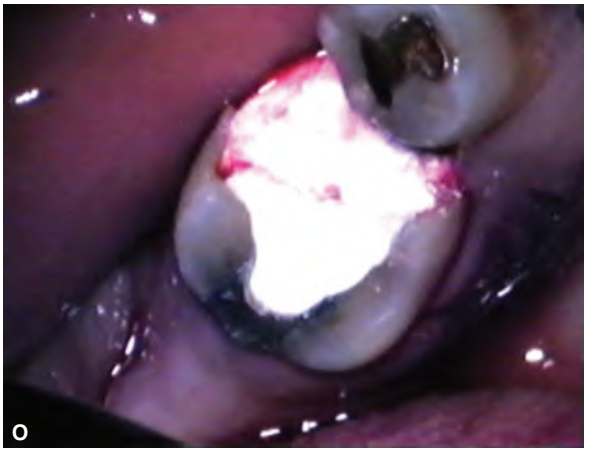

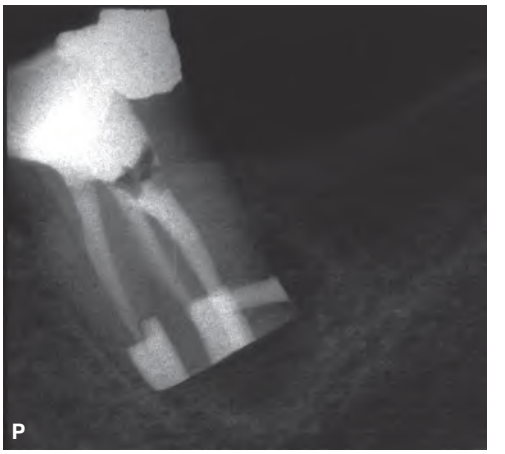

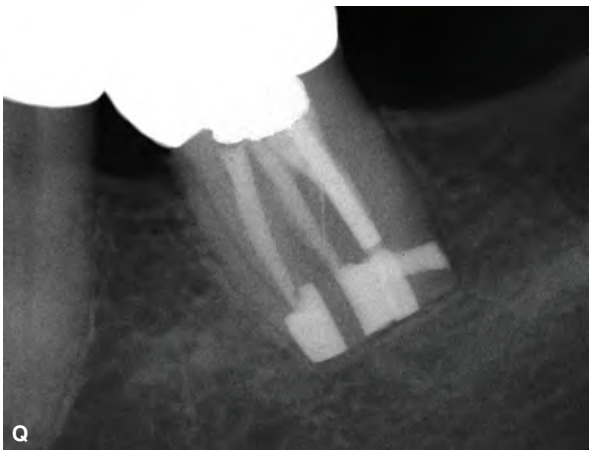

FIGURE 25-8 Step-by-step IR of a mandibular left first molar. ***A.*** Preoperative radiograph. Note the lesion at the distal aspect of the distal root (arrow) indicating possible presence of a lateral canal. ***B.*** The tooth is removed from the socket, and delivered to the basin containing the HBSS. The socket will be flushed with saline, and a sponge placed over the opening. ***C.*** The root is carefully examined in all aspects, noting any fractures, lateral canals, lesions or other anomalies. The overextended gutta-percha can be seen protruding through the apical lesion (arrow). The tooth is kept submerged during these, and all subsequent, observations and procedures. ***D.*** A low power view of the distal aspect of the distal root reveals the presence of a large lateral canal. Note the withdrawal of the PDL from the vicinity of this canals' foramen (Explorer tip). ***E.*** The tooth is positioned so that the root is horizontal and just below the surface of the HBSS. Resection of the root is performed with the root submerged in the fluid. The amount to be removed will be dictated by usual surgical protocols. ***F.*** A high power view of the resected surface reveals a "C shaped" anatomy, with an isthmus extending from the MB canal towards the distal root. The root-end preparation of the MB root will need to include this isthmus and any connections with the distal canal. ***G.*** The ML canal is being prepared using a #330 bur. The distal and MB canals have already been prepared; the MB preparation can be seen behind the fluid stream. All the preparations are 3 mm deep, prepared in the long axis of the selected canal. ***H.*** Preparation of the lateral canal, using the #330 bur. It was prepared to the same depth (3 mm) and centered on the long axis of the canal. Note the denuded root surface corresponding to the lack of PDL and cementum. ***I.*** Low power view of the relationship of the lateral and apical root end preparations. Note the PDL "halo" and the hint of gutta-percha in the lateral canal. ***J.*** High power view demonstrates the proximity of the MB and distal preparations. The communication can be seen at the depth of the preparations, as well as the communication with the lateral canal. ***K.*** Compaction of the root-end filling material (ZOE) into the distal canal resulted in extrusion of the ZOE into the base of the MB preparation; further evidence of the communication. The remaining apical root-end preparations were filled in a similar manner. ***L.*** Compaction of the root-end filling material in the apical preparations resulted in the extrusion of the material from the lateral canal; the excess was carved to the cavosurface and removed. ***M.*** The root end fillings were examined for extent and finish, with the excess removed prior to replantation. ***N.*** The sponge overlying the socket is removed, and the alveolus is thoroughly rinsed with saline or HBSS. This is done to remove any occult blood clots and present a fresh, bleeding surface on the bone. The surrounding gingival tissues are also swabbed with CHX prior to replantation of the tooth. ***O.*** The tooth is replanted, and the soft tissues gently readapted to the cervical portion of the tooth. The replantation position is verified radiographically and, if complete, the tooth is splinted (if required) and the occlusion checked before dismissal. ***P.*** Immediate postoperative radiograph. The radiolucent areas correspond to the resection space and the lateral lesion. ***Q.*** Nine-year postoperative recall radiograph demonstrates complete healing. The tooth is asymptomatic and functional.

Prophylaxis

Antibiotic premedication is prescribed when dictated by the patients' medical history, particularly if there are cardiovascular or prosthetic concerns. The most current recommendations and guidelines can be obtained from the American Heart Association (AHA) and American Association of Orthopaedic Medicine (AAOM) websites. Postoperatively, antibiotics are not routinely required unless a graft has been placed to augment the postreplantation support, or the rupture of a periapical lesion during the extraction occurs within the socket. In this case, a transient bacteremia may result, and antibiotic coverage for three to five days would be prudent.

Nonsteroidal anti-inflammatory drugs (NSAIDs) and/or analgesics, if not contraindicated, are recommended per os prior to the surgical procedure. In concert with the administration of a long-acting local anesthetic, preoperative administration of these drugs has been shown to minimize the inflammatory response and increase patient's comfort postoperatively.[49-51]

Chlorhexidine gluconate 0.12% (CHX) rinses are prescribed starting 24 hours prior to the replantation appointment, and continuing for the 14-day-postoperative period. This has been shown to minimize microbial contamination on the sulcus from plaque on the replanted tooth and on surrounding teeth.[52] This will also augment the local oral hygiene care, as the patients will be prohibited from brushing or flossing the involved areas until reattachment can be confirmed.

"Legal Prophylaxis" or informed consent for this procedure should contain all the elements normally found in the presurgical documentation, with particular emphasis concerning tooth fracture during the extraction, and postoperative re-attachment/resorptive complications. In the author's experience, most patients declining a conventional *in vivo* surgical correction elect, instead, to have the tooth extracted. In these instances, where patients have already resigned themselves to the loss of the tooth, an offer is made to extract the tooth for them. If the tooth can be removed without fracture, and the etiology of the periapical disease identified, the surgeon will perform the required root end procedure and replant the tooth. The rationale for the procedure is explained as a corollary to the accidental loss of a tooth by trauma, and the success of those replantations. It is also justified when the exarticulation force will be well controlled, and the tooth is not subjected to microbial contamination and desiccation effects, reducing the postoperative resorption potentials. Conversely, if the root does manifest a previously undisclosed irreparable condition (i.e., extensive root fracture), then the tooth should be safely removed, and appropriate socket preservation methods implemented. Offered this choice, nearly all patients will acquiesce and decide to give the tooth "one more chance"!

Anesthesia

The route of administration of the anesthetic for this procedure should not differ from a standard surgical protocol, that is, block or regional infiltrations, with the exception of interligamentary injections. Placement of the needle tip onto the coronal root surfaces could exert a physical disruption of the cementoblast layer already predisposed to resorption from the extraction forces.[29] The action of the locally pressurized injection will precipitate a temporary disruption of the microcirculation, with potential damage to the affected tissues, particularly the PDL.[53] Instead, a trans-osseous delivery (Stabident™) should be considered. Following the manufacturers' directions for use with regard to placement of the trephinating tip and rotational speeds, one carpule (1.8 ml) is all that is usually required to achieve the desired anesthetic effect.[54] Caution should be exercised with selection of the anesthetic for this administration route, as there will be transient systemic effects (tachycardia, cardio-pulmonary) due to the intraosseous nature of the injection and the microcirculation of the bone. Larger volumes of anesthetic with higher vasoconstrictor concentrations are contraindicated in those patients at risk for anticipated systemic reactions.

The choice of anesthetic type will also be similar to that of the surgeons' standard protocol, with the recommendation being one with extended duration. In combination with preoperative NSAIDs/analgesics, the use of a long acting anesthetic (bupivacaine, Marcaine™) has been determined to dramatically reduce the incidence of postoperative discomfort for the patient by delaying the onset of peripheral sensation.[55,56]

Finally, the opportunity for "vocal anesthesia" presents itself while awaiting the onset of the injected drugs. The procedural steps are outlined, with reiteration of the possible complication of root fracture but, moreover, to reassure the patients of their choice of the IR and confidence in the procedure. During the actual *ex vivo* surgical manipulations, the microscopic images are broadcast onto a video monitor while the patient is sitting upright and watching the screen. Oftentimes, patients will ask questions about what they are viewing, forgetting for the moment that this is their tooth on the screen. If there is a fracture present, they can visualize it firsthand. If the root repair is taking place, they gain an appreciation for the precision and elegance that is required. For the patient, it is truly a unique and an "out of body" experience!

Luxation/Delivery

The patient is reclined and draped with a sterile field that extends from the site of extraction to the table/cart where the operative procedures will take place. This is to insure that if the tooth dislodges from the forceps beak during the transfer from the patient to the basin, it will be captured on a sterile surface and can be retrieved without consequence. The tissues surrounding the extraction field are swabbed with CHX and the excess suctioned away. The coronal gingival attachment is severed circumferentially to the alveolar crest either with a small microblade or sharp periosteal elevator. The gingival tissues are carefully reflected to the depth of the sulcus, and the beaks of the selected forceps are adapted on the buccal (facial)/lingual (palatal) aspect of the crown, always mindful to keep the beaks away from the cementum surfaces.

The choice of forceps pattern will be dictated by the tooth type, location, and available coronal tooth structure. Limiting the adaptation to the level of the CEJ, or just slightly apical to that margin, will assure the safe occlusal-apical dimension and thus prevent a crushing force on the cementum during the exarticulation. The forceps are then used to exert a gentle buccal-lingual motion, slowly expanding the alveolus and allowing the PDL fibers to separate within the socket. Every effort is made to avoid undue force, so rapid rocking motions are too forceful and contraindicated.

Minute hemorrhages from the sulcus, in addition to decreased resistance to the forceps motion, signal the imminent delivery of the tooth from the socket. The extraction process is paused, and a different forceps is applied to the crown to secure the tooth from the extraction site to the emesis basin. These forceps have a sterile ligature wrapped around the handles. The tension of the ligature will convert the forceps into a makeshift hemostat, permitting a gentle and constant securing force while the root end procedures are performed. This relieves surgeons from having to maintain this force with their hands, thereby limiting their fatigue. The new forceps delivers the tooth from the socket and submerges it into the emesis basin that has been partially filled with HBSS. The socket is rinsed with saline and inspected with the SOM; if clear, the socket opening is occluded with 2 × 2 gauze sponges, and the patient is allowed to relax and close his or her mouth.

A recent change to the extraction phase utilizes a different type of forceps, the Physics Forceps™. There is no back and forth luxation in its use; instead, the movement is more akin to a pivoting elevation of the tooth, lifting it from the socket after the PDL fibers have fatigued and separated. One to two millimeters of elevation is all that is required (Figure 25-7C and D); at that point, the beaks of the transfer forceps are introduced, and the tooth delivered to the basin as described above.

If there is a full coverage crown on the tooth, a strong likelihood exists that it will become dislodged during extraction. If there is sufficient coronal core structure remaining, the surgeon can readapt the beaks to a fresh location and continue the procedure. However, if the crown shears horizontally at the gingival level, with little tooth structure protruding above the gingiva, the surgeon must reassess the availability of sound tooth structure above the level of the attachment. If there is sufficient room to adapt the beaks of the forceps without impinging on the attachment/cementum location, then the extraction can proceed with caution. When there is insufficient structure remaining, or the root has fractured below the alveolar crest, the extraction may not be possible without employing a root elevator or periotome. The use of these instruments is absolutely contraindicated because of the scraping/crushing damage they inflict on the cementum and PDL, with the ensuing resorption response all but guaranteed. This particular scenario should be well explained in the informed consent, with both parties in full disclosure as to how to proceed next (i.e., extract and graft the site, extract and place an endosseous implant, or other).

In those cases where such complication is anticipated because of root configurations or weakness due to multiple retreatments, a novel approach may provide a solution.[57] The tooth identified as one that poses a significant extraction risk (complicated root structure, thick cortical plate, and extensive core prosthetics) undergoes a two- to three-week period of preoperative orthodontic extrusion prior to extraction with their preferred instrument, the Physics Forceps™.[58,59] No extraction failures have been reported in the extrusion group, with over 25 months postoperative survival rate of 98.1%. Although the time required for extraction of the teeth was not significantly different between the extruded and nonextruded groups, the clinical impression was that the extruded teeth were easier to deliver than anticipated, given their preoperative complication set. Of singular importance is the fact that there were no signs of resorption observed in the extruded teeth, while 10 out of 123 teeth in the nonextruded group manifested some signs of resorptive defects. These authors proposed that the preextraction extrusion serves to increase the PDL volume around the tooth, which provides a protective component to the root surface and deterrent to root resorption. This theory is based on the observations of Hayashi[60] and his work on ortho-transplantation.

Evaluation

Once the tooth is submerged in the basin containing HBSS, the socket is examined for evidence of fracture or pathology. A partial or "green-stick" fracture of the alveolar cortical plate, even if it prolapses into the socket space, is of little concern as long as it can be repositioned prior to replantation. Complete fractures, especially if fixation is questionable postoperatively, should be removed to reduce the risk of spontaneous sequestration. In these instances, bone graft augmentation can be considered if the tooth is rigidly splinted after replantation.

The apical lesions are normally attached to the root apex(ices) and are recovered as a matter of course during removal of the tooth. Occasionally, a bolus of tissue remains in the depth of the socket. If it cannot be evacuated by gentle aspiration with the surgical suction, and it does not interfere with the repositioning of the tooth, it can and should remain undisturbed. Aggressive curettage of the socket, and root surfaces, have been reported to initiate root resorption, and should be considered when contemplating any manipulations in the socket.[33,61] The same rule applies to any root tips that have splintered off during the extraction; if they are mobile and easily harvested with minimal instrumentation and destruction, removal is indicated. Conversely, if it is firmly attached and smaller than 3 mm radiographically, it should remain in the alveolus to be subsequently remodeled and resorbed. Once the inspection of the socket is complete, it is rinsed with saline and covered with saline-moistened gauze sponges. The patient is then directed to gently bite down to hold the sponges in place, and returned to a comfortable seated position.

While fully submerged in the HBSS, the surfaces of the tooth are inspected with the aid of the SOM, noting any

fractures, lateral canals or other portals of exit (POE) from the root canal spaces. Any lesions loosely connected to the root surface can be teased off using Addison forceps. If no fractures are evident, then the root end resection(s) (RER) can be planned. The tooth can be readjusted in the forceps beak so that they are solely on the crown, and have a solid purchase that will resist dislodgement during the cutting motions of the handpiece. Resection is accomplished with the selected apex(ices) just below the surface of the fluid; this may necessitate the surgeons' fingers or even hand be placed in the basin to secure a solid finger rest position. The basin can be stabilized using surgical towels, propped up on one end to affect a more comfortable access position for the operations, and aid in the visualization with the surgical operating microscope. If there is more than one root resection planned, the angle of the resections may be different and will often oblige changes in the position of the forceps in the basin, or even readjustment of the tooth in the beaks itself.

Bur selection is based on a fast cutting and nonclogging flute design. Carbide fissure burs, either straight or tapered and of surgical length, are the instrument of choice. Diamonds, regardless of grit, leave a roughed surface that may create finishing issues during the root end filling (REF). Any rounded bur configuration can create a ripple effect in the resected surface unless it can fully span the breadth of the resection.

Once the root is submerged and comfortably stabilized just below the surface of the fluid, 1–3 mm of the root tip is resected at right angles to the long axis of that particular root. The amount to be removed is guided by microsurgical protocol and clinical judgment, with one minor exception; severe curvatures/dilacerations, if projected to hinder or prohibit a smooth replantation, should be resected to eliminate the offending curvature, regardless of the length. Only the tip of the fissure bur is used in the resection, with each cutting pass slightly deeper than the previous one. When done in this manner, the dentin at the interface of the bur will be adequately cooled and flushed, minimizing any frictional burning events. The fluid passing through the handpiece tip onto the revolving bur should also be HBSS. This is readily accomplished in a closed delivery system, where the solution is contained in a removable pressurized canister. The HBSS is flushed completely through the dental unit handpiece lines, delivering fresh fluid to the resection site continually during the procedure.

Once all the root resections are complete, the resected surfaces can be stained with a disclosing dye (methylene blue) and examined to discover the extent and location of canal(s), isthmus, or fractures. If the fractures are minor, and can be removed via resection, it is accomplished at this juncture. Otherwise, the tooth is repositioned in the forceps beaks in readiness for the root end preparation (REP).

The REP is most efficiently accomplished using an inverted cone- or pear-shaped carbide bur, cutting in the long axis of the canal to a depth of 3–4 mm, again according to microsurgical protocols. All canal spaces present, including the isthmus, are prepared to the same depth. The tooth has been repositioned so that the resected surface is just below the fluid surface in the basin, and facing towards the microscope. In this orientation, the REP motion with the handpiece is comparable to a drill press; a repeatable vertical motion vector with precise depth control.

The use of an ultrasonic root end preparation (USREP) tip has been reported in the literature[14,28,62] but should be utilized with caution in small, narrow roots. Early investigations into the safety and efficacy of these instruments reported incidences of fractures and cracking after USREP in extracted teeth.[63,64] The evaluation of these instruments was repeated in cadaver specimens[65] and clinical surgical patients,[66] using resin replica impressions of the prepared root ends in situ. Observations of the prepared replica casts revealed no evidence of cracking. It was hypothesized that the PDL afforded some cushioning effect to the ultrasonic vibrations, dampening the magnitude of the transmitted vibratory force through the root end. When executing IR preparations with an USREP tip, the surgeon is essentially duplicating the bench top study that produced the increased rate of observed fractures. There were no observed incidences of fracture when carbide burs were used to prepare the root ends,[64] and the root end preparation with a carbide bur can be accomplished in a matter of seconds, versus minutes with a USREP tip.

The completed REP(s) are evaluated for extent and depth under the SOM with low and high power magnification; if they are satisfactory, they are flushed with HBSS and dried using a Stropko™ irrigator (www.stropko.com) with microneedle tip. Again, the tooth is positioned so that only the resected surface is above the level of the HBSS.

The choice of an REF varies considerably in the literature, and most are multi-purpose restorative materials adapted for root end surgery; IRM,[67] ZOE,[26] Super EBA cement,[14,68] dentin bonding agent,[69-71] Spherical[72-74] or Zinc-free[36,75]amalgam, Calcium-enriched mixture (CEM),[76,77] Mineral Trioxide Aggregate (MTA),[57,78] or glass ionomer.[79] Selection is usually based on availability, ease of mixing and placement, and biocompatibility and stability during the replantation. The materials that complete their setting shortly after placement are easier to finish and are resistant to displacement, but are not as biocompatible as the bioceramic REF's, such as MTA.

The disadvantages of MTA for the purpose of IR are difficulty in delivery and placement, long setting time period (four to six hours) and lack of resistance to displacement (wash-out). The handling issues are easily addressed by using instruments meant to carry the material to the surgical site such as MAPS (Micro Apical Placement Syringe, Roydent, Johnson City, TN) or Dovgan carrier. The MTA is moisture sensitive, so the resected surface of the tooth must be above the level of the HBSS to prevent an influx of the fluid and premature soaking of the MTA. Once the

MTA has been condensed and finished, the surface of the MTA in the preparations is gently desiccated with a gentle stream of air from the Stropko™ irrigator directed across the surface of the material. This will effectively "skin" the surface of the MTA, forming a thin barrier for the material deeper in the preparation. The tooth is then completely submerged and allowed to remain undisturbed for five minutes. This allows for further setting of the MTA in a hydrated environment.

If a moisture-tolerant material is chosen instead, the preparations are dried as above but, because the materials are hydrophilic, the presence of small amounts of moisture, or fluid, in the preparations is not critical. Placement, condensing, and finishing of the REF material is done following the same protocols of conventional root end surgery. Regardless of the materials used, the excess REF is removed from the preparation site, and final inspection of the extracted tooth is conducted under the SOM.

Replantation

Prior to replantation, the socket is reexamined for fractures of the alveolus and any foreign material that may have contaminated the site. It is rinsed with sterile saline, or HBSS, to remove any occult blood clots, and the surrounding soft tissue surfaces are swabbed with CHX. The clots are evacuated because of their association with increased incidences of inciting postoperative ankyloses,[80] and the rinsing action creates a fresh bleeding surface. The security of the extracted tooth in the transfer forceps beaks is reexamined to confirm its' stability, and the tooth is removed from the basin and immediately redelivered to the socket. Gentle apical pressure is applied to reduce the tooth to its' original location and depth. If there is resistance to the reinsertion, the tooth is returned to the basin, and the socket reexamined to ascertain the obstruction. Once the tooth has been fully reinserted into the socket, the cortical plates are carefully coapted back against the root surface (if they were displaced or spread during the extraction), and a small gauze sponge is placed across the occlusal surface of the tooth. The patient is directed to carefully occlude down onto the sponge for a few minutes, after which a radiograph is taken to confirm the complete placement of the tooth in the socket. If the operator is satisfied with the placement, the tooth and surrounding tissues are swabbed once again with CHX, and the necessity for postreplantation splinting is evaluated.

The need for postoperative stabilization is dictated by several factors: length of the root, lack or loss of interseptal bone, excessive postreplantation mobility, and concerns about postoperative patient compliance.[81-84] The choice of splinting material should allow for a small amount of physiologic mobility, as this has been shown to aid in periodontal reorganization and healing.[85,86] If the fixation is too rigid, it increases the likelihood that remodeling will take place on the root surface, resulting in replacement resorption (ankyloses).[75,87,88]

The design can range from a single suture crisscrossed over the crowns' occlusal surface[89] to a 0.7 mm orthodontic wire reinforced composite splint[90] (Figure 25-9). The degree of instability will dictate the amount of rigidity required. The most commonly employed method utilizes a small amount of light-cured composite material (reinforced or not) spanning the interproximal gap to a stable adjacent tooth.[91-93] Once the splint is in place, the occlusion should be verified and all interferences removed. This is especially imperative if there is a suspicion of grinding, clenching, bruxism, or other parafunctional habit, as evidenced by wear facets or abfraction lines on the surrounding teeth. If present, but not accounted for, this degree of postreplantation occlusal stress can inhibit the reattachment, or prevent it completely.

The duration for the splinting can vary depending on the presplinted support and environment. However, generally, the tooth is stable enough after 7–14 days. At that time, the splint can be removed.[67,81,94,95] If a simple composite splint is affixed, the composite span can be severed with a small bur; if the tooth is stable, then the remaining material can be removed. If, however, the tooth is still slightly mobile, additional material can be adapted into the resected gap, and the splint reactivated.

The postoperative instructions mimic those for routine postsurgical care:

1. Soft diet (no seeds or hard/sticky foods) until the splint is removed, and chewing on the opposite side, if possible.
2. Avoid brushing or flossing the splinted area until it is removed. Instead, the area is gently rinsed with plain, lightly salted water to dislodge and remove any food debris.
3. CHX rinses twice a day until splint removal.
4. NSAIDs/Analgesics as prescribed for two to four days postoperatively. Antibiotics, if prescribed, should be taken as directed.
5. Avoid strenuous exercise or activity that can precipitate a hypertensive event. This rise in blood pressure can cause a local throbbing and, if significant enough, hemorrhaging in the sulcus or surrounding tissues. This will manifest as an ecchymosis if uncontrolled.
6. Cold compresses can be gently applied externally as a palliative comfort measure; hot packs should be avoided.

The patient is directed to return in seven days for re-evaluation of the site and splint assessment. Any sutures, other than those acting as a splint, are removed at this visit. The patient is seen again at day 14 for splint assessment and removal, if appropriate. Once the reattachment is confirmed, the patient is instructed to resume normal oral hygiene routines and schedules, with the caveat of no elective crown and bridge on the replanted tooth until the attachment has matured (usually three to four months). This also applies to any elective periodontal manipulations such as scaling, root planning,

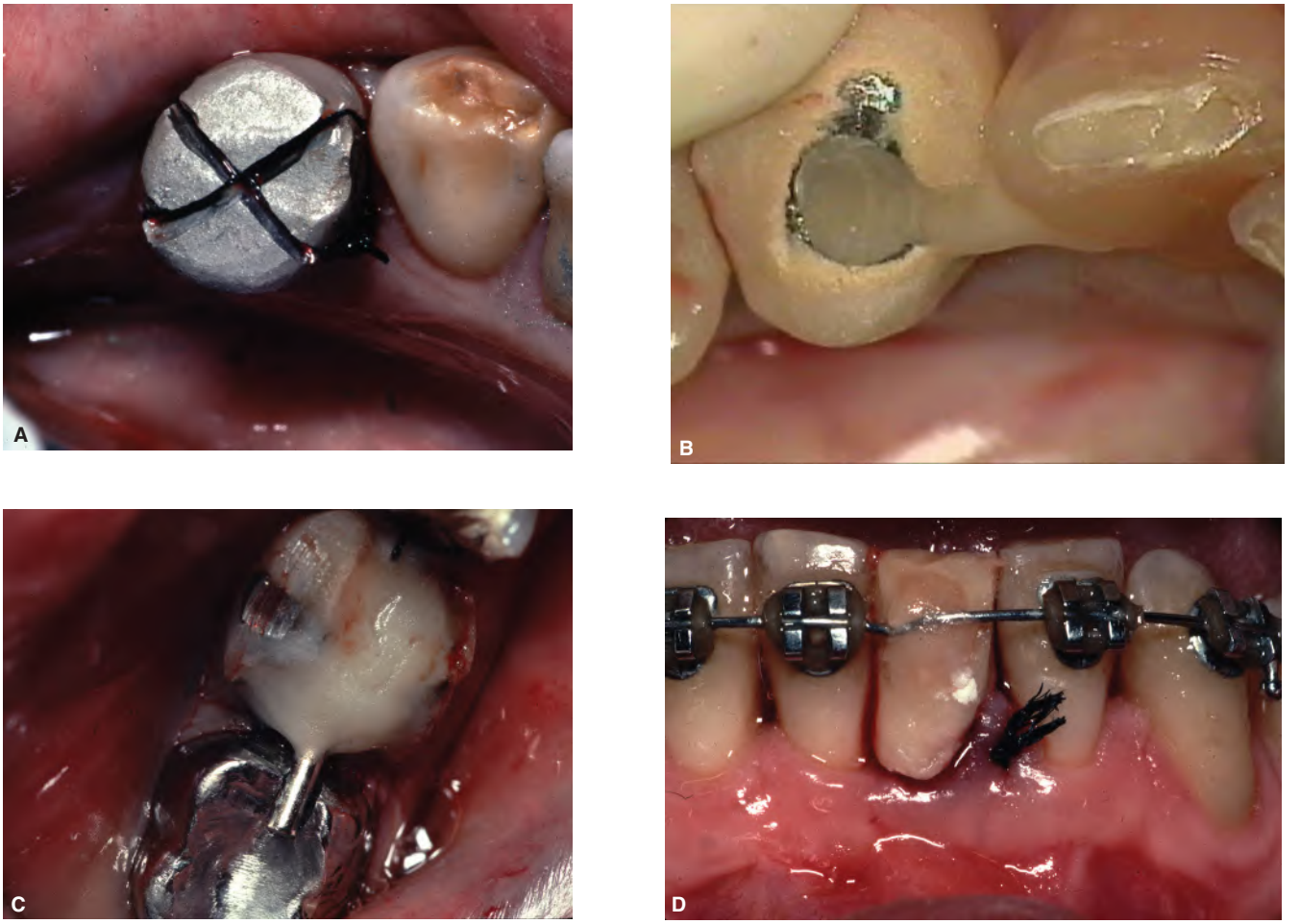

FIGURE 25-9 Stabilization of the intentionally implanted tooth. ***A.*** Depending on the degree of stability required, postoperative splinting can be a simple suture encircling the tooth or crisscrossed over the occlusal surface. ***B.*** When more rigid fixation is indicated, an acid-etched composite connection between two adjacent marginal ridges can be placed. This will allow for the connection to be sectioned and mobility of the tooth ascertained before removing the splint totally. ***C.*** If there has been a fracture of the alveolus bone, appropriate immobilization of the replanted tooth for a longer time period is warranted. ***D.*** If there are already splinting elements in the arch, such as an orthodontic wire, then the tooth can be temporarily secured to the wire with self-cured acrylic.

or curettage. The surgeons' postoperative recall schedule is enacted, traditionally six months and one year from the date of treatment. A clinical and radiographic examination is performed, evaluating periodontal and periapical healing, with notations of healing or abnormality/delayed/non resolution of the presenting signs and symptoms. While traditional radiographs provide preliminary insights into the progress of the periapical healing status, the resolution of the lesion may be more accurately evaluated and monitored using the CBCT.[96-98]

Complications

Complications of IR can manifest as either intra-operative or postoperative in nature. The intra-operative complications are usually technically derived. They may result from overt or misdirected application of force, inappropriate or incorrect instrument manipulation, or misdiagnosis of the root anatomy. The end result is often root fracture; the extent of which can be pivotal in whether the remaining tooth fragment is salvageable or not. As stated previously, this is one of the scenarios that should be discussed during the presurgical consult visit, with alternate treatment plans outlined in the informed consent. This discussion serves to streamline the decision tree if and when this complication does occur. If the remaining fragment is undamaged, and is large enough to be of prosthetic value, then the modified replantation can proceed as planned. An example of a salvaged root fracture and outcome is demonstrated in Figure 25-10.

Postoperatively, the two most common complications are nonattachment and resorptions. Aside from a failure of the splint to stabilize the replanted tooth, there is evidence to support that smoking and, in particular, nicotine can inhibit or prevent reattachment of human gingival fibroblasts (HGF). A recent study[99] demonstrated that high concentrations of nicotine and cotinine have adverse effects on the cell adhesion and proliferation of HGF cells. If these

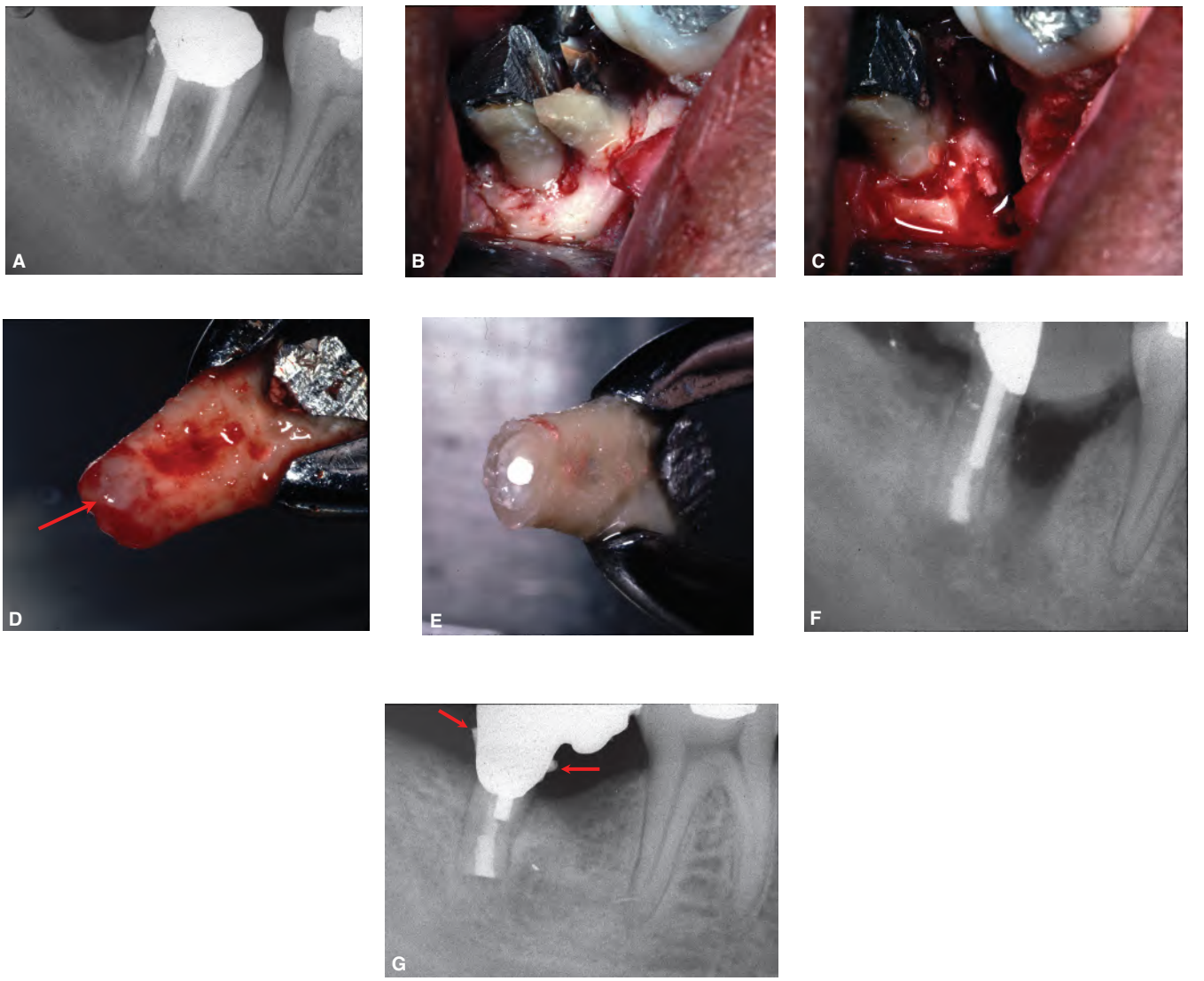

FIGURE 25-10 Salvaged root fracture in a mandibular right second molar. ***A.*** Preoperative radiograph. Note the large post in the distal canal, the convergence of the root forms and periapical areas on both root apices. The tooth has an amalgam buildup, and a temporary crown is in place. ***B.*** The mesial aspect of the root has fractured during the extraction, and the tooth has been hemisected through the furcation. The convergence of the mesial and distal roots is the most likely reason why the tooth was resistant to extraction and fractured. ***C.*** The mesial root has been atraumatically extracted, with minimal buccal cortical plate involvement. ***D.*** The distal root apex prior to resection. Note the small lesion attached to the root (arrow); it will be removed with the resected root tip. ***E.*** The distal root-end has been resected, and the root-end filling (Super EBA) has been placed and finished. ***F.*** Immediate postoperative radiograph. The distal root has been replanted, and the temporary crown relined and provisionally cemented over the root. Stabilization was achieved by bonding the temporary crown to the lingual of adjacent first molar, using orthodontic bonding resin. A small mylar plastic strip was placed over the exposed mesial extraction socket during the reline procedure to prevent migration of the acrylic into the socket. ***G.*** Eight-year recall radiograph. The area healed and periodontal probing depths were normal, in spite of the presence of deposits (arrows) on the abutment crown of this inlay mini-bridge.

are the cell populations and mechanism of action that need to be optimal for successful outcomes in IR, then the procedure may be contraindicated in patients who are heavy smokers. This is especially evident in teeth with trifurcations (i.e., maxillary molars). The large amount of surface area to be reattached can be adversely affected by exposure to the inhaled nicotine stream. Empirically, this is speculated to be the cause of the failures seen in the success and failure bar chart (Figure 25-11).

Resorptions are universally caused by alterations of the PDL or cementum, either as a response to necrotic pulpal tissue and infection, or as physical destruction of the attachment microstructures. As previously discussed, revision of an inadequate initial RCT by retreatment has a positive effect on successful outcomes.[20,21,23,100] This principle is relevant whether the surgical procedure is *in vivo* (conventional) or *ex vivo* (IR). Minimizing the impact of intracanal etiologies (necrotic debris, microbial infection)

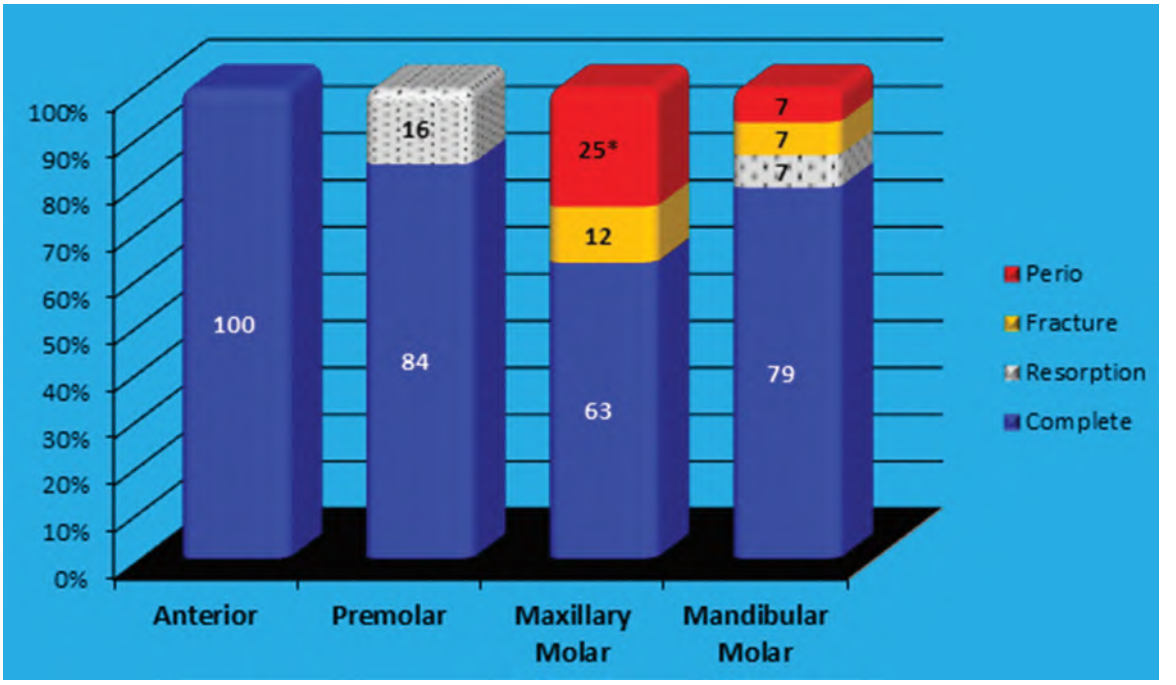

FIGURE 25-11 Bar graph comparing % success and complications of intentionally replanted teeth in different tooth groups. Resorptions that developed postoperatively coincided with sockets that received aggressive curettage. The fracture group represents those teeth where a portion of the tooth was fractured, but the remaining segment was replanted. In the periodontal category (recession, decreased attachment level, impaired re-attachment) the percentage in the maxillary molar contains an asterisk. In those patients, a significant number (66%) were moderate to heavy smokers; smoking affects periodontal healing and may have been the contributing factor in the complication seen.

theoretically decreases the likelihood that an inflammatory root resorption (IRR) will develop secondary to the intracanal contamination. The remaining consideration is the viability and structural integrity of the PDL and alveolus. Atraumatic extraction methods,[101] minimal contact with the root surfaces,[29,102] and constant hydration with the HBSS[46] safeguard the integrity of the tissues on the root surfaces.

What may not be as apparent is the health and disposition of the alveolar socket. Surgical alterations to the socket in the animal model[103] have resulted in significant differences regarding the extent (greater) and time of onset (shorter) of the root resorptions observed. The presence of an occult blood clot[80,104] or a socket that has been allowed to degrade[61] or become contaminated will also contribute to the initiation of the resorptive cascade on the replanted root surface. For these reasons, aggressive curettage of the alveolus is contraindicated. Gentle lavage of the socket, followed by careful insertion and evacuation of the socket via surgical suction, is usually sufficient to remove any infected tissues remaining after the extraction. Prior to repositioning the tooth, the alveolus should be thoroughly rinsed with saline or HBSS. This will assure the evacuation of any accumulated blood, and create a fresh, bleeding surface. When forceful apical curettage is done, a resultant resorption can develop.

Efforts to guide the cellular reattachment response with the aim of delaying or preventing resorption have been investigated for some time in animal studies and human trials. Application of fluorides to the root surfaces,[105] systemic and local steroid administration,[106,107] topical sodium alendronate (bisphosphonate)[108] and enamel matrix derivatives (EMD)[109-111] have all been employed, with the last (EMD) showing the most promising clinical results. One of the commercially available EMD preparations, Emdogain (Straumann USA, Andover, MA, U.S.A.) was developed specifically for the regeneration of periodontal tissues, either alone or in combination with other guided bone or tissue regenerative materials. (Note: the Straumann company announced in 2008 that the import detention on Biora products in the U.S.A that has been in place since the beginning of 2007, has been lifted following a re-inspection of the Group's Biora facility in Sweden by the FDA [US Food and Drug Administration], and the product is once again offered for sale.) Available as a gel, it contains an enamel matrix protein derived from developing porcine embryonic enamel in a sterilized aqueous solution of propylene glycol alginate.[112] These proteins are incorporated into the acellular cementum matrix during development, and are coincident with the site of Sharpey's fiber attachment on the root surface,[113,114] suggesting that topical treatment with Emdogain may stimulate

periodontal reattachment. However, the exact mechanism by which this occurs is still unknown.

Lyngstadaas et al.[115] proposed that a major component of EMD, amelogenin, interacts with cells and induces the release of signaling molecules and secondarily initiating the regenerative process. Healing of the supporting tissues in a replanted tooth depends on various cellular activities such as migration, reattachment, proliferation, and differentiation of various periodontal cells (cementoblasts, osteoblasts, and PDL cells). EMD appears to orchestrate this well *in vitro*, stimulating the migration and proliferation of osteoblasts, endothelial cells, and epithelial cells.[116] Although more controlled studies are required, a recent meta-analysis[117] concluded that, in the presence of a PDL, use of EMD resulted in an increase of normal periodontal healing (compared to the absence of the PDL), with greatly reduced incidences of inflammatory or replacement resorptions.

Auxiliary Procedures Employing Intentional Replantation

Delayed Replantation for Periodontally Hopeless Teeth

One of the contraindications of the IR procedure mentioned previously is compromised health and stability of the periodontal tissues. Incumbent in this recommendation is the stipulation that the root surface and/or the socket will remain unaltered. In the presence of overt periodontal disease and bone loss, spontaneous reattachment is doubtful. There are instances, however, where retention of the natural tooth is preferable to extraction, especially when considering the prosthetic rehabilitation. It has been demonstrated that following loss of a tooth, there is a 25% decrease in local ridge bone volume in the first year alone, with a 40–60% loss of width in the succeeding three years post extraction.[118,119] Augmentation and site preservation of the socket can reduce, but not predictably eliminate, these sequelae. The extracted tooth, once suitably prepared and stabilized, can serve as a "biologic space maintainer," preserving bone volume and function. Even if the root eventually succumbs to resorption, the resultant replacement resorption will result in an increase of the alveolar height, and in a more favorable platform for a removable or implant-supported prosthesis. This process, termed "Delayed Intentional Replantation" (DIR),[120] is predicated on two conditions: sufficient decontamination of the root surface (plaque, calculus, necrotic PDL, infected cementum) and delayed re-implantation into the socket.

The removal of necrotic PDL was advocated by Lindskog et al.[121] where resorptions of the root surfaces occurred whenever the diseased PDL was allowed to remain upon replantation. These authors concluded that chemical debridement and removal of the PDL tissue rendered the remaining cementum less vulnerable to clastic attack and subsequent replacement resorption. This effect was also reported by other investigators, with no evidence of resorptions or ankyloses evident radiographically at the six-month re-evaluation.[122] In DIR, the extracted tooth root surfaces are gently debrided with an ultrasonic scaler, then polished with a fine diamond bur. Subsequently, the tooth is stored in a dexamethasone-fortified medium at 4°C for 10–14 days. Prior to replantation, the root ends is ultrasonically prepared, and filled with a suitable root end filling (Super EBA was used in this study).

Any granulation tissue in the socket is removed by curettage, the socket rinsed with saline to evacuate any debris, and a blood clot is allowed to form. The socket is allowed to partially heal, undisturbed, for two weeks. Ten to fourteen days following an extraction, woven bone begins to form in the socket.[123,124] This will function as a scaffold for the regenerating tissues, and provide some apical support for the replanted root. At this stage, the socket transitions from the inflammatory phase (three to seven days post extraction) to the healing phase, where woven bone formation and angiogenesis are actively evolving.

After two weeks, the coronal portion of the socket is incised, and the stored tooth is replanted under local anesthesia. A resin wire splint is applied for initial stabilization, and the occlusion adjusted to remove all interferences. The patient is given oral antibiotics for three days, and instructed to rinse with 0.12% chlorhexidine (CHX) for two weeks. The replantation site is rinsed with saline after seven days, and the splint removed at the one-month recall appointment. At this appointment, the exposed tooth surfaces are polished and radiographs are taken to monitor the reattachment.

In one study,[120] a success rate of 66% was reported at 21-month follow up. Results showed an increase (45%) in the amount of bone gained around the replanted tooth, and no radiographic evidence of resorptions. These results are encouraging with regard to the observations of a similar study[125] where the roots were debrided (chemically), but replanted within a short period of extra-oral time. The increase in bone gained was lower (16%), but these teeth were splinted for the duration of the study (six months), and may not be representative of the true clinical outcome. An interesting corollary to this procedure involves preparation of the socket using water-cooled implant drills of corresponding size to the replanted root. The sites are prepared to allow the conditioned teeth to be replanted in infra-occlusion. The replanted teeth are then splinted for a period of three to six months, with osseous grafts either at the time of replantation,[126] or at the three-month recall appointment.[127]

Prosthetic Repositioning

There are numerous reports of parents repositioning their child's tooth quickly after an avulsion, only to realize they have rotated to tooth 180° in the process. As long as the cells are viable, the tooth reattaches without consequence, for the PDL is not unique from one side of the tooth to

the other. This phenomenon has been utilized in the treatment of crown-root fractures,[128,129] where the teeth have been removed and intentionally repositioned into a more favorable prosthetic position. A similar scenario, where a conventional surgical approach would have removed too much bone, and the tooth being orthodontically repositioned for crown placement purposes, are demonstrated in Figure 25-12.

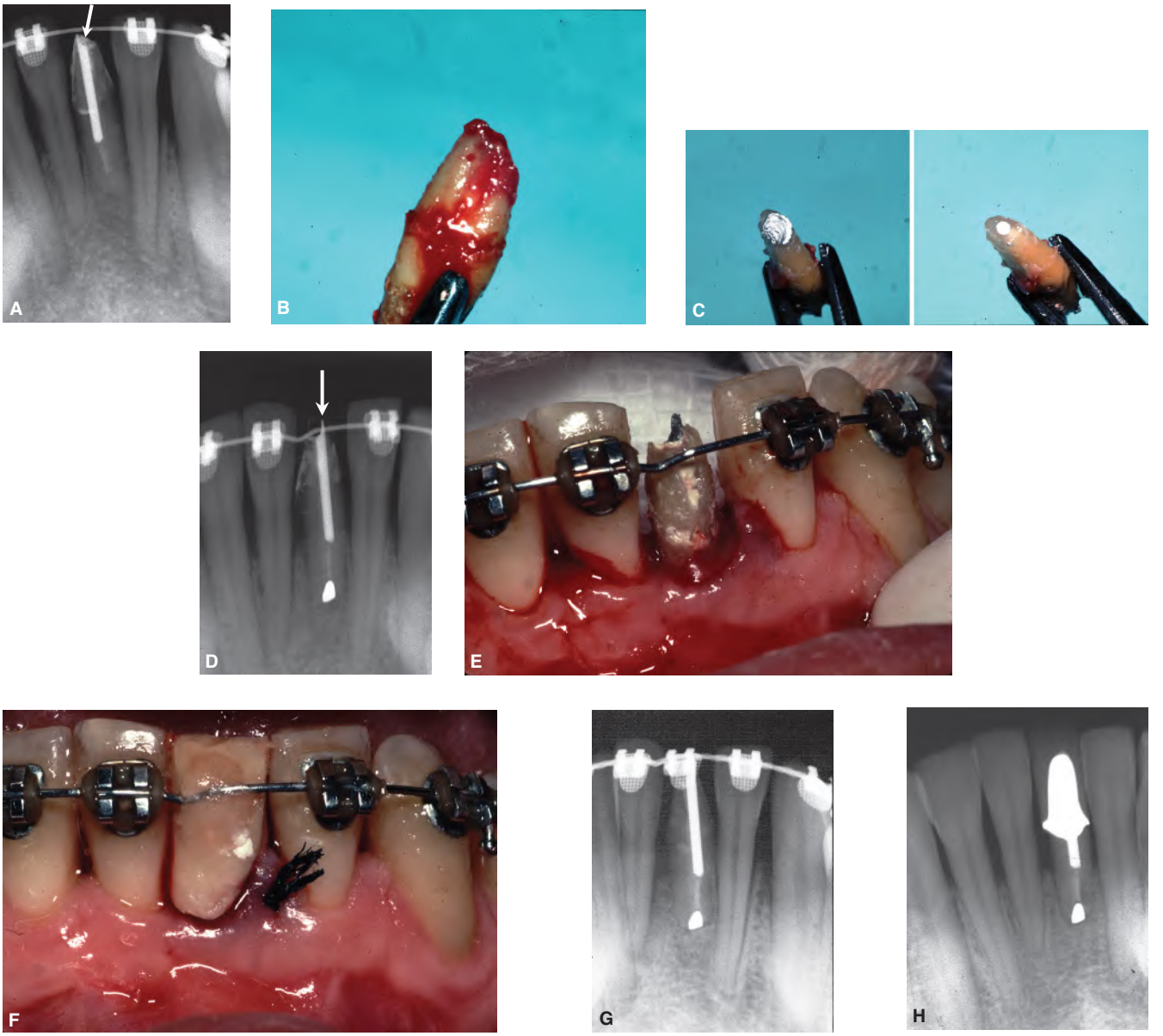

FIGURE 25-12 Intentional replantation in a mandibular left central incisor. ***A.*** Preoperative radiograph showing a large post, root bevel from the previous surgery, and periapical radiolucency. The tooth is undergoing orthodontic movement to rotate the tooth and correct the root proximity problem mesial and distal. (Note: the notch of the post is facing distal.) ***B.*** Clinical appearance of the extracted root. The lesion is removed and the root disclosing the bevel from the previous apicoectomy. ***C.*** The root apex is shaved to preserve as much length as possible, and prepared with a #330 carbide bur to a depth of 2 mm. The root end filling is placed and carved flush to the shaved surface. ***D.*** The root replanted and rotated 180°. Note the notch on the post is now facing mesial, and there is more space between the adjacent roots. ***E.*** The clinical image of the tooth replanted 180°. The arch wire was re-bent to accommodate the new orientation of the temporary crown. ***F.*** The temporary crown was cemented backwards, reflecting the 180° rotation of the tooth. The crown was splinted to the wire with acrylic, and a 4/0 silk interrupted suture placed to secure the distal papilla. ***G.*** Immediate postoperative radiograph showing the replanted tooth with the temporary crown in place. The tooth remained splinted for four weeks, and the tooth re-prepared after eight weeks to readjust to the new orientation. The soft and hard tissues healed without complication. ***H.*** 17-year recall radiograph showing evidence of complete healing. The tooth has been asymptomatic and in good function.

Replacement Resorption (Ankylosis)

The development of replacement resorption can be a consequence of a traumatized tooth. In the adult patient, the prosthetic options are more varied because skeletal maturation is complete and inter-arch relationships are established. In the child and adolescent, however, replacement root resorption proceeds at an accelerated rate, with the potential to exert a profound effect on the growth of the alveolus in the affected area. If the tooth remains in infraocclusion, the arrested eruption could affect function and phonetics. If the tooth is extracted, the resultant osseous defect may be difficult to correct with conventional augmentation methods. Classically, the treatment options for these ankylosed teeth were autotransplantation (usually a canine or premolar) or decoronation. A third option offers a compromise between those two concepts.[130]

Once the diagnosis of ankyloses, or evidence of infraocclusion, is confirmed, using a Periotest® (Gulden, Bensheim, Germany), the tooth is extracted atraumatically, and the resorbed areas of the root resected away. The canal space is prepared through the crown with a standard post drill (in this study, the Retropost, Brasseler-Komet, Lemgo, Germany was used), and a titanium post cemented. The length of the post approximates the original length of the tooth, and the appearance is similar to the endodontic implant devices that were placed in the early 1960's through 1975, although not extending apically nearly as long. The teeth are rinsed and dried with sterile gauzes, and Emdogain™ is applied to all exposed root surfaces before replantation. The teeth are physiologically splinted for 10–14 days, and appropriate recalls scheduled to monitor the healing and recurrence of resorptions. A six-year survey reported favorable results when small areas of ankylosis are diagnosed,[109] but more extensive areas of resorption tend to recur, and are better managed with the more traditional modalities such as autotransplantation[131] or decoronation.[132]

Perforation Repair

In cases where the location of the perforation is inaccessible by conventional microsurgical approach,[133] or the risk to surrounding structures is deemed unacceptable,[77] then extraction and repair or revision of treatment are indicated.

Patient Refusal of Treatment

Patients who have been subjected to multiple nonsurgical endodontic therapies, with perhaps a surgical revision of one of those teeth, are understandably reluctant to undergo a second surgical intervention. This is not an atypical scenario, and affords the perfect opportunity to suggest IR as an alternative to extraction (the patient desires to retain their tooth, but is unwilling to present for another invasive procedure). An example of this is outlined in a case report,[73] detailing the endodontic treatment of a mandibular second molar with suspected C-shaped anatomy and persistent symptoms.

Maxillary Sinusitis

Sinus complications secondary to an endodontic infection are not unusual. Resolution generally coincides with correct diagnosis and root canal therapy. In a case report,[89] the patient presented with persistent, painful symptoms eight months post endodontic treatment, and sinus occlusion associated with the tooth was evident on a CT scan. The surgery began as an *in vivo* approach, and the tooth was extracted upon suspicion of a vertical fracture. Upon close examination, the "fracture" was revealed to be a radicular defect, and the IR was proposed. When the tooth was extracted, a purulent discharge was observed. Prior to replantation, the alveolus and affected sinus membranes were lightly curetted and rinsed with saline. The root ends were prepared and sealed with amalgam root end fillings, and the tooth secured postreplantation with 3/0 silk sutures. Healing was uneventful, and the patient was comfortable and symptom-free two years postoperatively.

Paresthesia

Paresthesia is a classical sensory response resulting from neuropraxia, and is defined as "a burning or prickling sensation or partial numbness caused by neural injury."[134] Inferior Alveolar Nerve (IAN) injuries can arise from many causes, both disease based and iatrogenically induced. The most common endodontic etiologies are over-instrumentation, irritation from root canal medicaments, over-extension of root canal obturative materials, and root end surgery.[135]

The mechanisms by which paresthesia can occur are direct mechanical disturbances via over-instrumentation, neurotoxic effect of a chemical applied, or introduced, and pressure edema of a foreign object inserted into the neurovascular canal.[136] In the instances of over-instrumentation/over-extension of obturation materials, it is imperative to relieve the edema and harvest the material from the proximity of the bundle as quickly as possible. Delay in decompression and retrieval of the offending material can inhibit, or prevent, a successful recovery of sensation and function. Figure 25-13 demonstrates this very scenario. The patient had undergone conventional therapy, with under-instrumentation of the mesial canals, and over-instrumentation of the distal. Patient experienced a mild paresthesia after the first visit that degraded to a dysesthesia following the final obturation. The condition persisted three weeks after completion of endodontic treatment. Periapical surgery was contraindicated due to the proximity to the mandibular canal, and the preexistence of neural injury. The patient was apprised of IR. Possible complications were explained, and the patient elected to have the procedure performed. The procedure was completed, with a minor complication, and her dysesthesia resolved within three weeks. She was symptom-free after three months, and remained so throughout her subsequent recall appointments.

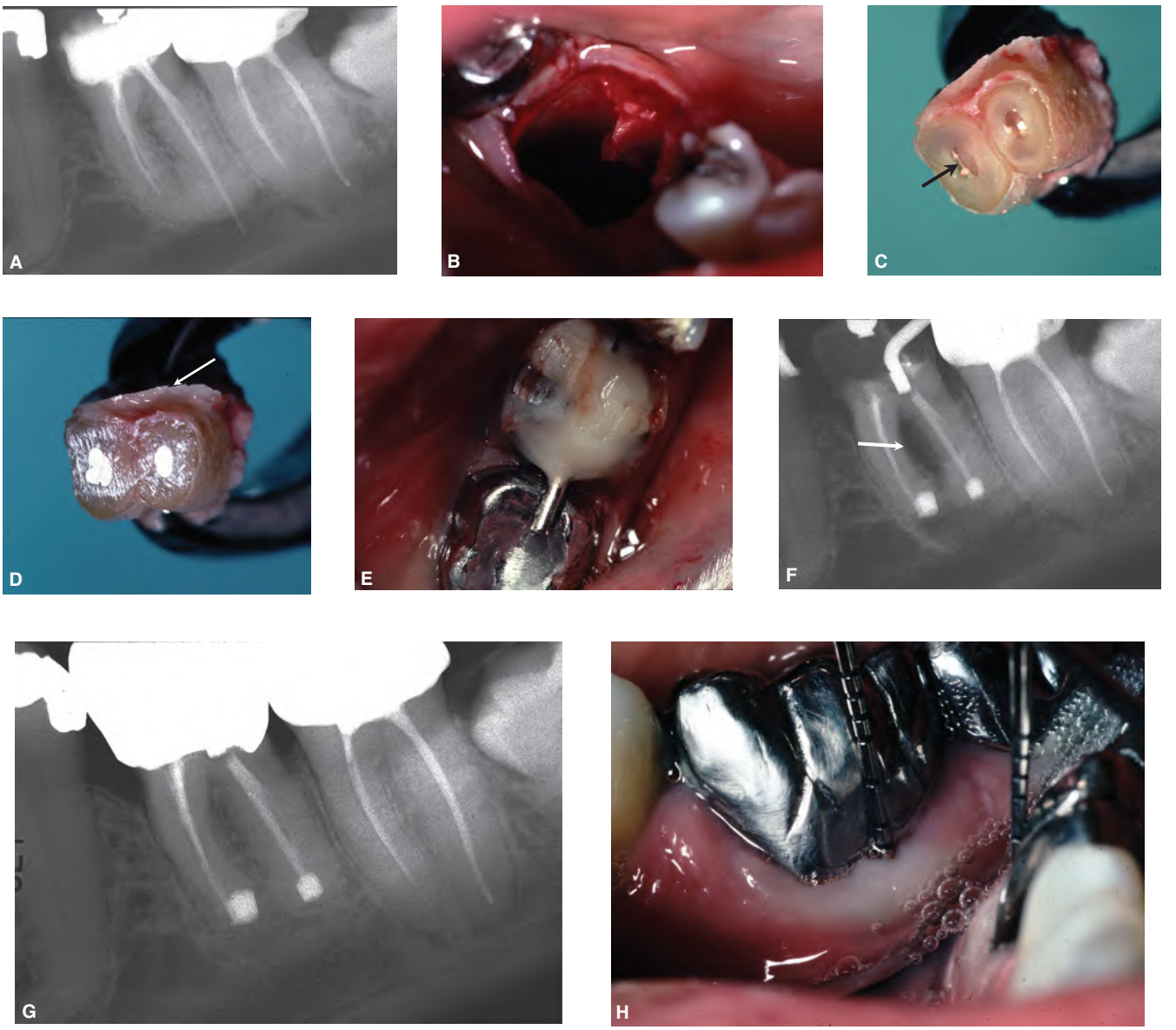

FIGURE 25-13 Intentional replantation in mandibular left first molar with dysethesia following root canal treatment. ***A.*** Preoperative radiograph. Evidence of over-instrumentation/overfilling in the distal canal, and underfilling/blockage in the mesial canals. The patient has had dysethesia since the completion of the RCT, and the apices may be in contact with the mandibular canal (radiographically). Conventional apical surgery is very risky, and may result in a permanent neurological deficit. ***B.*** During the extraction, a "snap" was heard. The tooth was delivered in whole, with no evidence of root fracture. Examination of the socket revealed that the buccal cortical plate fractured from the furcation, and was retained on the surface of the extracted tooth. The lingual portion of the plate will function as an index for the replanted tooth. ***C.*** Root-end resection and preparation has been completed. Note the isthmus between the MB and ML canals in the mesial root (arrow). ***D.*** Root-end fillings (Zinc Oxide Eugenol) in the mesial and distal root-end preparations. The buccal plate (arrow) inhibited replantation and was removed. The tooth will now require rigid splinting. ***E.*** The tooth has been replanted, and an orthodontic wire is secured in the access space, bent to index with the mesial marginal ridge of adjacent second molar. This will prevent a buccal luxation of the tooth, and the lingual index of bone prevents an occlusal displacement from the socket. ***F.*** Immediate postreplantation radiograph. The tooth is reduced in the alveolus to the correct depth, and will remain splinted for four to five weeks. Note the radiolucent area in the furcation corresponding to the loss of the buccal cortical plate (arrow). ***G.*** Eight-year recall radiograph. The bone has reformed in the furcation, and crowns have been placed on the first and second molars. The dysethesia resolved within three weeks, and normal sensation returned within six months. The patient is asymptomatic despite the poor endodontic treatment in the second molar; the patient declines any retreatment of the tooth, electing instead to have the IR done in case it becomes symptomatic. ***H.*** Periodontal probing depth of the buccal sulcus at an eight-year recall visit; 1.5 mm depth recorded in the furcation where the cortical plate was lost, indicative of regeneration of the plate.

CONCLUSION

Clinical experiences with the modified IR technique are represented by these two graphs (Figures 25-11 and 25-14). It is no surprise that the majority of the teeth chosen for this surgical procedure were mandibular molars. A survey of the case reports in the literature are overwhelmingly from that location. The combination of a thick cortical plate of bone and complex apical anatomy can present significant challenges to an in situ correction. Providing the tooth can be extracted without incident, it can afford unencumbered visualization and access to the root end, thereby increasing the chances for a successful outcome.

With the exception of maxillary molars, the observed long-term healing rates rival those reported for more conventional microsurgical approaches. The reasons for the lower success rates encountered with maxillary molars may be multifactorial; more delicate root anatomy, divergent curvatures and complex furcation configurations all combine to thwart atraumatic extraction and uneventful reattachment. Until a more predictable method can be developed to safely remove these teeth, an accurate three-dimensional survey preoperatively will either enhance the planned extraction, or discount that tooth from the procedure.

The IR technique has been shown to be a valid clinical alternative to extraction. In select situations where a conventional *in vivo* surgical approach is difficult or places the patient at unacceptable risk, the IR technique serves to expand the potential treatment alternatives while permitting the patient to retain their natural tooth.

Good prognosis of IR can be achieved provided that the following principles are followed:

1. Crushing or scraping contact with the root surface or socket lumen should be avoided.
2. The exposed root surfaces must be continually hydrated with the HBSS.
3. The tooth should be supported postoperatively to enhance reattachment.
4. Soft diet and hygiene instructions must be reinforced and strictly followed.

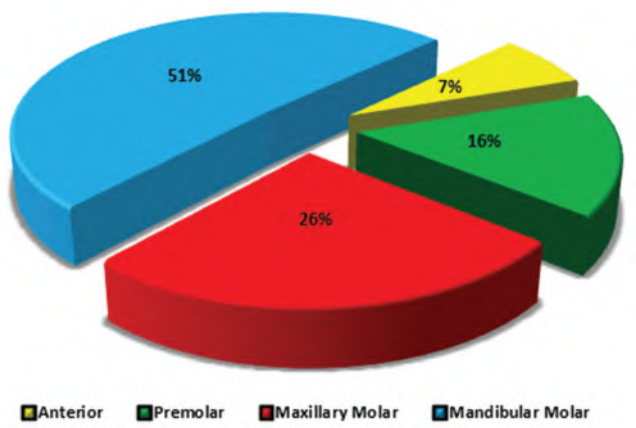

FIGURE 25-14 Graph demonstrating the distribution of intentionally replanted teeth by tooth groups. The majority of the teeth undergoing this procedure are mandibular second molars. Maxillary second and third molars comprise of about 25% of teeth treated.

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Chapter 26

Endodontic Therapy in Teeth with Anatomical Variations

Ilan Rotstein, Eduardo Llamosas, Kyung-Soo Choi

Teeth with anatomical variations can present a challenge to the clinician when endodontic diagnosis, case selection, treatment, and prognosis assessment are required. While not common, these variations are definitely not rare.

Dental anatomical variations are classified according to their size, shape, number, structure, or growth alterations.[1,2] Variations in ***shape*** are the ones most commonly referred for treatment in an endodontic practice. In many such cases, long-term success of endodontic treatment is obtained, and the natural tooth may be retained.

This chapter discusses the most common anatomically shaped variations treated in an endodontic practice. For more details on radicular anatomy and morphology, common and uncommon, see Chapter 1, "Anatomy and morphology of teeth and their root canals."

GEMINATION AND FUSION

Definitions

Gemination is an anomaly resulting from abnormal division attempt of a single tooth bud. ***Fusion*** is a union of two, or more, separately developing tooth buds.[3,4]

Teeth that are fused or joined together by dentin have been referred to as *double teeth*, *fused teeth*, *joined teeth, linking teeth*, *twinning dichotomy*, *connation, geminfusion*, *vicinifusion, synodontia*, *schizodontia*, *mirror-image double teeth*, and *germinated composite odontomes*.[5–8]

Characteristics

Clinically, it is often difficult to differentiate between gemination and fusion. Gemination usually presents as a tooth with two crowns, either totally or partially separated, with a single root and one canal.[9] Fused teeth may have more than one canal system.

Often, the total number of teeth in the arch can provide a clue. Generally, if a tooth is missing from the arch, it is reasonable to assume that the affected tooth is fused.[10–12] However, in cases of fusion with a supernumerary tooth (Figure 26-1), this diagnostic method is not accurate.

Mader[7] suggested a "two-tooth rule" to differentiate between gemination and fusion. If the resulting anomaly is counted as two teeth and the normal number of teeth is present, then fusion is suggested. If it is counted as two teeth and an extra tooth is present, then gemination or fusion between a normal and supernumerary tooth is suggested. However, in adult dentition, an exact differentiation between gemination and fusion may not be possible with absolute certainty because of the clinical similarity between the two. Furthermore, it has little clinical significance for endodontic treatment.[13,14] The mere recognition of these anomalies should suffice to enhance vigilance and consideration of treatment options.[15]

Geminated and fused teeth have bifid crowns, or grooved incisal edges, in a buccolingual direction that are continuous with well-defined vertically running grooves on the buccal and lingual surfaces.[16] This gives the appearance of two or more separate structures with varying degrees of conjunction between the dental units.[5] The conjunction may occur either in the crown or root; sometimes in both. This depends on the developmental stage of the tooth/teeth when the anomaly occurred.[17] Radiographically, a single broad root with a common pulp canal space is visible.[4]

There is usually a union between the dentin of fused teeth. In some cases of complete fusion, there may be a single pulp chamber and root canal. The pulp chamber may be continuous, whereas the root canals are separate. On the other hand, it may have a discrete pulp chamber and root canals. The dentin, however, throughout the fused tooth, is not uniform. The interstitium between the root canal systems, with a common pulp chamber, may have dentin that closely resembles irregular secondary dentin, with numerous vascular channels, named vasodentin.[16]

Etiology

The exact etiology of gemination and fusion is not completely clear, however, developmental aberration of both ectoderm and mesoderm is involved.[6] There are several theories attempting to explain their formation. One theory suggested that fusion occurs purely by chance,[18] whereas others proposed that contacts of individual tooth germs is brought about by a physical force or pressure, acting on the tooth germs, leading to necrosis of the intervening tissues.[16] This allows the enamel organ and dental papillae of two or more adjacent teeth to fuse. If fusion takes place, prior to the commencement of calcification, it is regarded as complete, whereas if it happens thereafter it is regarded as partial. Other theories have suggested genetic predisposition, nutrition factors, and local metabolic interferences as the etiology.[19–21]

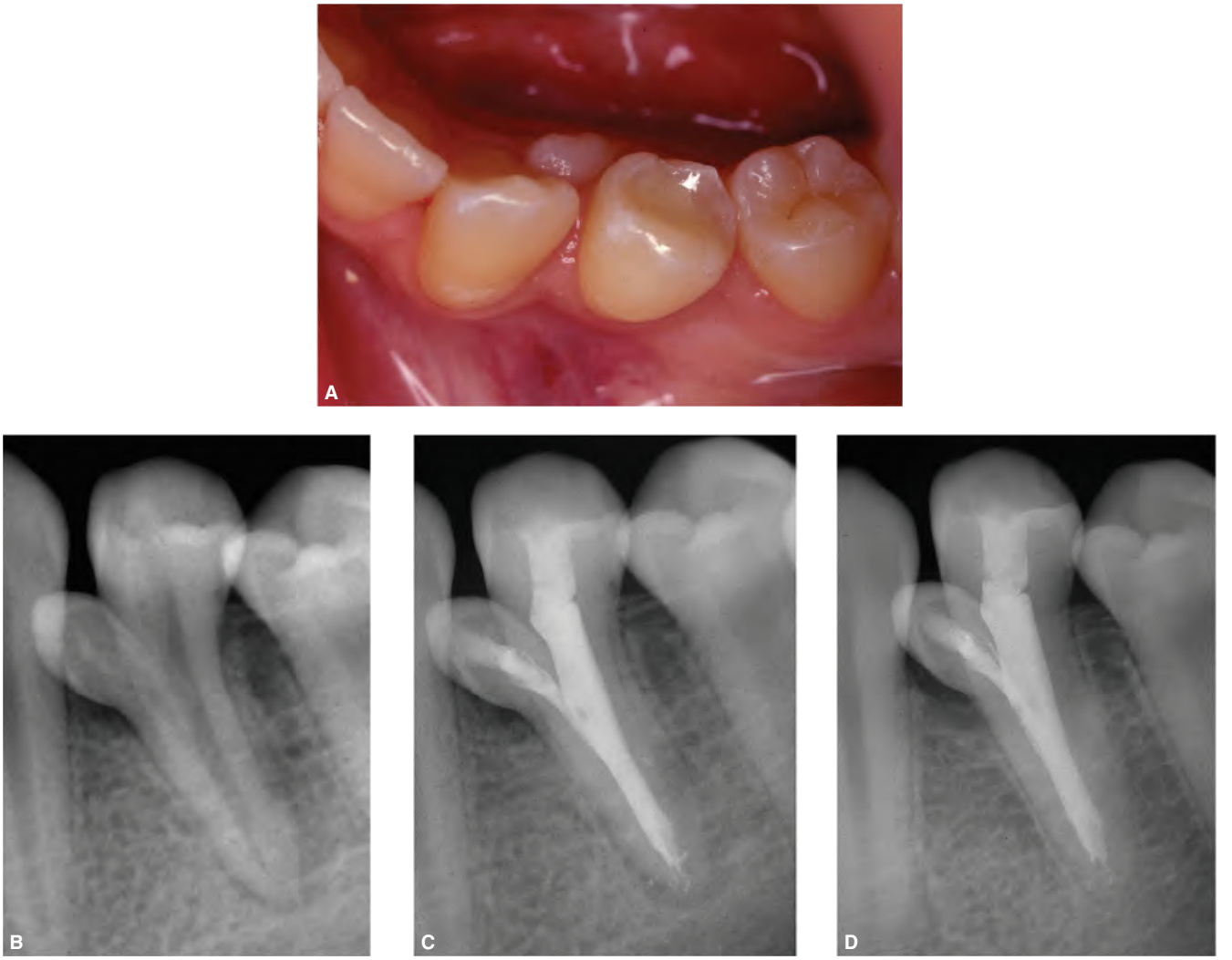

FIGURE 26-1 Fusion of a mandibular first premolar with a supernumerary tooth in a 15-year-old male. ***A.*** Clinical view. The crown of the supernumerary tooth is located mesio-lingually to the premolar. ***B.*** Preoperative radiograph showing the fused teeth with a joint root canal system. Periapical radiolucency is evident. Pulp vitality tests were negative. ***C.*** Immediate post operative radiograph. ***D.*** Four-year follow-up radiograph showing resolution of the periapical radiolucency. (Courtesy of Dr. Fernando Goldberg, Buenos Aires, Argentina.)

Prevalence

The reported prevalence, in the primary dentition, varies between 0.5%-2.5%.[22,23] In the permanent dentition, however, it is estimated to vary between 0.1%-1%.[5,24,25] Gemination and fusion occur predominantly in incisors and canines with apparent equal distribution between the maxilla and the mandible[25] as well as between males and females.[8] Occurrence in the posterior arch (Figure 26-2) is less frequent than in the anterior arch.[25] Bilateral occurrence is rare, not exceeding 0.6% in the permanent dentition.[26]

Clinical Implications

Teeth affected with this anomaly are usually wider and often have a distinctive groove on the crown in the union between the dental units involved. This causes an esthetic defect as well as arch spacing alterations such as diastema, loss of proximal contact, tooth crowding, and even impaction of neighboring teeth.[27] Consequently, malocclusions and functional impediments may develop.[7] When the grooves are deep, plaque can easily accumulate, making the tooth more susceptible to caries and periodontal disease.[28]

Numerous treatment options have been proposed including extraction, recontouring, reduction of tooth size, or hemisection.[3,21] Surgically dividing or reducing the size of the tooth may result in pulpal exposure due to microscopic extensions in the pulp chamber occupying the dentin adjacent to the main pulp chamber, in an irregular manner, requiring endodontic treatment.[14,29]

Careful clinical examination and a thorough imaging analysis are necessary to assess the complex internal anatomy and root morphology of the tooth, before starting endodontic treatment. Cone beam computed tomography (CBCT) could be advantageous.[30]

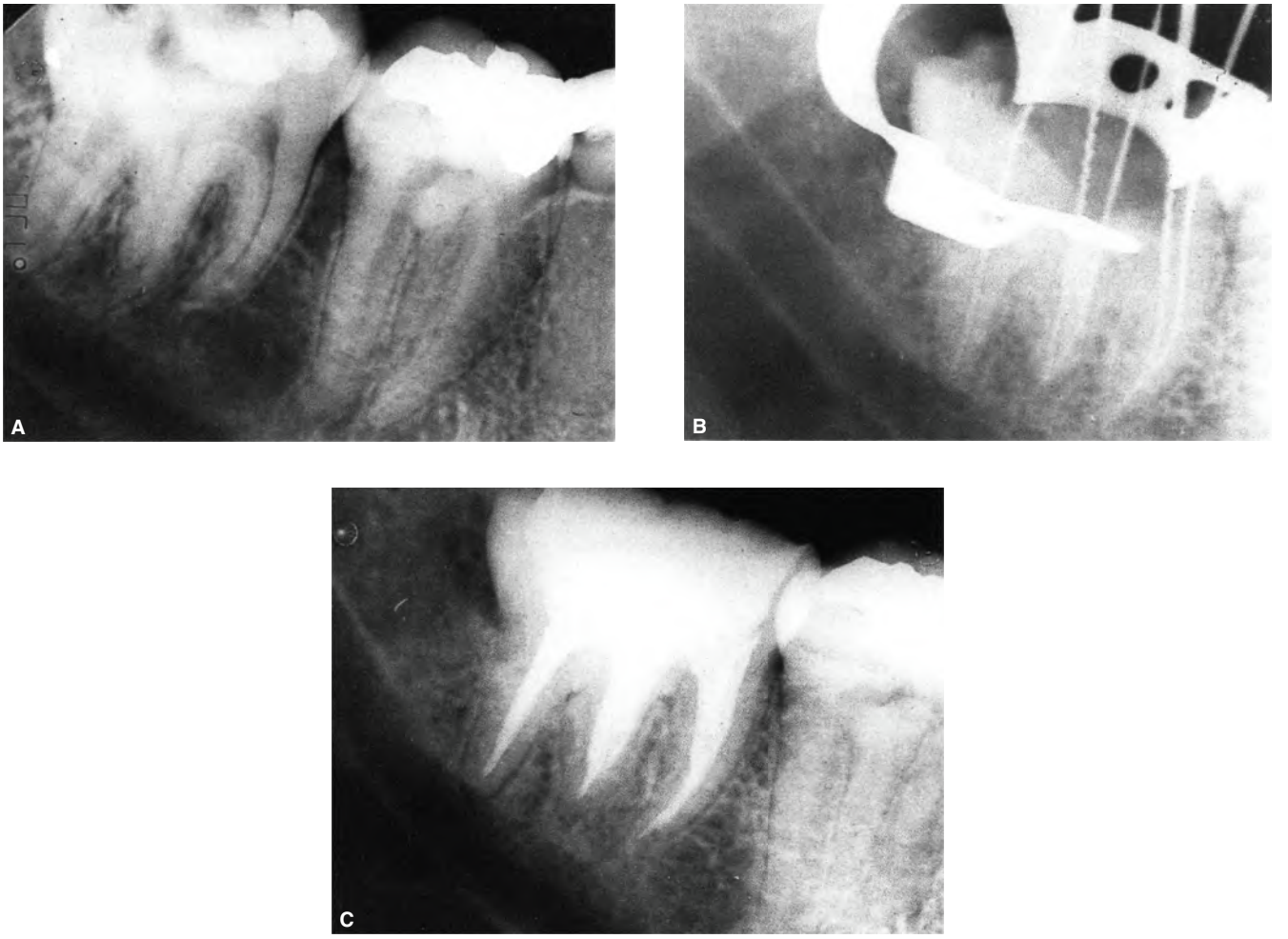

FIGURE 26-2 Fused mandibular right second molar in a 25-year-old male. The patient complained of irradiating pain to the right ear. The tooth was tender to percussion and the surrounding mucosa was slightly inflamed. ***A.*** Preoperative radiograph. ***B.*** Working length assessment radiograph showing five endodontic instruments in five separate root canals. There were two canal orifices in the mesial aspect of the pulp chamber floor, two on the middle and one on the distal aspect. ***C.*** Immediate postoperative radiograph of root canal obturation with gutta-percha and sealer. (Courtesy of Dr. Ilan Rotstein, Los Angles, CA, U.S.A. Adapted with permission from Rotstein et al.[31])

Whenever a gemination/fused tooth is treated endodontically, the root canal should be considered as one system, even if a communication between the pulp chambers or root canal systems is initially not clinically discernible.[31] Separate endodontic access cavities may be necessary. Communication between the pulp chambers of geminated or fused teeth is a common feature.[15,32,33] Careful examination of the pulp chamber and removal of overlaying dentin will facilitate the location and treatment of all root canals.[31]

DILACERATION

Definition

Dilaceration (Latin: *dilacero* = tear up) is an angulation or a sharp bend in the root or crown of a formed tooth.[1,2] The term *scorpion tooth* was suggested when the dilaceration is labial, and *bayonet* when the tooth presents more than one dilaceration.[34]

Characteristics

Dilaceration is characterized by an abrupt deviation in the longitudinal axis of a tooth.[34] It may affect any part of the tooth and follow any direction[35] (Figure 26-3). It is observed in both erupted and unerupted teeth and may be associated with prolonged retention of the primary predecessor, or with apical fenestration of the labial cortical plate.[37]

A dilacerated tooth can become ectopic, or not erupt, because the longitudinal axes of the root and crown are not aligned.[34] The more apically and less severe the dilaceration, the greater is the chance for spontaneous tooth eruption.[38]

Coronal dilaceration may be observed visually, however, radicular dilaceration requires radiographic images.[39] Radiographically, if the root is curved mesially or distally, the dilaceration is clearly apparent. If the dilaceration is directed labially or lingually, the root may not appear severely curved. When the root is curved labially, it may present a rounded opaque area with a dark spot in its center,

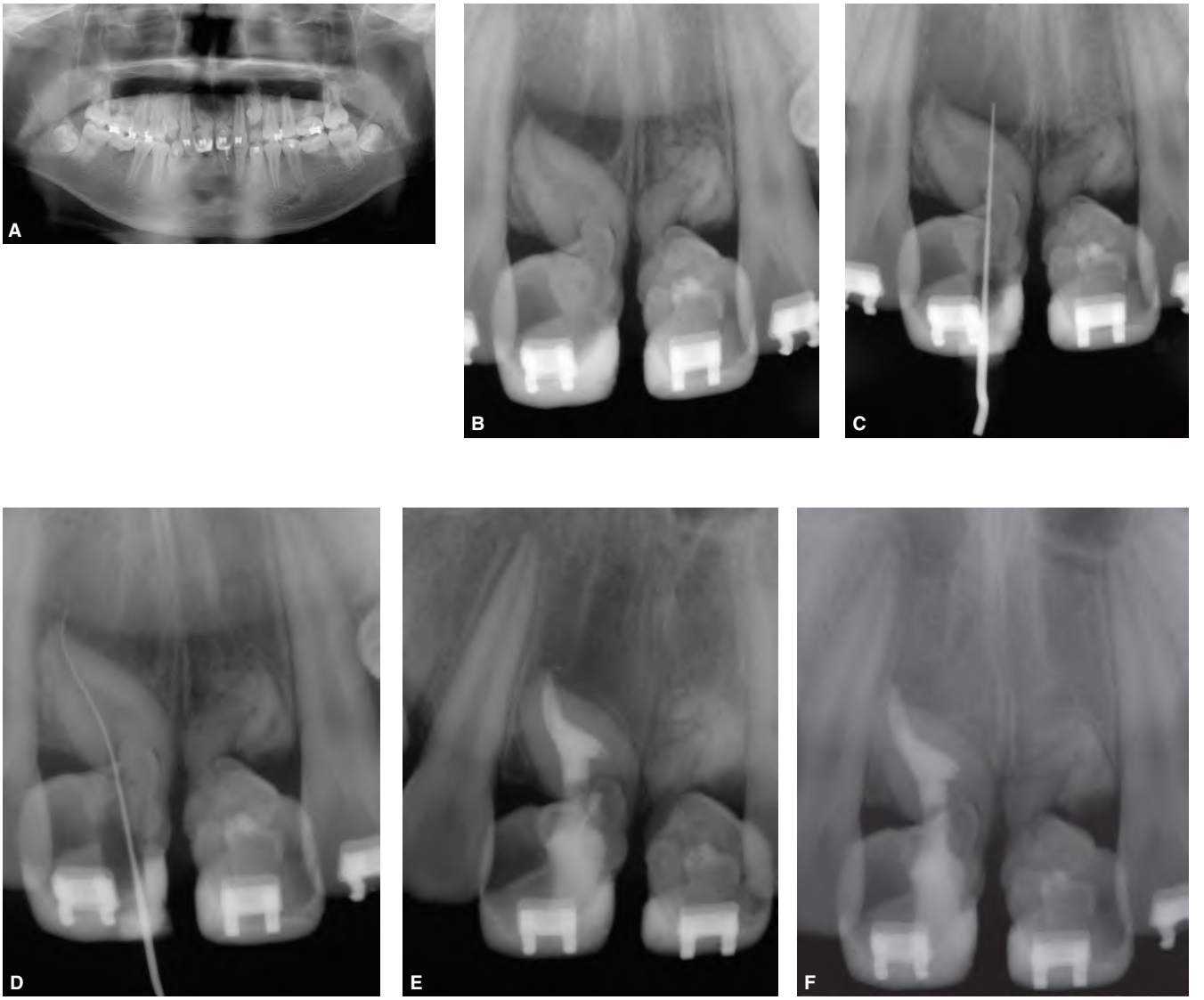

FIGURE 26-3 Endodontic treatment in a dilacerated maxillary right central incisor of a 12-year-old male. The tooth showed class II mobility with swelling and a sinus tract in the buccal mucosa and periapical radiolucency. The tooth was previously treated by the referring dentist. The palatal access cavity was sealed with a temporary filling material. ***A.*** Panoramic radiograph showing multiple dental anomalies. Medical history revealed that the patient suffered from cerebral meningitis at four months after birth and cerebral subdural emphysema at six months that required head surgery and long-term anticonvulsant medication for one year. ***B.*** Preoperative radiograph showing the affected tooth associated with a large periapical radiolucency. ***C.*** Radiograph taken by the referring dentist. Endodontic file demonstrates a perforation at the cervical area of the root. Perforation occurred during attempts of the referring dentist to access the root canal. ***D.*** The perforation site was repaired with MTA and resin-modified glass ionomer and the canal was accessed correctly. Following cleaning and shaping, the canal was dressed with calcium hydroxide paste for a period of 4 months. ***E.*** Postoperative radiograph. The canal was obturated with gutta-percha and sealer and the coronal access restored with light-cured composite resin. ***F.*** Three-month recall radiograph following obturation. The periapical radiolucency has resolved and the tooth was asymptomatic. (Courtesy of Dr. S-Y Kim, Seoul, Korea. Adapted with permission from Byun C et al.[36])

showing the apical foramen.[37] This appearance is frequently referred to as a *bull's eye* or *target*.[40]

The periodontal ligament space around the dilacerated portion of the root may appear as a radiolucent halo. The radiopacity of the dilacerated segment appears greater than the rest of the root. This is due to increased thickness of the tooth structure that the x-ray beam has to transverse.[37,41] Additional radiographs, from different angles, can assist in diagnosis.[42]

Etiology

It has been suggested that the cause of dilaceration is mechanical trauma disturbing the relations between the calcified and noncalcified portions of the tooth during development.[43,44] This hypothesis proposes that an acute mechanical injury to the primary predecessor may cause the calcified part of the permanent tooth germ to displace in such a manner that the remainder of the noncalcified part of the permanent

tooth germ forms an angle.[45,46] Some authors propose that trauma may result in an imbalance of dentinogenesis causing the Hertwig's epithelial root sheath to produce dentin in a certain area at a markedly reduced rate.[47] However, this may not be the only etiologic factor. In several reported cases of trauma to the predecessor primary teeth, root formation of the succedaneous permanent teeth has not yet been started.[48]

Dilacerations have been also reported in patients without history of trauma.[39,49] High prevalence of dilacerations in mandibular third molars suggests that ectopic development of the tooth germ and lack of space, rather than trauma, may play an important role.

Other factors that may contribute to the formation of dilaceration include scar formation, developmental anomaly of the tooth germ, facial clefting, infection, cysts, tumors, premature extraction of primary teeth, ankylosed primary teeth, hereditary factors as well as anatomical structures such as cortical bone of the maxillary sinus, mandibular canal, or the nasal fossa.[37] Certain syndromes and developmental disorders, such as Smith-Magenis syndrome, hypermobility type of Ehlers-Danlos syndrome, Axenfeld-Rieger syndrome, and congenital ichchyosis, have also been implicated.[48]

Prevalence

Dilaceration may be found in both permanent and primary teeth, yet the prevalence in the latter is much lower.[45,50,51] Radicular dilacerations are more common than coronal dilacerations[37] (Figure 26-4).

The reported prevalence of radicular dilaceration depends on its exact definition. Reports vary considerably, ranging from 1.8% to 98%.[42,52] Different criteria for defining root dilacerations may explain the vast difference in the reported prevalence. When a 20-degree angle or greater deviation from the long axis of the tooth was used as criteria, the reported prevalence of root dilacerations in maxillary lateral incisors was 98%.[52] However, when a 90-degree angle or greater was used as criteria, the prevalence dropped to less than 7%.[39,49]

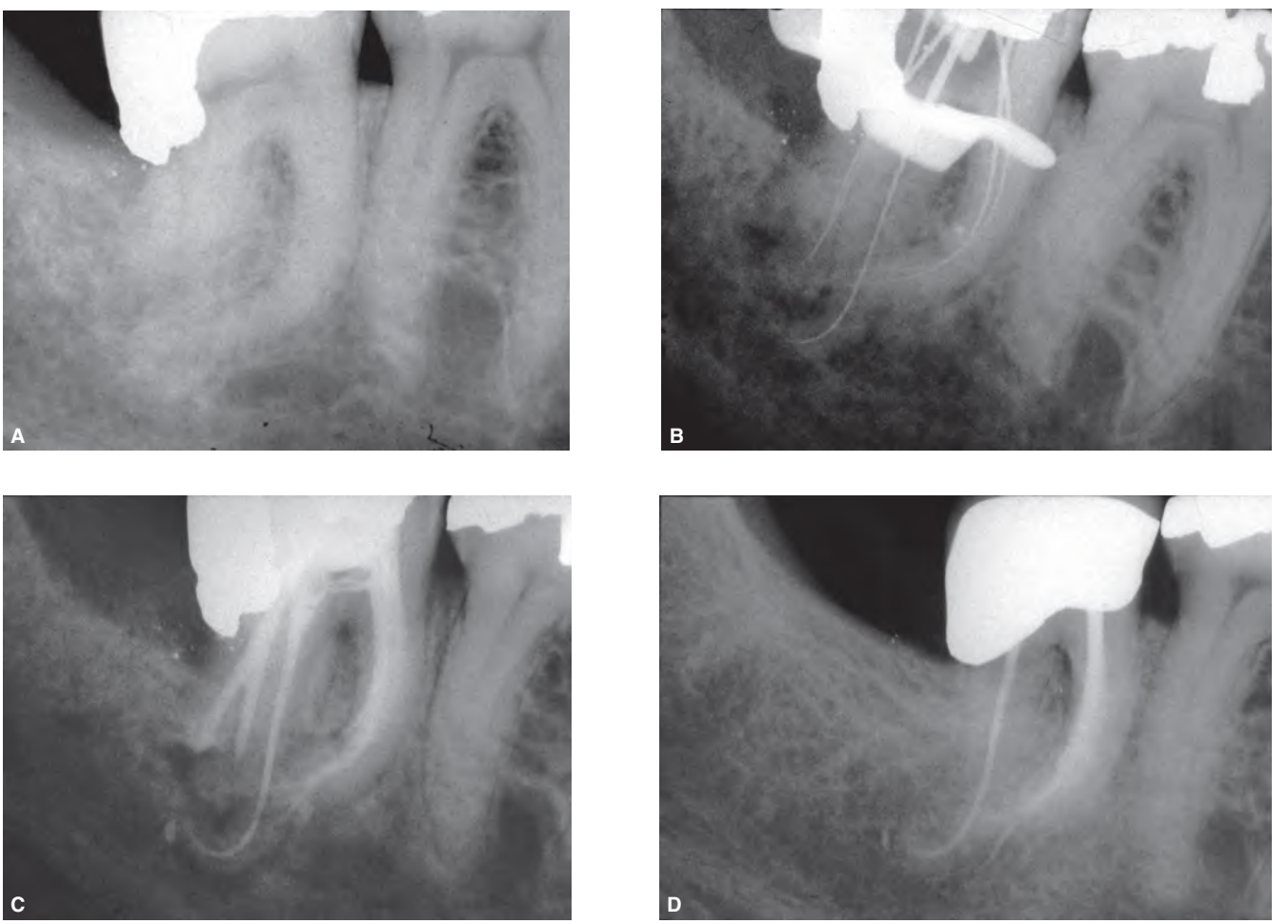

FIGURE 26-4 Endodontic treatment in severely curved roots of a mandibular second molar of a 38-year-old male. The patient complained of lingering pain to cold stimuli and the tooth was painful to percussion. ***A.*** Preoperative radiograph. ***B.*** Length assessment radiograph showing instruments in five separate canals and severe root curvature. ***C.*** Immediate postoperative radiograph. ***D.*** 11-year recall radiograph. The distobuccal/lingual root complex was resected two years prior because of deepening periodontal pocketing. The patient is asymptomatic and the tooth is stable and restored to function. (Courtesy of Dr. Stephen P. Niemczyk, Broomall, PA.)

Root dilaceration is more frequently observed in the apical third of the root in incisors, canines and premolars, in the middle third of the root in first and second molars, and in the radicular cervical third of the root in third molars.[49]

Clinical Implications

An erupted dilacerated tooth requiring root canal treatment may present some clinical challenges to the clinician.[53] Diagnosing root dilacerations before initiating endodontic treatment is important to prevent potential procedural complications. Preoperative radiographs from different angles should be taken to recognize the multiplanar nature of the dilaceration. CBCT can confirm the diagnostic findings and help to recognize a dilaceration that may not be completely identified by conventional 2-D periapical radiographs.[54, 55]

Straight-line access cavity preparation is essential for the safe use of endodontic instruments. Sufficient tooth structure needs be removed in order to allow endodontic instruments to move freely. The use of a scouting file will provide critical information about the extent and direction of the dilaceration. Copious irrigation and frequent file recapitulation are recommended.[37,56] Rotary instruments should be used with great caution.

DENS INVAGINATUS

Definition

Dens invaginatus is a developmental malformation resembling the appearance of a "tooth within a tooth". Many terms have been used to describe this anomaly, such as *dens in dente*, *dilated composite odontome*, *gestant odotome*, *invaginated odontome*, *tooth inclusion*, and *dentoid in dente*.[1,2] Dens invaginatus, however, is considered a more accurate terminology since it describes a wider range of variations of this tooth anomaly.[57,58]

Characteristics

Dens invaginatus presents a wide variety of morphological variations affecting the tooth crown, root, or both (Figures 26-5 to 26-7). The shape of the crown may vary from a normal appearance,[59] exaggerated or bifid cingulum, hypoplastic appearance,[60] to a substantial deep infold that reaches the apical foramen.[61] Other crown malformations such as peg shape (Tsurumachi 2004), barrel shape,[62] conical shape,[63] and increased labio-lingual diameter[64] have been described.

Dens invaginatus is usually detected during routine radiographic examination.[65] It may be symmetric or asymmetric and occur either unilaterally or bilaterally.[66–69] An invagination shape, varying from a narrow and undilated fissure to a tear-shape loop pointing towards the main body of the pulp, may be detected on the radiograph.[69,70] The invagination may appear as a radiolucent pocket surrounded by a radiopaque border of equal density to the enamel and extending from the crown toward the root canal.[41] It may be completely separate from the pulp (pseudocanal) and manifested as a deep enamel-lined fissure with its own opening into the periodontal ligament. In such cases, response to pulp sensibility tests can remain normal despite the presence of a periapical radiolucent lesion associated with the psuedocanal.

Different classifications have been suggested to describe dens invaginatus.[66,71,72] The most commonly used was proposed by Oehlers,[57] dividing it to 3 types according to depth of penetration and communication with the periapical tissues or periodontal ligament. In t*ype I*, an enamel lined minor invagination occurs within the coronal part of the crown without extending beyond the cemento-enamel junction. In *type II*, an enamel lined invagination extends into the root, beyond the cemento-enamel junction and remains as a blind sac without reaching the periodontal ligament, or periapical tissues. In *type III*, an invagination penetrating through the root perforates the apical or the periodontal area. There is no immediate communication with the pulp. The invagination may be completely or partially covered by a thin layer of enamel or cementum.

Etiology

The etiology of dens invaginatus is controversial.[60,65,66] It was suggested that it may result from infold of the enamel organ into the dental papilla prior to the calcification stage of tooth development.[66] Rushton[73] hypothesized that the invagination may be a result of rapid and aggressive proliferation of a part of the internal enamel epithelium invading the dental papilla. On the other hand, Kronfeld[74] suggested that the invagination was the result of retardation, or focal growth failure of the internal enamel epithelium while the surrounding epithelium continued to proliferate normally and engulfing the static area. Atkinso[75] suggested that the invagination was the result of external forces, from growth pressure during development of adjacent tooth germs, causing the enamel organ to buckle. Other external factors such as trauma and infection were also suggested.[1,2]

Dens invaginatus was associated with certain genetic disorders.[65,68,76,77] Other dental anomalies, such as microdontia and hypodontia, were also associated with dens invaginatus. It therefore appears that genetic factors may play a certain role in the occurrence of dens invaginatus.

Prevalence

The reported prevalence of dens invaginatus, in the permanent dentition, varies from 0.17%[78] to 26.1%.[42] It is more common in the primary dentition. Maxillary lateral incisors are most frequently affected.[79,80] Kirzioglu and Ceyhan[80] reported that maxillary lateral incisors were the most affected teeth (62%), followed by maxillary central incisors (35%) and maxillary canines (3%). Occasionally, permanent premolars may be affected.[81] However, posterior permanent teeth are less likely to be involved.[82–84]

Using Oehlers' classification, *type I* occurs most frequently.[60] Kirzioglu and Ceyhan[80] reported an overall

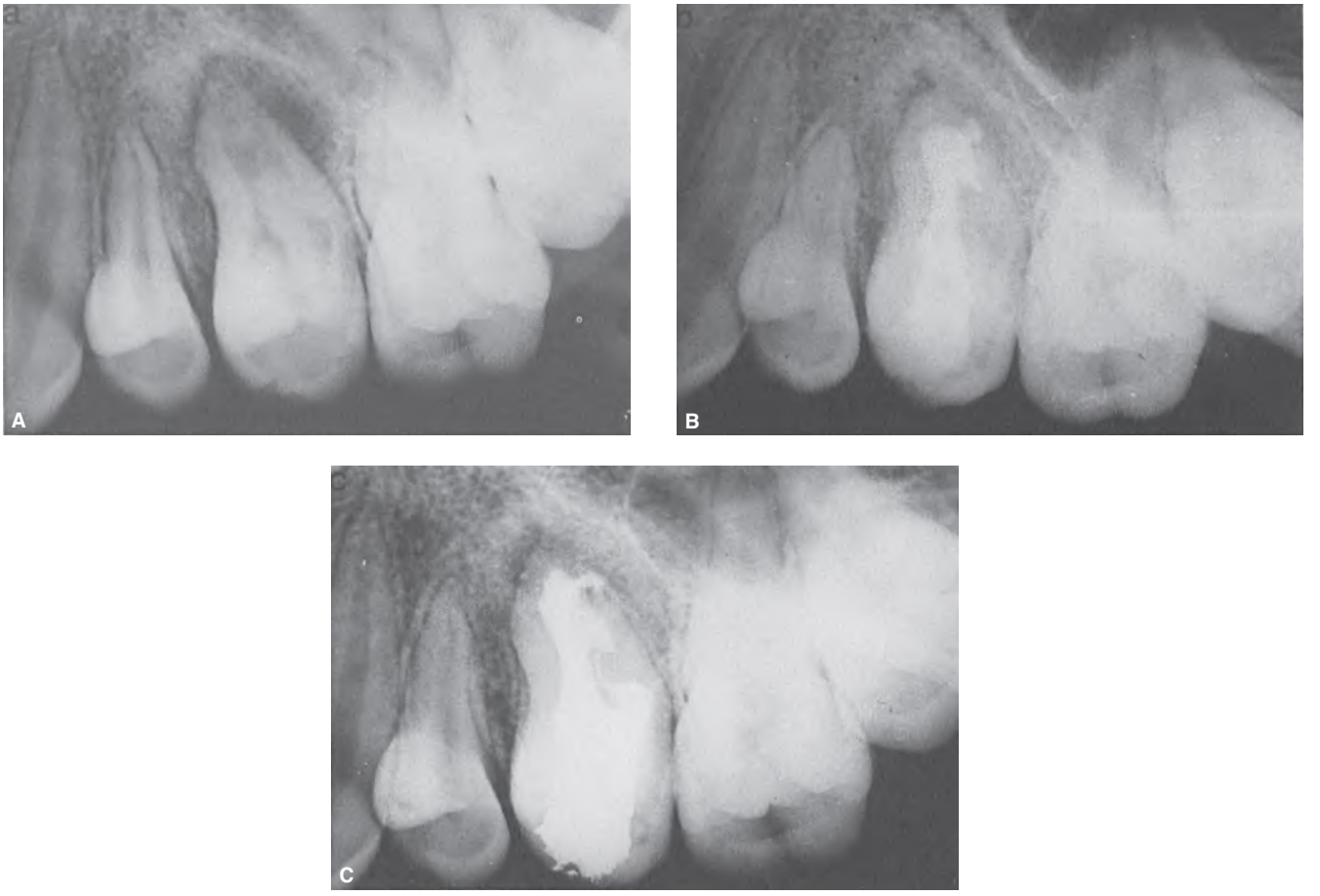

FIGURE 26-5 Endodontic therapy of dens invaginatus in a permanent maxillary second premolar of a 14-year-old female. Pulp sensibility tests were negative. ***A.*** Preoperative radiograph showing large periapical radiolucency associated with the tooth. ***B.*** Six months following introduction of calcium hydroxide to facilitate apical closure. Note considerable reduction in size of the periapical radiolucency. The patient was asymptomatic. ***C.*** Postoperative radiograph. The complex root canal system was obturated with softened gutta-percha and sealer. The coronal access was restored. (Courtesy of Dr. Ilan Rotstein, Los Angles, California. Adapted with permission from Rotstein et al.[88])

prevalence of 94% for *type I*, 3% for *type II*, and 3% for *type III*. Colak et al.[78] found that *type I* occurred in 73% of cases, *type II* in 20% and *type III* in 7%.

Clinical Implications

The main clinical concern associated with dens invaginatus is the ingress of microorganisms and irritants into the pulp via the invaginated area where dentin is covered only by a thin layer of enamel. The dentin of the invaginated tissue may contain vital connective tissue,[85,86] or fine canals that communicate with the dental pulp.[57,73,74] This dentin may be hypo-mineralized, or irregularly structured.[72,85,87]

The structure and thickness of the enamel lining also varies.[75,87] The invagination may contain remnants of the dental papilla or periodontal connective tissue that becomes necrotic, and a nutrient-rich environment following microbial contamination from the mouth.[74,86]

Teeth affected with dens invaginatus are more prone to developing pulpal pathosis a short time after eruption. The risk of pulpal complication is due to the inherent poor anatomical features, both on a macro and microscopic level that encourage microbial contamination.[65] Early diagnosis and preventive treatment measures can reduce the future need for endodontic treatment.[60] Rotstein et al.[88] suggested early sealing of the invagination by means of restorative materials in cases where no pathosis has yet been detected. Use of dye such as methylene blue and high power magnification and illumination can help identify the openings. When the tooth does not present a detectable entrance clinically, and a minimal invagination is seen radiographically, acid-etched fissure sealant or flowable composite resin should be used.

Unerupted teeth with dens invaginatus (diagnosed by a radiograph) should be considered for preventive treatment as soon as practical after tooth eruption. When dens invaginatus is suspected, additional radiographs from different angulations should be taken.[58] CBCT can be helpful for diagnosis and accurate anatomic representation.[89]

When a periradicular pathosis is present but the pulp in the main canal is healthy, efforts should be made to preserve pulp vitality. In such cases, the invagination can be treated

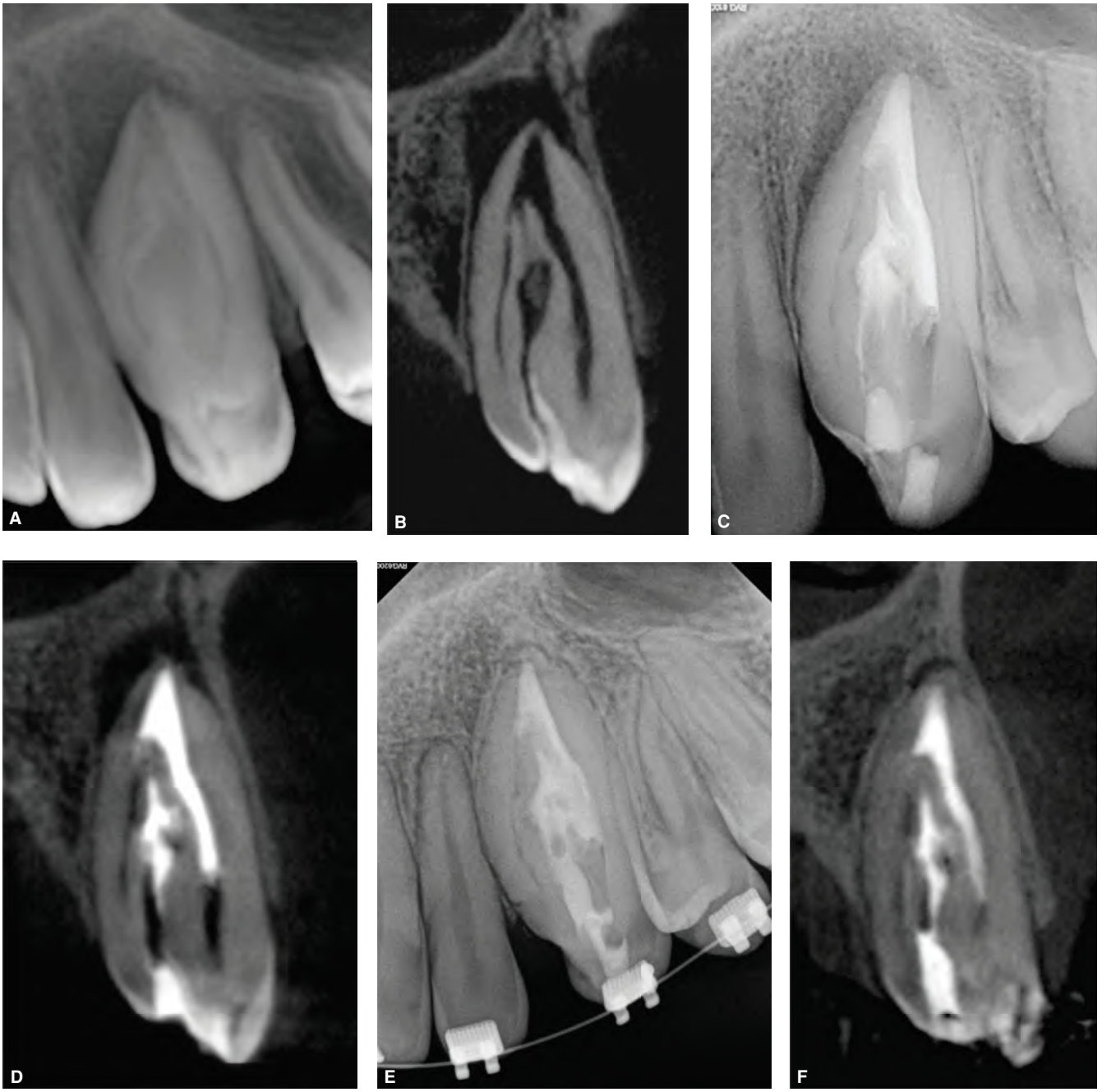

FIGURE 26-6 Dens invaginatus in a maxillary left canine of a 14-year-old male associated with traumatic injury 2 months previously. The tooth was painful to percussion and exhibited localized swelling. Pulp sensibility tests were negative. ***A.*** Preoperative radiograph showing periapical radiolucency associated with the tooth. ***B.*** Cone beam computed tomography (CBCT) sagittal cross-section was taken using a CS 9300 unit at 90 µm voxel size (Carestream Health, Rochester New York). Note, dens invaginatus type II with one foramen and evidence of associated periapical pathosis. Endodontic treatment was done. ***C & D.*** Immediated postoperative radiograph and sagittal CBCT cross-section. ***E & F.*** 28-month follow-up radiograph and CBCT sagittal cross-section showing evidence of bony healing. The patient is asymptomatic and undergoing uneventful orthodontic treatment. (Courtesy of Dr. Jose-Maria Malfaz, Valladolid, Spain.)

separately from the root canal.[58] When the pulp is necrotic, the entire root canal system needs to be treated. Occasionally, it may be possible to treat the root canal and invagination separately.[90–95] Care should be taken when joining the root canal with the invagination because it may weaken the tooth structure or compromise the apical constriction. Therefore, attempting to keep the invagination and root canal separately during endodontic treatment is advisable whenever possible.[58] Nevertheless, the proximity of the root canal to the invagination may often necessitate treating them both.

The large and irregular volume of the root canal system in dens invaginatus makes proper cleaning and shaping

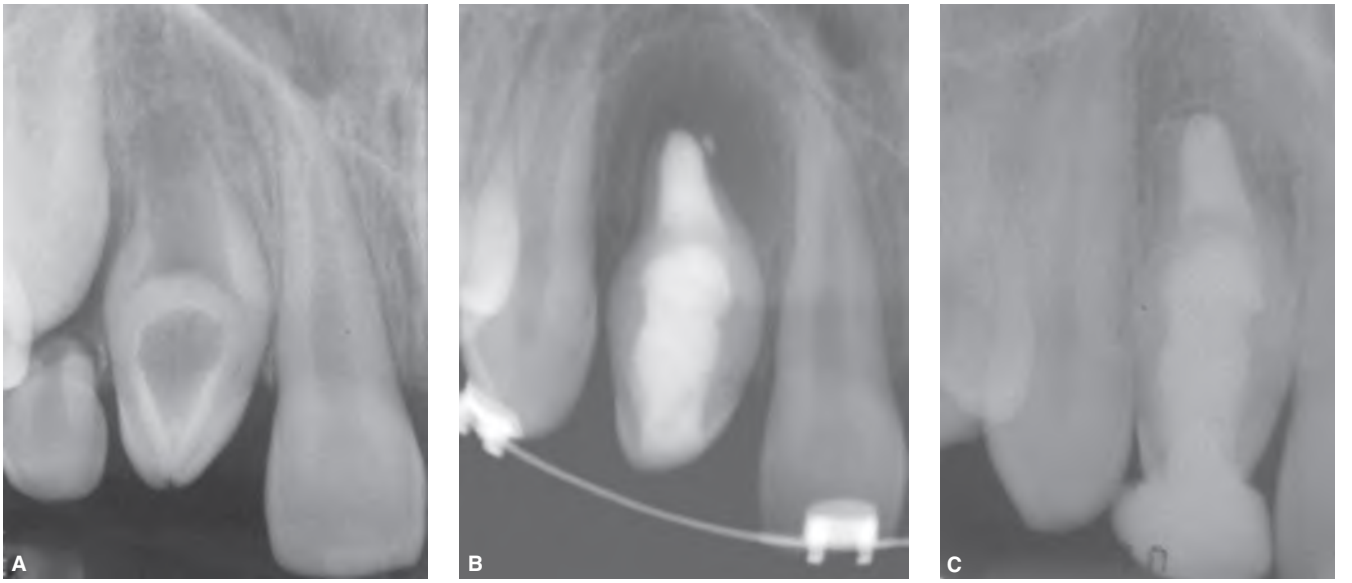

FIGURE 26-7 Dens invaginatus variant in a symptomatic maxillary left lateral incisor with necrotic pulp of a 9-year-old male. ***A.*** Periapical radiograph taken three years prior to endodontic treatment. ***B.*** Postoperative radiograph at age 12 showing large periapical radiolucency associated with the tooth. The root canal system was obturated with MTA and bonded core was placed. ***C.*** 11-year recall radiograph showing resolution of the periapical radiolucency. (Courtesy of Dr. Mark Olesen, North Vancouver, BC, Canada.)

difficult. The use of ultrasonics with irrigation for thorough chemo-mechanical preparation may be beneficial.[65] Surgical treatment should be considered when conservative treatment has failed.[88]

DENS EVAGINATUS

Definitions

Dens evaginatus is a developmental malformation presenting a tubercle, elevation, protuberance, excrescence, extrusion, or bulge from the involved surface of the affected tooth.[96,97] It is also referred to as an *interstitial cusp*, *tuberculated premolar*, *Leong's premolar*, *odotome of the axial core type*, *evaginated odontome*, *occlusal enamel pearl*, *occlusal anomalous tubercle*, and *supernumerary cusp*.[98,99] However, the term "dens evaginatus" is mostly used today because the protuberance can include enamel as well as dentin and pulp tissue.[100,101]

Usually, when the tubercle is present on the lingual surface of an anterior tooth, projecting either from the cingulum or the cementoenamel junction, the term *talon cusp* is used.[102,103] However, a talon cusp may also occur on the labial surface of anterior teeth.[104,105]

Dens evaginatus and talon cusp are basically the same tooth anomaly. Talon cusp is a subset of dens evaginatus presenting similar developmental, morphological, and histological characteristics.[96,106]

Characteristics

The tubercle of dens evaginatus appears as a pronounced elevation on the occlusal surface of premolars often in the form of a drop, nipple, or a pointed or cylindrical cone of varying size. It is most often located between the buccal and lingual cusps, usually on the buccal triangular ridge or in the central groove.[107] The location of the tubercle on the occlusal surface may vary from the central groove to the inclined plane of the buccal cusp.[6]

The tubercle dimensions in posterior teeth have an average of 2 mm width and up to 3.5 mm length.[97,108] Other than the cusp-like variable sized and shaped tubercles of teeth with dens evaginatus, the remaining portion of the crown presents a normal anatomy.[97,107] The tubercle often contains variant forms and extensions of pulp tissue that are surrounded by dentin and enamel.[100,101] Dens evaginatus is considered as the antithesis of dens invaginatus.[109]

When tubercles occur in anterior teeth (talon cusp), the projection may appear as an exaggerated cingulum, or a prominent well-defined accessory cusp, that can extend beyond the incisal edge, forming a T-shaped or a Y-shaped crown contour.[102,104,110] Tubercle dimensions in anterior teeth are of 3.5 mm width and 6.0 mm length on average.[111] A talon cusp is delineated by deep developmental fissures or grooves, that accumulate plaque and can become susceptible to caries.[103]

Etiology

The exact etiology is not completely understood. The anomaly is considered a result of an outward folding of the inner enamel epithelial cells proliferating into the stellate reticulum of the enamel organ or due to a transient focal hyperplasia of the mesenchymal peripheral cells of the dental papilla.[101,103] It has been viewed as one variation of a range of hyperactivity of the dental lamina.[112,113]

It has been suggested that both autosomal dominant and chromosome X-linked traits may play a role.[106] Localized developmental pressure exerted upon the developing tooth

bud due to trauma has also been suggested.[97,110] It, therefore, appears that the etiology may be multifactorial, combining both genetic and environmental factors and causing aberrant hyperactivity of the dental lamina.[113,114]

Prevalence

Dens evaginatus has been reported to occur predominantly in people with Mongoloid genetic traits such as Chinese, Thai, Malaysians, Japanese, Indians, Eskimos, and Filipinos. The prevalence ranges from 0.5% to 4.3 %.[96,97] Isolated cases in African-Americans and Caucasians have also been reported.[115,116]

Dens evaginatus frequently occurs bilaterally without sex predilection.[96,107] Premolars are the most frequently affected posterior teeth, and mandibular premolars tend to have higher prevalence than maxillary premolars.[100]

The reported prevalence of talon cusps varies from 0.06% to 7.7%. The large variation is probably due to the lack of uniform classification criteria.[104,117,118]

Talon cusps are mainly found in the permanent dentition with twice, or higher, male predilection than female.[104,113] They are most frequently found in maxillary lateral incisors, followed by central incisors and canines that are the least affected teeth.[98,119] Although talon cusp has not been linked to any specific syndrome, it has been reported in patients with Rubinstein-Taybi syndrome, Mohr syndrome, Struge-Weber syndrome, incontinentia pigmentia achromians, Ellis-van Creveld syndrome, hypomelanosis of Ito, and Alagille's syndrome.[104] It has also been associated with peg-shaped lateral incisors, unerupted permanent canines, and impacted mesiodens.[110]

Clinical Implications

Because the tubercle of dens evaginatus extends above the occlusal surface, traumatic occlusal interference with opposing teeth may occur following tooth eruption.[97] Traumatic occlusion causes abnormal wear or fracture of the tubercle leading to pulpal inflammation and necrosis (Figure 26-8), sometimes

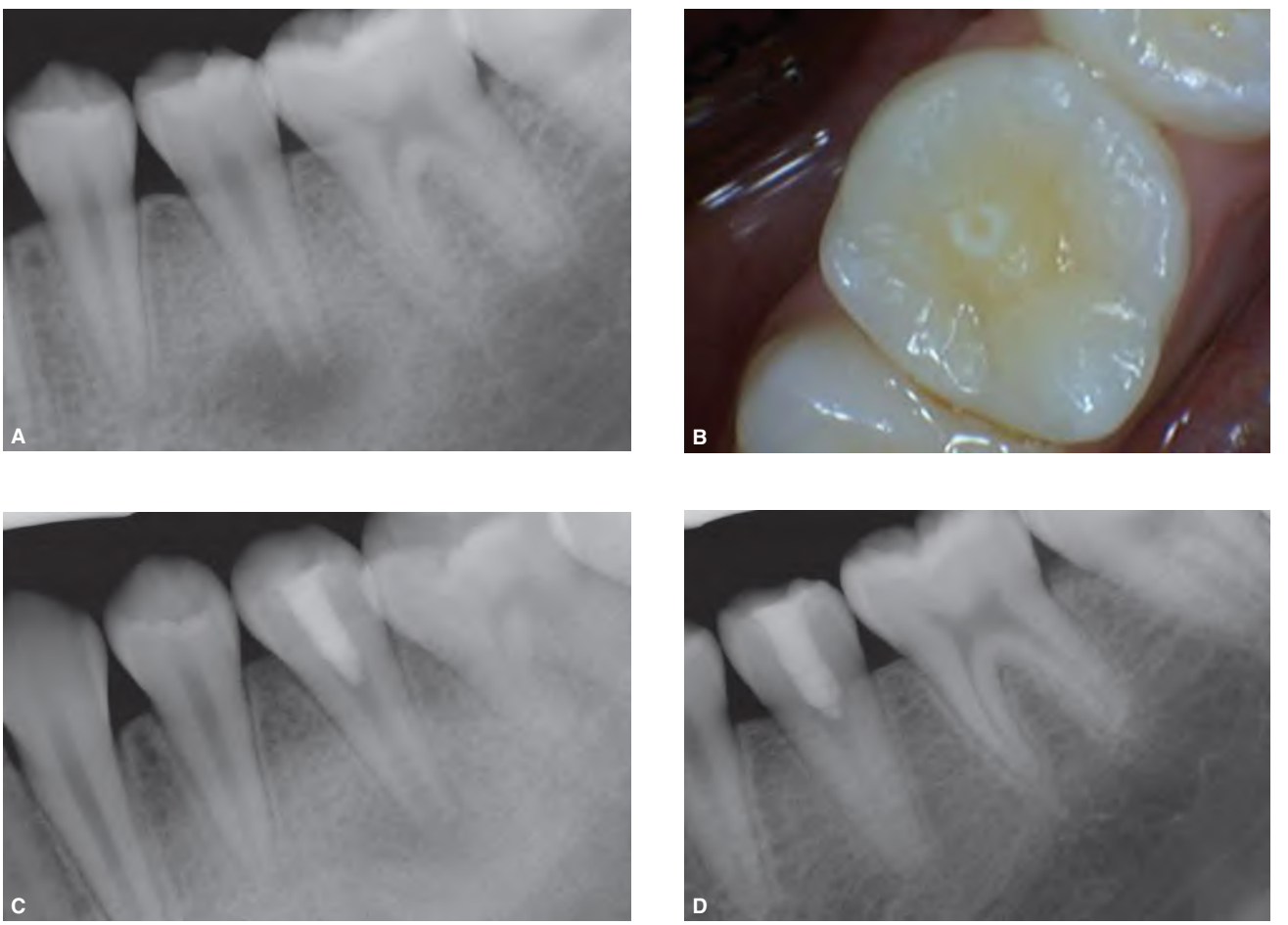

FIGURE 26-8 Dens evaginatus in a mandibular second premolar of a 12-year-old male. Patient complained of pain while biting on his left side. Pulp sensibility tests were negative. ***A.*** Preoperative radiograph showing periapical radiolucency associated with the tooth. ***B.*** Clinical view showing fractured occlusal protuberance (arrow). This frequent occurrence causes pulpal irritation, and often pulp necrosis at an early age. Pulp regeneration therapy with a triple antibiotic paste was performed (for more details about this procedure, see Chapter 29, "Regenerative endodontics"). ***C.*** Immediate postoperative radiograph. ***D.*** 18-month recall radiograph showing evidence of active bony healing. Note the narrowing of the pulp space. The patient was asymptomatic and the pulp responded positive to sensibility tests. (Courtesy of Dr. Rajiv G. Patel, Flower Mound, TX.)

before the completion of root formation.[96] In these cases, caries is usually not the prime cause of pulpal damage.[109]

It is imperative to recognize this anomaly early during clinical and radiographic examination, even before eruption, to take appropriate preventive measures.[96,97,106] Several methods have been advocated to prevent pulpal damage. Levitan and Himel[97] proposed that occluding surfaces of opposing teeth be reduced to eliminate traumatic occlusion with the tubercle. This should be followed by an application of topical fluoride to increase the enamel's hydroxyapatite resistance to acid breakdown. Subsequently, incremental layering of an acid-etched flowable light-cured resin should be applied to the tubercle and surrounding surface to support the base of the tubercle and to prevent the fracture.[97] The occlusion should be examined at six-month intervals in teeth with mature apices (3–4 months in immature teeth) and any necessary adjustments performed. Once radiographs have confirmed adequate pulp recession, the tubercle should be reduced to the level of the normal occlusal plane. Any exposed dentin should be protected with acid-etched microhybrid light-cured resin.[97]

Patients showing heavy occlusal forces on the tubercle may have the projection excised aseptically under dental dam isolation. This is followed by indirect or direct pulp capping (or parital pulptomy) and placement of a restorative material.[120,121] The superficial layers of the pulp are gently removed to a depth of 2 mm below the exposure where pulp capping material is placed onto the remaining pulp tissue to promote calcific bridge formation and continued root development.[97]

Past treatment modality suggested "spot grinding" or "selective grinding" of the tubercles to stimulate reparative dentin formation.[99,122] However, this method may not be always reliable since grinding of the tip of the tubercle would only stimulate a minor deposition of reparative dentin, running an additional risk of exposing the pulp under the tubercle.[100] It may only be successful in cases with no pulpal extension into the tubercle.[96]

Treatment becomes more complex when the pulp develops necrosis in teeth with immature roots. Some authors recommended extraction.[109] Conventional root canal therapy can be successfully performed after obtaining root-end barrier.[123–126] However, the affected tooth may remain with thin and fragile dentinal walls that are susceptible to fracture. Additionally, in young permanent teeth, the amount of root length remaining, general alignment of the tooth, and the overall arch length adequacy should be assessed prior to the treatment decision.[127] Regenerative endodontic techniques may be applied to allow continued root development.[128]

TAURODONTISM

Definition

The term ***taurodontism*** derives from Latin and Greek ("*tauro*" = "bull" and "*odus*" = "tooth"). It was first introduced more than a century ago to describe molar teeth resembling those of cud chewing animals, particularly bulls.[129]

Characteristics

Taurodontism is an anatomical variation in tooth morphology characterized by a vertically enlarged pulp chamber.[1,2] The pulpal chamber is displaced apically and the roots are short, presenting bifurcations, or trifurcations (Figures 26-9 and 26-10). Anatomically, this malformation shows a normal crown, but it lacks constriction at the cemento-enamel junction (CEJ) level, creating the appearance of a rectangular shaped root.

Etiology

Taurodontism is caused by failure of the Hertwigs' epithelium root sheath to invaginate at the proper horizontal level. Usually, taurodontisms can be observed in healthy individuals without any signs of systemic disorders.[130] However, it has also been associated with autosomal dominant conditions such as Apert syndrome, Axenfled-Reiger syndrome, basal cell nevus, ectodermal dysplasia, otodental dysplasia, tricho-dento-osseous syndrome, as well as chromosomal linked conditions such as Klinefleter syndrome and Down syndrome.[131] Therefore, identifying patients with taurodontism may sometimes aid in early recognition of certain systemic disorders.

Taurodontism can be classified into 3 types depending on the severity of the condition: *hypo-*, *meso-* and *hyper-*taurodontism. Hypotaurontsim is the least pronounced form, mesotaurodontism is moderate, and hypertaurodontism is the most pronounced. In cases of hypertaurodontism, the pulp chamber is unusually large, nearly reaching the root apex.[130,132]

Prevalence

The reported prevalence of taurodontism ranges from 0.1%[61] to 48%.[133] This wide range may be due to the different subjective diagnostic criteria used. Biometeric methods such as taurodontic index,[134] or other indices,[135,136] can help to objectively diagnose this anomaly.

Taurodontism can be found in both deciduous and permanent teeth, unilaterally, or bilaterally, and in any combination of teeth or quadrants.[137–139] A higher prevalence was observed in molars; prevalence increasing from the first to the third molar.[137] Similar prevalence was found in both genders.[140] One study, however, suggested a higher prevalence in Chinese females.[141]

Clinical Implications

Taurodontism may present a wide variety of pulp chamber size, shape, configuration, and position of canal orifices. Identifying the canal orifices, negotiating, instrumenting, and obturating the large root canal system may present some challenges. Because of the unusual anatomical features of the pulp chamber and root canal system, a careful exploration of all grooves between canal orifices is recommended. CBCT can be helpful for diagnosis and treatment since each of the taurodont root canal can be visualized individually.[142]

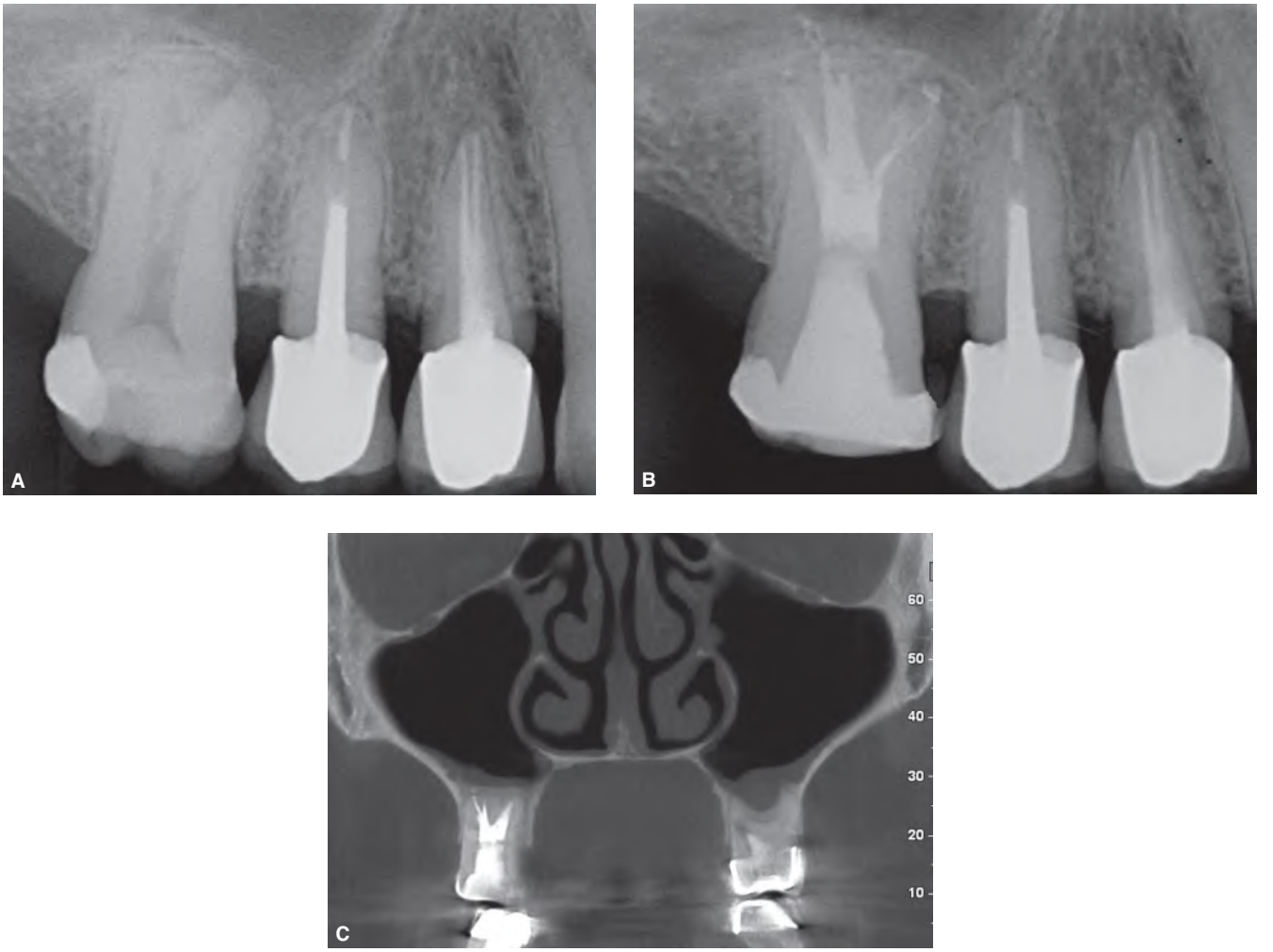

FIGURE 26-9 Taurodontisms (hyper-type) in a maxillary first molar of a 55-year-old male. Patient complained of pain and chronic sinusitis. ***A.*** Preoperative radiograph. ***B.*** Immediate postoperative radiograph following nonsurgical endodontic treatment. ***C.*** CBCT (Newtom) image showing the complex anatomy. (Courtesy of Dr. Jean-Yves Cochet, Paris, France.)

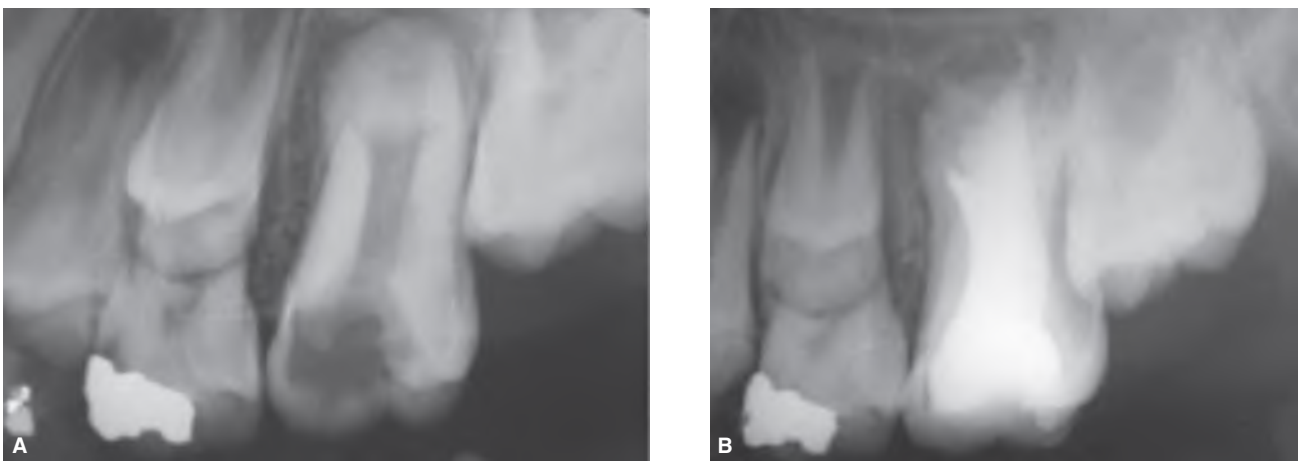

FIGURE 26-10 Taurodontism (hyper- type) in a symptomatic maxillary first molar of a 10-year-old male. A buccal apical swelling was detected. ***A.*** Preoperative radiograph. ***B.*** Postoperative radiograph following obturation with thermoplasticized gutta-percha and sealer. The patient is asymptomatic. (Courtesy of Dr. Lourdes Lanzagorta Rebollo, Instituto de Endo-Meta-Endodoncia, México.)

Due to the voluminous pulp tissue in the pulp chamber, in some cases, excessive bleeding can be observed during access preparation. This should not be confused with root perforation. Cervical dentin should be removed carefully, particularly in cases of hypertaurodontism, since the cervical tooth structure is thinner.[143]

Because the roots of the taurodont tooth are short, usually only the apical active part of the file is used, making the canal instrumentation time consuming.[144] Irrigation of the root canal system with sodium hypochlorite activated by ultrasonic energy may be beneficial to ensure complete removal of the pulp tissue.

Endodontic treatment in teeth with taurodontism can be very successful.[130,139,145] When performing endodontic treatment on a taurodont tooth, the operator should be aware of the complex anatomy of the root canal system. Judicious practice of endodontic principles such as identifying the canal orifices under magnification, thorough canal instrumentation and irrigation, and careful obturation of the entire root canal system is paramount for endodontic treatment success.

It has been suggested that only a small surface area of a taurodont tooth is embedded in the alveolus, thus compromising the stability of the tooth. Therefore, its use as an abutment for prosthetics or orthodontic anchorage purposes may be less desirable.[146] Post placement in a taurodont tooth is not recommended due to the large and elongated pulp chamber and short roots.[148]

From periodontal and surgical standpoints, the apically located furcation area may present a more favorable prognosis to treatment since attachment loss, resulting from a periodontal pocket or gingival recession, would involve less of the furcation area. Additionally, if the roots are not widely divergent, extraction of the tooth is easier. This can be beneficial in case intentional replantation procedure is indicated.

C-SHAPED CANAL

Definition

This anatomical variation presents a continuous slit connecting some or all canals, and forming a shape of the letter "C" in a cross-sectional configuration (Figure 26-11).

Characteristics

C-shaped canals are typically found in teeth with fused roots presenting with longitudinal grooves on the root surface. The groove can be located either on the buccal or lingual aspect of the root, or on both sides.[149] The floor of the pulp chamber is often deeply situated, assuming an unusual anatomical appearance.[150] The canal orifice may appear as a single, continuous ribbon-liked shape and often does not present the same configuration throughout the entire root length. It may present with a 180° or more arc that links the main canals, instead of having several discrete orifices. The arc may start at either the mesio-lingual or mesio-buccal line angle and sweeps around the tooth buccally, or lingually, to end at the distal aspect of the pulp chamber.[151,152]

Significant variations of root canal configurations at different levels of the root are found below the pulp chamber orifices without a consistent pattern.[153,154] The main anatomic feature of the root canal system is the presence of fins or webs that connect the individual canals.

Melton et al.[155] proposed a classification based on cross-sectional shape. *Category I* represents a continuous C-shaped orifice configuration without any separation of the outline; *Category II* represents a canal orifice shape resembling a semicolon (;) where dentin separates the main C-shaped outline from the same buccal or lingual direction, making a discrete canal to discontinue the "C" outline; and *Category III* represents two or more discrete and separate canals. Fan et al.[150] proposed a modified classification by adding two more categories. *Category IV* represents a single, round or oval, canal in the cross-section; and *Category V* where no canal lumen is observed near the root apex.

Etiology

It is hypothesized that failure of the Hertwig's epithelial root sheath to fuse on the lingual or buccal root surface is the main cause for this variation.[156–158] Fused roots may form by coalescence of cementum deposition over time.

Prevalence

C-shaped canals are not rare. The reported prevalence ranges from 2.7% to 44.5%.[152,159] Ethnic variation is considered an important factor. This particular tooth variation is more commonly seen in Asian ethnic groups.[156] Chinese populations[160,161] have shown higher prevalence of C-shaped mandibular second molars than Caucasians.[151]

Generally, C-shaped canal is most frequently found in mandibular second molars (Figures 26-11 and 26-12). In such cases, it may also be found in the contralateral tooth in over 70% of cases.[162] Mandibular second premolars are the second most frequent site, with a reported incidence rate varying from 14% to 18%.[163,164]

C-shaped canals, however, may also occur in other teeth such as mandibular first molars,[165,166] maxillary first molars,[167–169] maxillary[170] and mandibular[171] third molars, and even in maxillary lateral incisor.[172]

Clinical Implications

C-shaped canals should be carefully diagnosed and treated. Radiographically, an endodontic instrument placed in the canal may appear to perforate the furcation. Radiographs taken from several horizontal angulations[157] or CBCT images[173] will prove beneficial.

There are several radiographic hints that can help the operator diagnose C-shaped canals prior to accessing the pulp chamber. Simon[174] suggested the following:

1. The root appears conical and as if representing one root with a furcation. This is usually in contrast to adjacent molars (especially the first molar), that have two distinct roots with a normal furca.

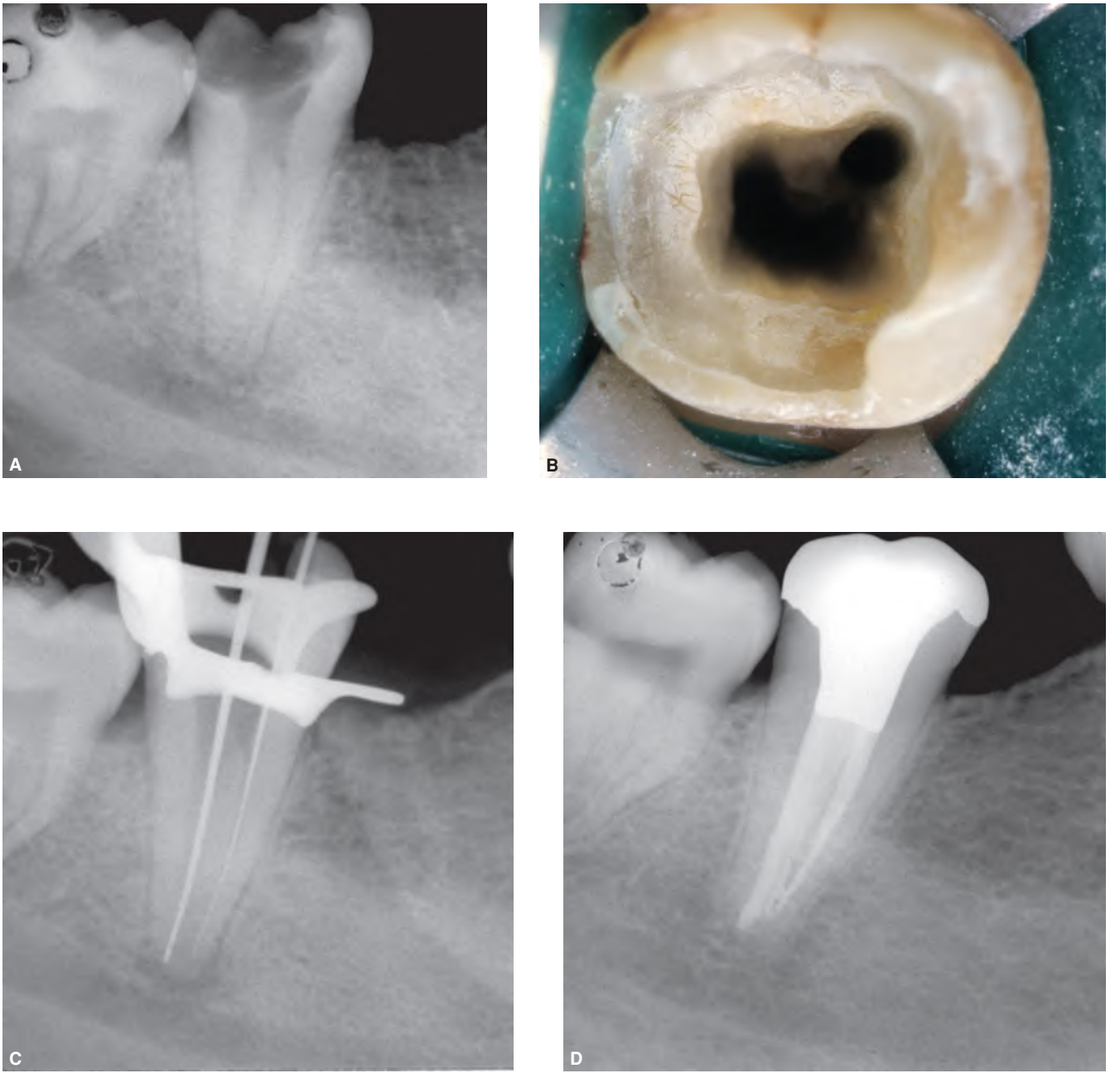

FIGURE 26-11 Endodontic treatment in a mandibular second molar with C-shaped canal and necrotic pulp of a 25-year-old male. ***A.*** Preoperative radiograph showing the affected tooth associated with a periapical radiolucency. ***B.*** Clinical view of the access cavity showing an independent mesiolingual canal and a C-shaped configuration connecting the mesiobuccal canal to the distal canal. ***C.*** Working length assessment radiograph. ***D.*** Three-year recall radiograph showing resolution of the periapical radiolucency. The patient is asymptomatic and the tooth is functional. (Courtesy of Dr. Domenico Ricucci, Cetraro, Italy. Adapted with permission from Ricucci et al.[147])

2. The pulp chamber appears longer than that of the adjacent molars.
3. The pulp chamber becomes indistinct apically. The pulp chamber's floor is not usually visible and little or no furca is apparent on the radiograph.
4. At the apex, the periodontal ligament is indistinct or indiscernible. Canals cannot be followed in the apical third portion of the tooth. The exit of the apical foramina are not clearly observed.

Thorough debridement of the C-shaped canal can be exhausting due to the multiple connecting fins and isthmi.[149,155,175] Since canal bifurcations may be observed at any level between the orifice and apical terminus, careful exploration with a small, pre-curved endodontic file using the operating microscope is helpful.[153]

Supplemental canal cleaning with ultrasonics may be beneficial.[176–178] Canal preparation by mechanical instrumentation should follow the same configuration as

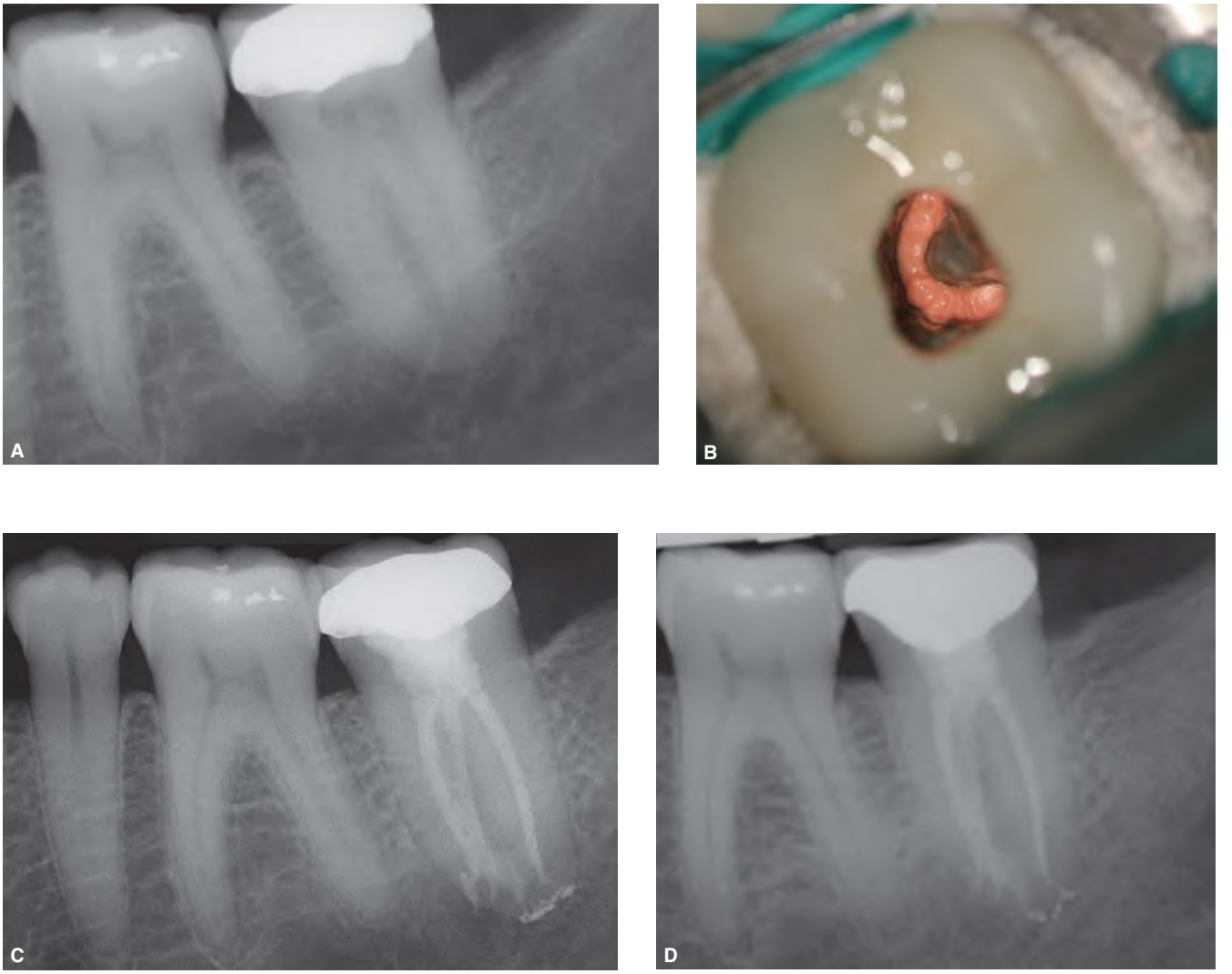

FIGURE 26-12 C-shaped canal in a mandibular second molar of a 40-year-old female. Pulp sensibility tests were negative. ***A.*** Preoperative radiograph. ***B.*** Clinical view of the access cavity after obturation of the root canal system. ***C.*** Immediate postoperative radiograph. Note the "shift" of the mesial canal toward the furcation area. This is a typical phenomenon. ***D.*** One-year recall radiograph. The patient is asymptomatic and the tooth is functional. (Courtesy of Dr. Rajiv G. Patel, Flower Mound, TX.)

the canal because the deep groove and the thin connecting isthmi are prone to perforation.[159] A simple enlargement of the canals can lead to perforation, stripping, or both[174]

Obturation may require technique modification.[179] Thermoplasticized gutta percha obturation technique is preferred over cold lateral condensation since softened gutta-percha and sealer will adapt more effectively to the aberrant canal walls.[180]

Restoration of the C-shaped root will require special attention when placement of post or core are needed. Thin dentin is usually present between the canal walls and the outer surface of tooth. Both the buccal and lingual canal walls are frequently narrower at the mesial aspect.[181]

When sound biological treatment principles are followed, a tooth with C-shaped canals should have equal prognostic outcome as other teeth.[174]

SUPERNUMERARY ROOTS AND CANALS

Definitions

Extra roots or root canals are those that show up more than the average detected in the population. Their presence in the human dentition varies considerably.[182] Often, the name of the supernumerary root or root canal derives from its location as related to the main root/canal (e.g., middle-mesial, mesiobucal/MB-2). However, sometimes

they receive specific names (e.g., *radix entomolaris*, *radix paramolaris*, *radix distomolaris*).[183,184] In maxillary molars, *radix distomolaris* was proposed to describe a supernumerary root located distally and *radix distolingualis* or *mesiolingualis* when it is located distolingually or mesiolingually respectively.[185,186] In maxillary premolars, the term *ridiculous* has been proposed because of their resemblance to the adjacent maxillary molar.[187,188]

Characteristics

The characteristics of supernumerary roots and root canals vary.[189] External contour of the supernumerary root furcation is more complex than the one with average root anatomy.[190–192] An extra cusp (*tuberculum paramolare*), or more prominent occlusal distal or distalolingual lobe, in combination with a cervical prominence or convexity, may indicate the presence of an additional root. Radiographically, if the mesiodistal width of the root appears equal to or greater than the crown, variation in root number is likely to exist.[193–195]

An increased number of cusps is not necessarily related to a greater number of roots/canals.[197] However, an additional root is almost always associated with a greater number of cusps and root canals.[192]

Etiology

The exact etiology is not completely understood. In cases of dysmorphic supernumerary root formation, it could be related to external factors occurring during odontogenesis, or to atavism (reappearance of a trait after several generation of absence). In eumorphic roots, racial genetic factors, influencing the more profound expression of a particular gene, result in the more pronounced phenotypic manifestation.[192,198]

Prevalence

Supernumerary roots and root canals can affect any tooth (Figure 26-13) but are more prevalent in molars (Figure 26-14) and premolars (Figure 26-15). The presence of a separate root in the mandibular first molar is associated with certain ethnic groups.[199–205] While European and African populations present a prevalence lower than 5%,[197,204] populations with Mongoloid traits may reach more than 40%.[199] Because of the high prevalence in these populations such tooth anomaly is considered a normal morphological variant of mandibular first molars.[192] It also tends to appear bilaterally. The reported bilateral occurrence ranged between 50% and 69%.[192,204]

Radix root (Figure 26-16) may be found in all mandibular molars. However, it is most frequently found in the first molar and least frequent in the second molar.[202] Its occurrence is similar in males and females.[206–208] The entomolaris type occurs more frequently than the paramolaris type.[192]

The prevalence of four rooted maxillary molars can vary between 0.4% and 1.4%.[209,210] The occurrence of two canals in the distobuccal root of maxillary molars may rage between 1.9% and 9.5%,[211–213] and of three canals or more in the mesiobuccal root of maxillary molars between 1.3% and 2.4%.[214] Nevertheless, no specific predilections of supernumerary roots and root canals in maxillary molars have been established.

Middle mesial canals in mandibular molars have been reported to occur in up to 36% of cases,[215] 20% showing independent apical foramen.[216]

The reported prevalence of two roots in the mandibular first premolar was 1.8%; in three roots, 0.2%; and in four roots, fewer than 0.1% of cases.[182] Three root canals have been reported to range from 0.4% to 5%.[195,211] Maxillary premolars have been reported to present 3 roots in fewer than 2% of cases.[217]

Clinical Implications

An accurate diagnosis of supernumerary roots and root canals is important, to avoid complication or a "missed canal" during endodontic treatment.[218] Radix entomolaris is often located in the same buccolingual plane as the distobuccal root, resulting in superimposition of both roots on the preoperative 2-D radiograph.[192,219] A thorough inspection of the preoperative radiograph and interpretation of particular marks such as an unclear outline of root/canal contour, may indicate the presence of a supernumerary root. Additional radiographs, taken from several angles, may help the diagnosis. Multiple intraoral radiographs, however, do not guarantee the identification of all relevant anatomic variation of the tooth, prior to commencing root canal therapy.[204] CBCT can be advantageous for some of these cases.[219]

Using a periodontal probe may aid in identifying additional roots during clinical inspection of the crown and examination of the cervical morphology of the roots.[192,198] Also, the location of the orifice of the root canal may provide clues, whether additional canals are present or not. For example, the orifice of the radix entomolaris is often located distolingually to the main distal root.[204,218] The middle mesial canal in mandibular molars is frequently located closer to the mesio-buccal canal than to the mesio-lingual canal.[216,220] A modification of the access cavity and eliminating the dentinal protuberances may be necessary to better locate and access the additional canals.[192,213,218,221] Enhanced magnification, illumination, and ultrasonics are excellent adjuncts for a better identification and localization of the supernumerary root canal orifices.[219]

Supernumerary roots and root canals are treated routinely in an endodontic practice. A thorough knowledge of the complexity of root canal systems, operator's skill, and increased time per appointment can aid in identifying and managing these anatomical variations for endodontic purposes.

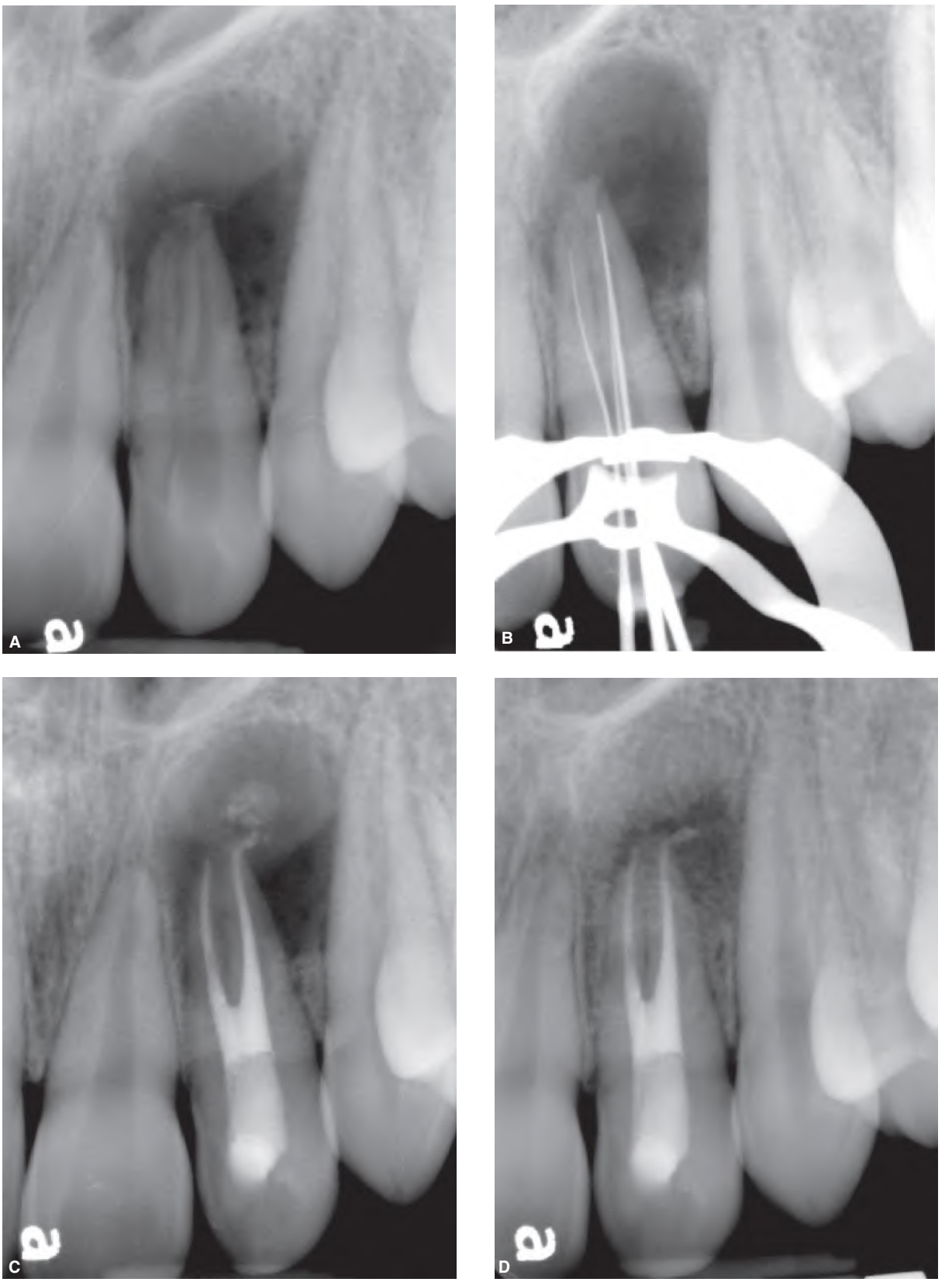

FIGURE 26-13 Supernumerary root in a maxillary lateral incisor of a 12-year-old female. The tooth was symptomatic and diagnosed with pulp necrosis. ***A.*** Preoperative radiograph showing presence of a large periapical radiolucency associated with the tooth. ***B.*** Working length assessment radiograph reveals presence of two roots associated with confirmed apical root resorption. ***C.*** Postoperative radiograph showing canals obturation with Mineral Trioxide Aggregate (MTA) and bonded composite core. Minor cement extrusion beyond the apical foramen is noted. ***D.*** Six-month recall radiograph showing a reduction in size of the periapical radiolucency. (Courtesy of Dr. George Bogen, Los Angeles, CA.)

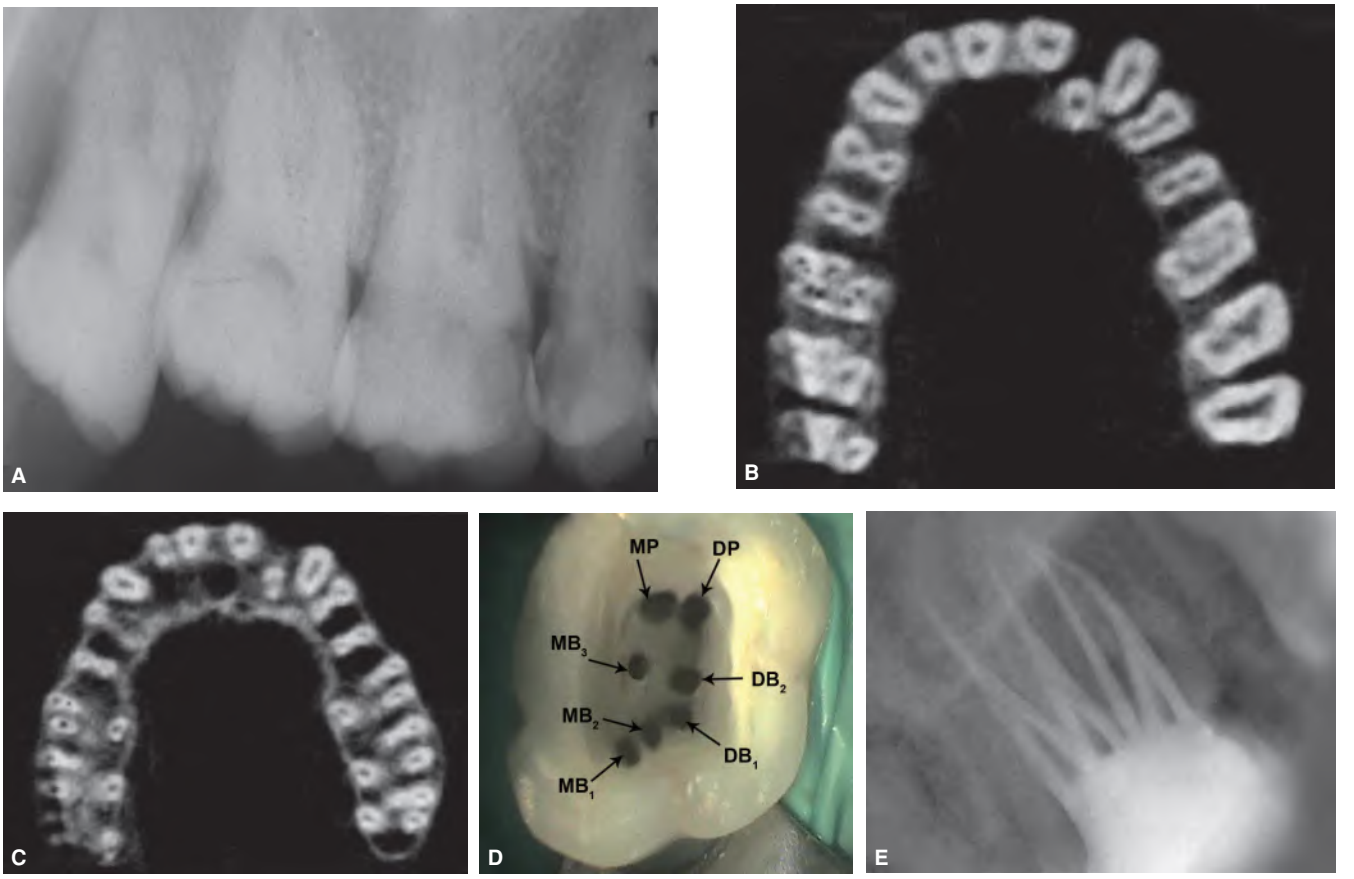

FIGURE 26-14 Supernumerary root canals in a maxillary right first molar of a 37-year-old male. The patient complained of pain from this tooth that intensified by thermal stimuli and mastication ***A.*** Preoperative radiograph. ***B.*** CBCT axial view at the cervical level of the root showing seven canal orifices. ***C.*** CBCT axial view at the apical level showing four distinct canals indicating that some canals have joined. ***D.*** Clinical view of the access opening showing seven root canal orifices. ***E.*** Postoperative radiograph. The canals were obturated with gutta-percha and AH Plus sealer. The tooth was then restored with a posterior composite resin core. (Courtesy of Dr. Jojo Kottoor, Tamil Nadu, India. Adapted with permission from Kottoor et al.[196])

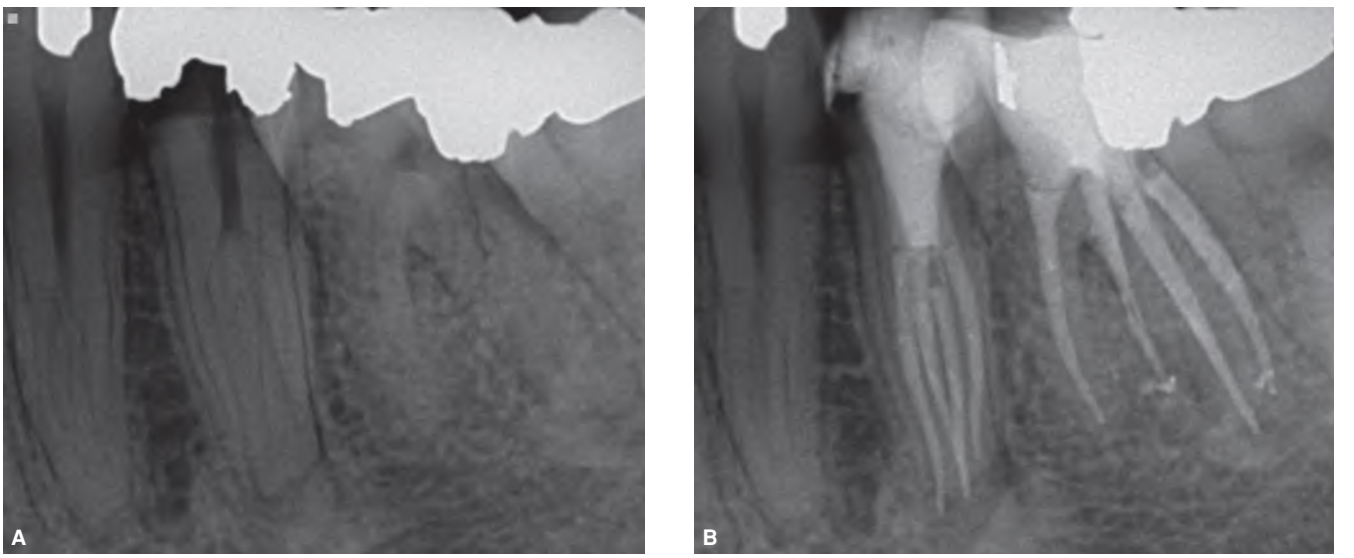

FIGURE 26-15 Supernumerary root canals in a mandibular second premolar of a 21-year-old female diagnosed with irreversible pulpitis and symptomatic apical periodontitis. ***A.*** Preoperative radiograph. ***B.*** Immediate postoperative radiograph showing 4 root canals. Canal orifice distribution was one buccal, one lingual, and two middle (one mesial and one distal). (Courtesy of Dr. Taoheed O. Johnson, Advanced Endodontics Program, University of Southern California, Los Angeles, CA.)

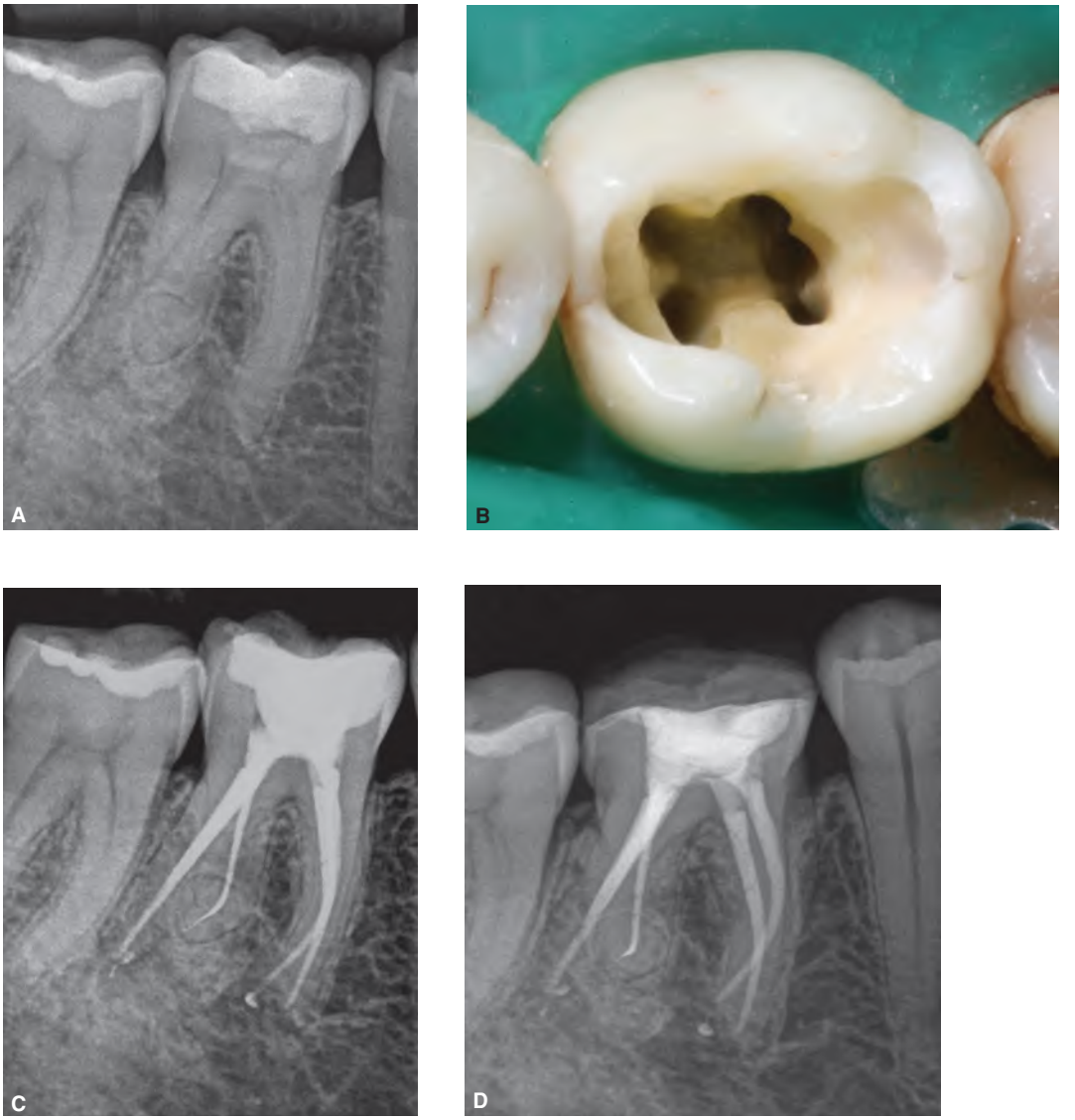

FIGURE 26-16 Radix entomolaris in a mandibular first molar of a 52-year-old female. Patient complained of spontaneous pain associated with the tooth. ***A.*** Preoperative radiograph showing presence of additional root. ***B.*** Clinical view of the access cavity showing two mesial canal orifices and three distal orifices. ***C.*** Immediate postoperative radiograph. ***D.*** One-year recall radiograph. The patient is asymptomatic and the tooth restored to function. (Courtesy of Dr. Francesc Abella, Barcelona, Spain. Adapted with permission from Abella, et al.[219])

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Chapter 27

Vital Pulp Therapy

George Bogen, Nicholas P. Chandler

Science is nothing but perception

—Plato

Vital pulp therapy is advanced treatment devised to conserve, protect, and maintain a healthy pulp. This includes pulp tissue that may have been compromised by anatomical anomalies, caries, trauma, or restorative procedures. The prime objective of the treatment strategy is to promote the formation of a reparative hard tissue barrier after pulpal injury, in order to retain the tooth as a functional unit. This is critically important in the immature adult tooth, where apical root development may be incomplete.

This chapter describes advances in direct pulp capping and pulpotomy, where treatment therapies have been redefined by the introduction of bioactive capping materials, and clinical protocols that more predictably encompass the microbiological aspects of failure. Attention will be directed toward the preservation of the pulpally involved **permanent tooth**, based on the contention that pulp tissue has an inherent active repair capacity, when microorganisms and their by-products are effectively excluded.[1,2]

Over the last two decades, remarkable progress has been made in the field of vital pulp therapy, primarily due to the introduction of bioactive materials.[3] Since Pierre Fauchard, in the 18th century, first introduced the concept of indirect pulp capping, clinicians have recognized the innate healing capacity of dental pulp tissue, for cell reorganization and calcific bridge formation.[4] Phillip Pfaff is credited with placing gold foil against an exposed pulp in order to promote pulpal healing; this could be the first documented direct pulp capping procedure.[5] Ever since the early times of modern dentistry, researchers and clinicians have endeavored to expand their knowledge of pulp physiology, microbiology, and caries progression, in order to improve pulp preservation methods. Progress has coincided with the quest to identify physiological mediators and bioinductive materials that reliably promote reparative bridge formation and protect the pulp against further injury from microbial ingress.

There has been a long-standing view that direct pulp exposures, in a carious field, have an unfavorable prognosis. Unfortunately, more aggressive procedures, such as pulpotomy or pulpectomy, are still being recommended.[6–8] These treatment strategies were based on traditional protocols and capping materials, that did not provide a uniform environment for pulp cell recruitment, differentiation, and hard tissue repair. Furthermore, prior to treatment, the histological condition of the pulp can be difficult to ascertain, based on the limitations of pulp testing methods, a limited knowledge of pulpal physiology, the subjectivity of the patient's symptoms, and the presence of inflammatory mediators that cannot be assessed clinically.[9] Careful and prudent case selection, based on our current understanding of inflammatory mechanisms that are responsible for producing irreversible changes in pulpal tissue, can successfully identify teeth with a higher likelihood for favorable outcomes.

The major challenge in vital pulp therapy has been to identify a reliable pulp capping/pulpotomy agent and a simplified/reliable delivery technique. The introduction of Mineral Trioxide Aggregate (MTA) and other calcium silicate-based cements (CSCs) has provided a new paradigm for treating the reversibly inflamed pulp. This changes the long-held, yet venerable, perception that pulp capping the cariously exposed tooth is unpredictable and, therefore, contraindicated. Additionally, the impact and success of vital pulp therapy will depend largely on the patient's age, size of the pulp, degree of microbial contamination, pulp capping material, and the seal of the final restoration. Moreover, proper case selection based on an accurate differential diagnosis, using multiple diagnostic tests to establish pulpal status, is essential for a predictable outcome. According to the American Academy of Pediatric Dentistry,[10] "Teeth exhibiting provoked pain of short duration, that is relieved, upon the removal of the stimulus, with analgesics, or by brushing, without signs and symptoms of irreversible pulpitis, have a clinical diagnosis of reversible pulpitis and are candidates for vital pulp therapy."

The assessment of a definitive pulpal status, prior to treatment, can be challenging in younger patients. However, a diagnosis of reversible pulpitis increases the probability of favorable healing and repair. A negative patient report, that is variable and subjective, does not always indicate that the pulp capping/pulpotomy procedure cannot proceed successfully. Moreover, pain or discomfort prompted by cold testing prior to treatment, or a direct pulp exposure during caries excavation, does not necessarily condemn the tooth to a poor prognosis and more aggressive therapies.

THE VITAL DENTAL PULP

The dental pulp is a highly vascular tissue that has the unique distinction of being encased within a rigid chamber composed of dentin, cementum, and enamel.[11] These hard tissues provide mechanical support and protection from oral microorganisms.[12] The tissue provides several important functions,

including immune cell defense, dentinogenesis, calcified barrier formation, nutrition, and proprioreceptor cognizance. The survival and maintenance of the dental pulp is crucial to the long-term retention and function of the tooth, since the healthy pulp produces secondary, tertiary, and peritubular dentin as a protective response to various biological and pathological stimuli.[13]

Composed of four distinct zones, the dental pulp exhibits a cell-rich zone, a core composed of major vessels and nerves, a cell-free zone, and the odontoblastic layer at the periphery. The major cell populations found in the pulp include undifferentiated mesenchymal stem cells, fibroblasts, odontoblasts, macrophages, and other immunocompetent cells. The odontoblasts have the distinction of forming a single layer lining the entire perimeter of the pulp and feature odontoblastic processes extending into the dentin, sometimes to the dentoenamel junction.[14–16] For more details, see Chapter 2, " Structure and function of the pulp–dentin complex."

If the hard casing of the tooth is compromised and the pulp is subjected to microbial invasion, inflammatory changes can lead to pulp necrosis and further pathological changes, including infection and its consequences.[17,18] Circulating immune competent cells limit microbial challenges, and functioning proprioceptors and pressoreceptors guard against excessive occlusal loading.

When cariogenic microorganisms challenge the healthy pulp, odontoblasts, in concert with circulating immune cells, exhibit both innate and adaptive immune responses.[19] Innate responses begin within the dentinal tubule at the carious front, where acidogenic Gram-positive bacteria are initially exposed to a positive outward flow of dentinal fluid containing immunoglobulins and serum proteins, that slow the diffusion of bacterial antigens. The odontoblasts also exhibit Toll-like receptors that stimulate pro-inflammatory cytokines such as interleukin 1,8,12, tumor necrosis factor alpha, transforming growth factor beta, gamma-interferon, and the complement system.

If the bacterial front advances, vasoactive neuropeptides are released that increase vascular permeability and intrapulpal blood flow.[20] Innate immune cells that participate in this progression include lymphocytes, dendritic cells, monocytes, and macrophages. Increases in neuropeptide concentration and nerve sprouting characterize neurogenic inflammation that can increase transient and interstitial tissue pressure contributing to painful pulpitis. Persistent infection engages the adaptive immune system characterized by increased edema, intrapulpal pressure, tissue destruction, and cell death, leading eventually to pulpal necrosis. Odontoblasts also participate in adaptive responses by synthesizing a mineralized matrix known as reactionary dentin, a hard tissue differentiated by reduced tubularity. Because of the specific location of microorganisms during caries progression, bacterial neutralization by phagocytosis (killing) is not activated until the caries front is in direct contact with the dental pulp (Figure 27-1).[21] It has been proposed that bacterial colonization (a focus of necrosis) represents the histological transition from reversible to irreversible pulpitis.

When the dental pulp is injured by carious exposure, or trauma, and competently treated, the mechanism of healing is similar to that observed in normal connective tissue. Hemostasis, inflammation, proliferation, and remodeling characterize the continuous healing process sequenced over four overlapping phases.[22] Tissue near the exposure is characterized by extravasated erythrocytes, inflammatory cells, and potentially necrotic tissue, directly after injury. Neutrophils dominate the acute pulpal response in the presence of blood coagulation and exudated fibrinogen. Proinflammatory cytokines, released by immunocompetent cells, alter vascular permeability in response to both trauma and bacterial by-products. Transmigration of inflammatory cells is enabled by chemotactic signaling that prompt adhesion molecule interactions between leucocytes and endothelium. Chemoattractant/activator molecules, interacting with sets of cell adhesion molecules, form an adhesion cascade through these cell interactions.[23] Since the vital pulp is, therefore, capable of demonstrating competent immune defense mechanisms, it is desirable to preserve the vitality of an exposed pulp and promote repair since its retention (longevity) is paramount to the tooth's long-term survival.

REPARATIVE BRIDGE FORMATION

The formation of a protective hard tissue barrier is the goal of vital pulp procedures. After the loss of the primary odontoblasts, due to direct pulp exposures, pulpo-dentinal defects are repaired by the development of a hard tissue barrier by cells recruited from the pulp. These odontoblast-like cells are, theoretically, derived from either perivascular cells, bone marrow stem cells, or dental pulp stem cells.[24,25] The resultant hard tissue generation has long been regarded as tertiary reparative dentin, or described as reparative "dentinogenesis" after the death of the original odontoblasts for lack of a better term. However, histological examinations of reparative bridge formation, in directly capped human teeth using calcium hydroxide (CH), have revealed that the initial cellular response proximal to CH generates pulpal necrosis and stimulates the formation of a dentin-like hard tissue bridge forged by cells that morphologically resemble fibroblasts.

A recent light microscopy study examined ninety-six extracted human teeth, characterized by either untreated deep caries, large restorations or treated by direct pulp capping procedures after carious exposures.[26] Observational data revealed that the reparative hard tissue formed in teeth with deep caries and large restorations without pulp exposures, exhibited tubules continuous with the tubules of the secondary dentin, consistent with tertiary (reactionary) dentin generation. This tertiary dentin was associated with a reduced

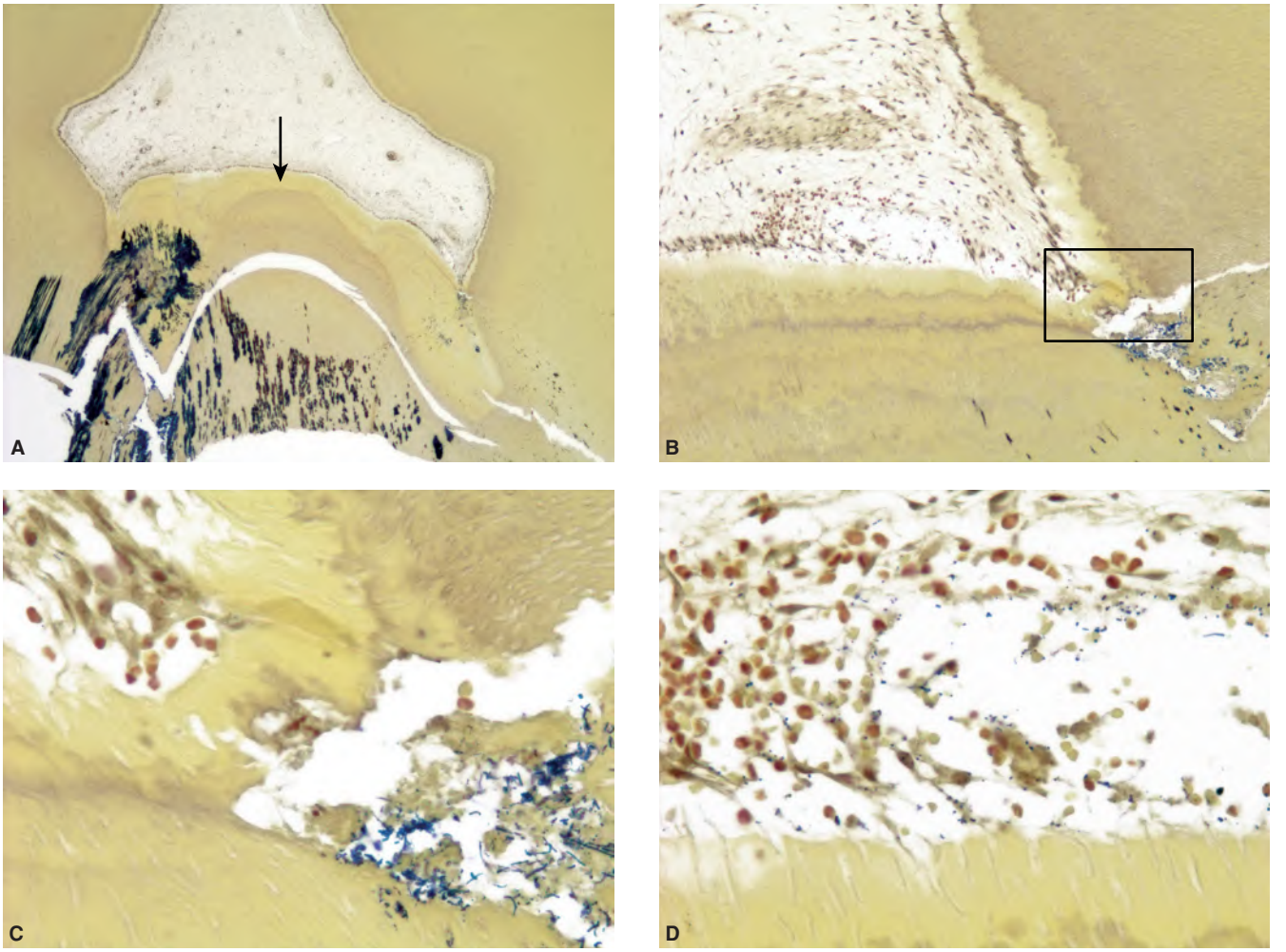

FIGURE 27-1 ***A.*** Photomicrograph of a pulp chamber in a symptomatic human maxillary third molar exhibiting a deep occlusal carious lesion and bacterial invasion with a clinical diagnosis of irreversible pulpitis (longitudinal section). Note presence of abundant reactionary (tertiary) dentin on the pulp chamber roof (arrow) and the relatively normal appearance of the pulp tissue, except in the area of the mesial pulp horn (right), where a small empty space can be seen. The empty spaces in the carious dentin are artifacts, due to detachment of soft dentin during processing (Original magnification ×16). ***B.*** Detail from the right pulp horn in A (×100). ***C.*** High-power view of the carious tissue adjacent to the pulp horn demarcated by rectangle in B. Note presence of inflammatory cells associated with bacterial penetration (×400). ***D.*** Higher magnification of the empty space in the pulp horn. Scattered bacterial cells surrounded by inflammatory cells engaged in active phagocytosis (killing) (Taylor modified Brown & Brenn stain, ×400). (Courtesy of Dr. Domenico Ricucci, Cetraro, Italy.)

single palisade layer of flattened primary odontoblasts. To the contrary, teeth that had been directly pulp capped in the absence of the primary odontoblasts, histological examination showed the exposures were repaired by an amorphous and atubular calcified tissue, sometimes exhibiting remnants of embedded necrotic tissue and pulp capping material. These dystrophic calcific bridges were affiliated with collagen fibrils and the direct presence of pulpal fibroblasts (Figure 27-2).

During pulpal healing, electron microscope images show that initial calcification of the bridge is characterized by a profusion of extracellular matrix vesicles situated between the amputated pulp surface and the forming cells.[27] The vesicles contain needle-like crystals and an osmiophilic material that accumulate and form a calcified front after the disappearance of the vesicular membrane. Similar to calcification in other biologically normal or diseased tissues, the accumulation of calcium and phosphate ions within the crystals suggests their generation during the calcification process. Calcific bridge formation can be seen after one month at the site of the surgical wound. However, pulp healing defects in the hard tissue can include tunnel defects, pulpal inflammatory cell activity, operative debris, and bacterial microleakage, depending on the pulp capping agent.[28] Reparative bridge mineralization may actually be more dependent on the extracellular matrix than the pulp capping or pulpotomy agent applied.[29–31]

It is apparent that reactive tertiary dentinogenesis can only occur with the retention of the primary odontoblasts during

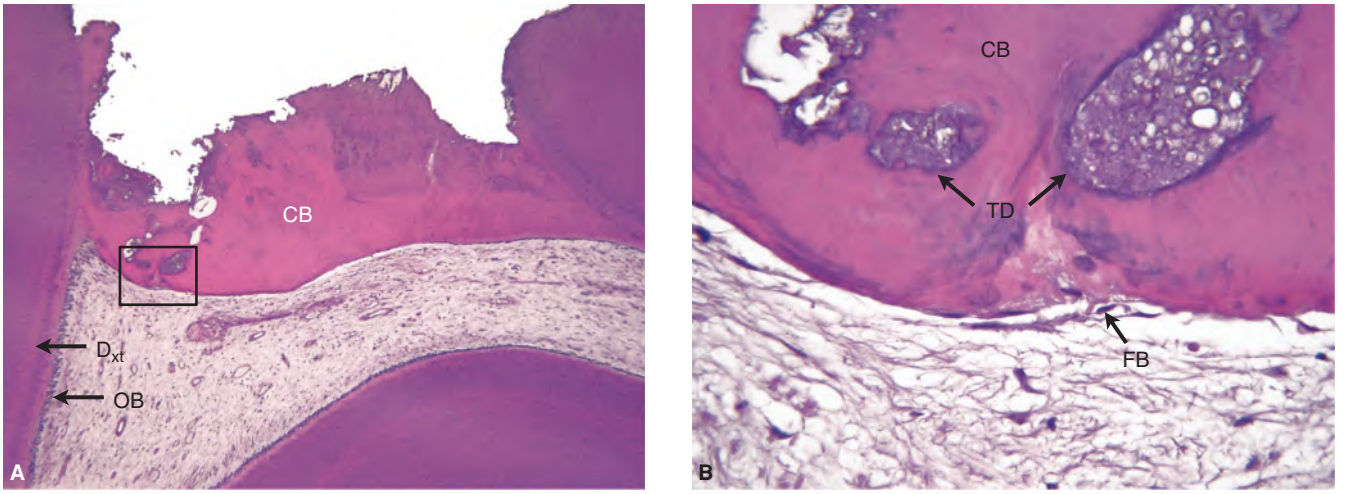

FIGURE 27-2 ***A.*** Reparative bridge formation in a human mandibular third molar directly pulp capped with calcium hydroxide and extracted after two years and four months. Photomicrograph shows calcified barrier (calcific bridge) formation (CB), dentin and predentin (D) associated with palisading odontoblasts (OB). Note absence of primary odontoblasts directly below the reparative bridge (pulp capping area) (Original magnification ×50). ***B.*** Higher magnification of area demarcated by rectangle in A. The calcified repair tissue (CB) is devoid of dentinal tubules and associated with tunnel defects (TD) containing necrotic debris. Note presence of collagen fibrillar material, scattered fibroblasts (FB) and no evidence of an odontoblast layer. (H & E, ×400). (Courtesy of Dr. Domenico Ricucci, Cetraro, Italy.)

caries progression and in indirect pulp capping procedures. Hard tissue barriers, formed after the loss of the primary odontoblasts, in direct pulp capping and pulpotomy procedures, are calcified hard tissues produced by cells that appear to mirror pulpal fibroblasts, and histologically the hard tissue produced in response to trauma is dentin-like rather than being genuine dentin. This new understanding of intrapulpal repair mechanisms does not change the common goals; to induce pulp healing, protection and survival. Further studies are required, to elucidate the bioactive molecules, growth factors and cellular mechanisms, responsible for regenerating hard tissue and physiological dentin.

TECHNIQUES FOR GENERATING REPARATIVE BRIDGES

Direct Pulp Capping

For permanent teeth, direct and indirect pulp capping and partial or complete pulpotomy are the most widely employed strategies in vital pulp therapy. ***Direct pulp capping*** is defined as the "treatment of an exposed vital pulp by sealing the pulpal wound with a dental material placed directly on a mechanical or traumatic exposure to facilitate the formation of "reparative dentin" and maintenance of the vital pulp."[32] Pulp exposures, as a result of caries removal, tooth preparation, or trauma are all indications for direct pulp capping. However, pulp tissue jeopardized by acute inflammation, with longstanding exposure to oral microorganisms, may be an inappropriate candidate for direct pulp capping. Direct pulp capping for carious exposures will be reviewed in detail later in this chapter under treatment recommendations (Figure 27-3).

Indirect Pulp Capping

Indirect pulp capping is defined as "a procedure in which a material is placed on a thin partition of remaining carious dentin that, if removed, might expose the pulp in immature permanent teeth."[32] Non-symptomatic teeth with no radiographic evidence of pathosis now show remarkable success using this technique, although it has been controversial for decades.[33,34]

Caries removal is completed using various techniques, to a perimeter near the tissue, without directly exposing the pulp. Selected teeth with deep carious lesions that receive conservative caries excavation, where direct pulp exposures were avoided, have demonstrated strong potential for repair since the primary odontoblasts are preserved.[35,36] One or two-stage (step-wise) indirect pulp capping techniques are completed using either CH, zinc oxide-eugenol materials (ZOE) or CSCs. A major challenge with the clinical procedure (one or two-stage) is determining at what point the excavation is terminated. Other important considerations include the voids under the restorative material that form during the remineralization process, as the carious dentin desiccates and volume diminishes. Other complications can be the rapid reactivation of a dormant lesion or restoration failure.[37] Improvements in indirect pulp capping techniques have been made and currently show remarkable potential as a treatment strategy for immature permanent teeth (Figure 27-4).[38–40]

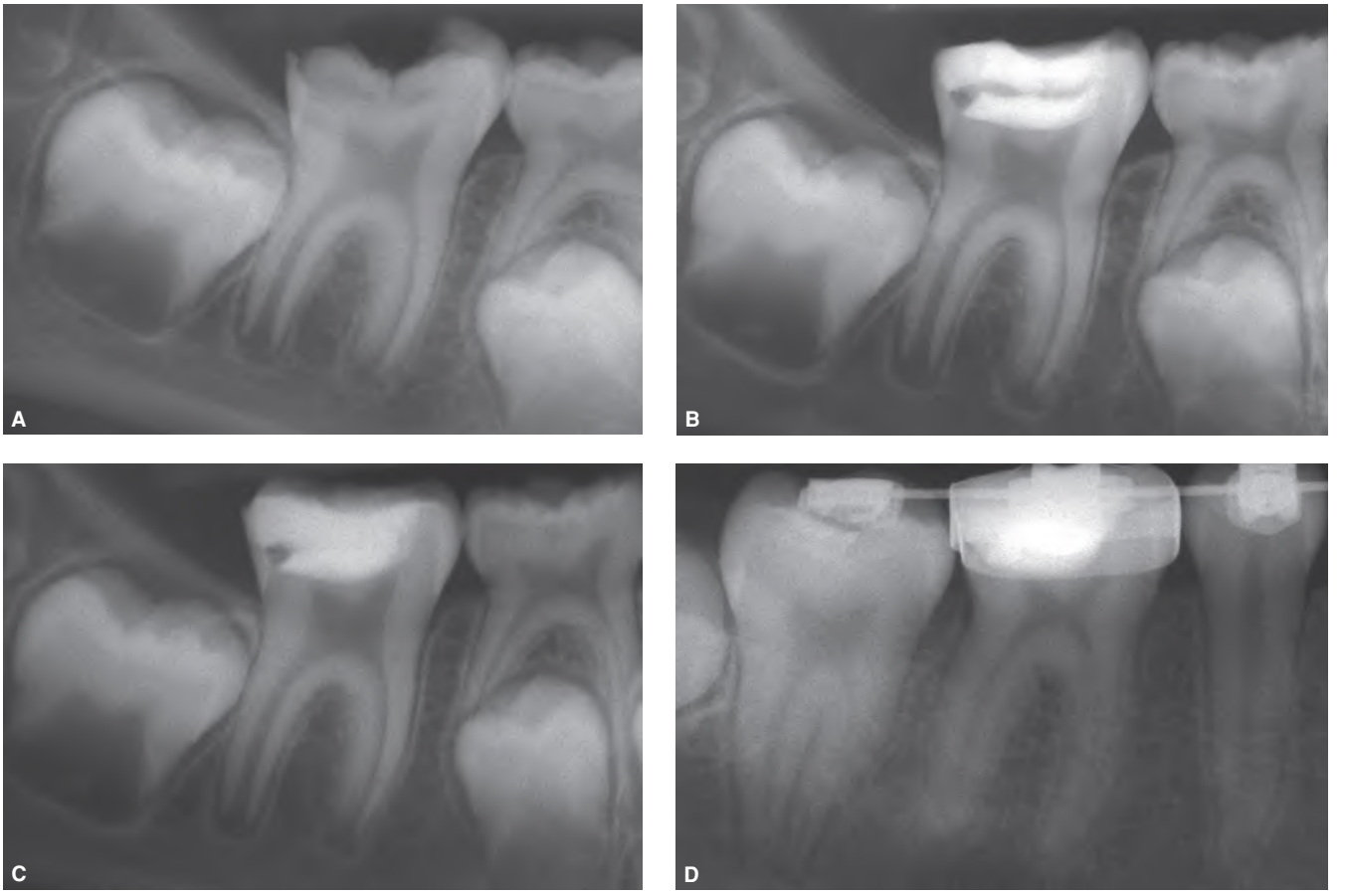

FIGURE 27-3 ***A.*** Seven-year-old female patient presents with a symptomatic mandibular first molar exhibiting deep caries and open apices. ***B,C.*** Patient received direct MTA pulp capping and bonded restoration in a two-visit sequence. ***D.*** Radiographic recall at seven years shows mature apices. Orthodontic treatment ongoing. Normal response to cold testing. (Reproduced with permission from Bogen and Chandler.[2])

Pulpotomy

Pulpotomy is a more invasive procedure defined as "the removal of the coronal portion of a vital pulp as a means of preserving the vitality of the remaining radicular portion. It may be performed as an emergency procedure for temporary relief of symptoms or therapeutic measure, as in the instance of a Cvek pulpotomy."[32]

The entire coronal pulp is removed to the level of the pulpal floor, or cementoenamel junction, and a capping material is placed over the canal orifices, or remaining radicular tissue. Some dressing materials with varying toxicity used for this purpose include ferric sulfate, phenol, creosote, glutaraldehyde, polycarboxylate cement, ZOE, CH, and formaldehyde that embalm the remaining tissue.[41] Although studies indicate that short-term success rates are favorable, this procedure is generally recommended for deciduous rather than permanent teeth. For more details, see Chapter 38, "Endodontic therapy in the pediatric patient." However, recent studies using partial and complete pulpotomies on adult permanent teeth have been published showing favorable outcomes in teeth exhibiting irreversible pulpitis.[42] Although formocresol is used widely as a pulpotomy agent in young adult teeth, the material presents distinct disadvantages.[43,44] Data suggests that the material possesses potential harmful systemic effects in humans and experiments in nonhuman primates have demonstrated a greater incidence of internal resorption.[45] Furthermore, conventional root canal treatment is difficult to complete due to the changes in the pulp apical to formocresol placement.[46] Recent investigations support MTA and other CSCs as practical replacements for formocresol in primary and immature permanent teeth (Figure 27-5).[47–50]

Partial Pulpotomy

Partial pulpotomy (Cvek pulpotomy) is described as "the removal of a small portion of the vital coronal pulp as a means of preserving the remaining coronal and radicular pulp tissues."[32] Healthy coronal pulp tissue is, therefore, exposed after removing inflamed or necrotic tissue.[51] Both direct pulp capping and partial pulpotomy are considered comparable procedures and differ only in the extent and volume of vital tissue remaining after treatment (Figure 27-6).

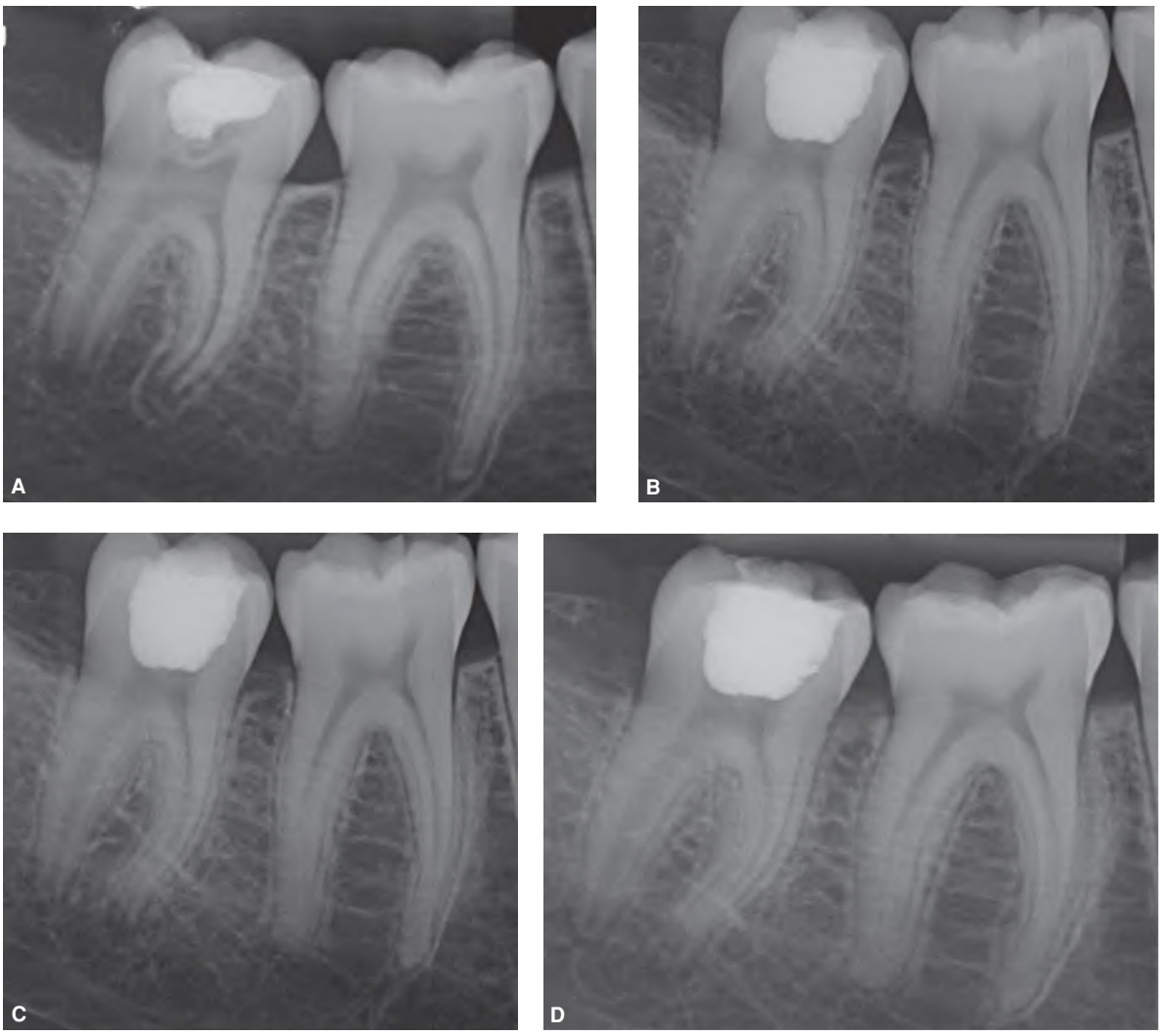

FIGURE 27-4 Indirect pulp cap in a mandibular right second molar in a 13-year-old male patient. ***A.*** First step indirect pulp cap (stepwise excavation) after treatment interval of four months with retained arrested deep layer of carious dentin in innerpulpal quarter. ***B.*** Post-operative radiograph after completed step-wise excavation (two-step indirect pulp cap). ***C.*** One-year radiographic review. ***D.*** Two-year follow-up with completed apexogenesis, pulp sensibility present and well-defined lamina dura evident. (© Dr. Lars Bjørndal 2015. All rights reserved.)

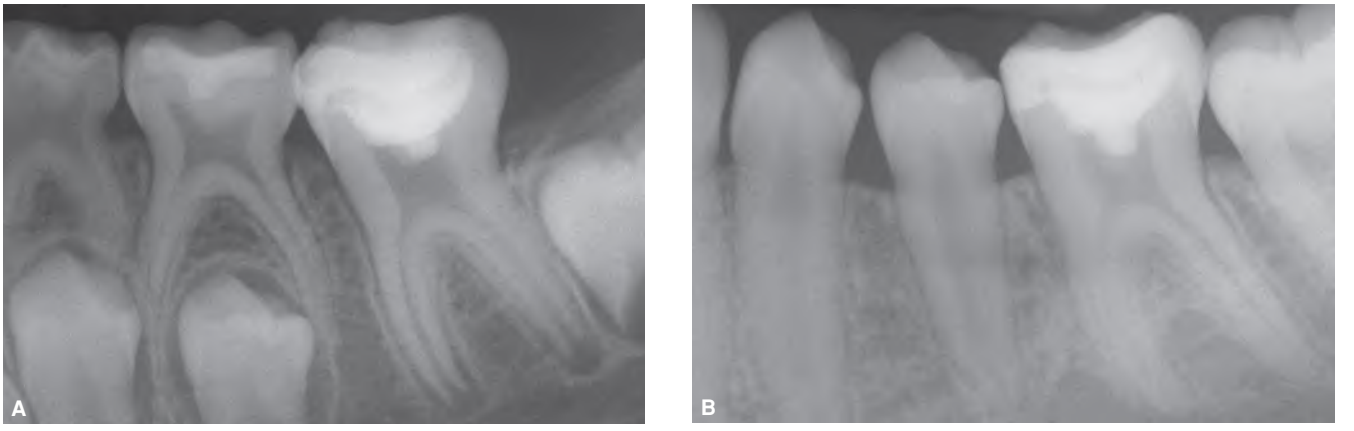

FIGURE 27-5 ***A.*** Post-operative radiograph of mandibular left first molar in a 9.5-year-old female patient with hypoplastic enamel and extensive caries. The molar was treated with MTA partial pulpotomy, after NaOCl hemostasis, and restored with bonded composite. ***B.*** Seven-year follow-up radiograph showing complete apical root formation. (Courtesy Dr. Jonathan Lo, Long Beach, CA.)

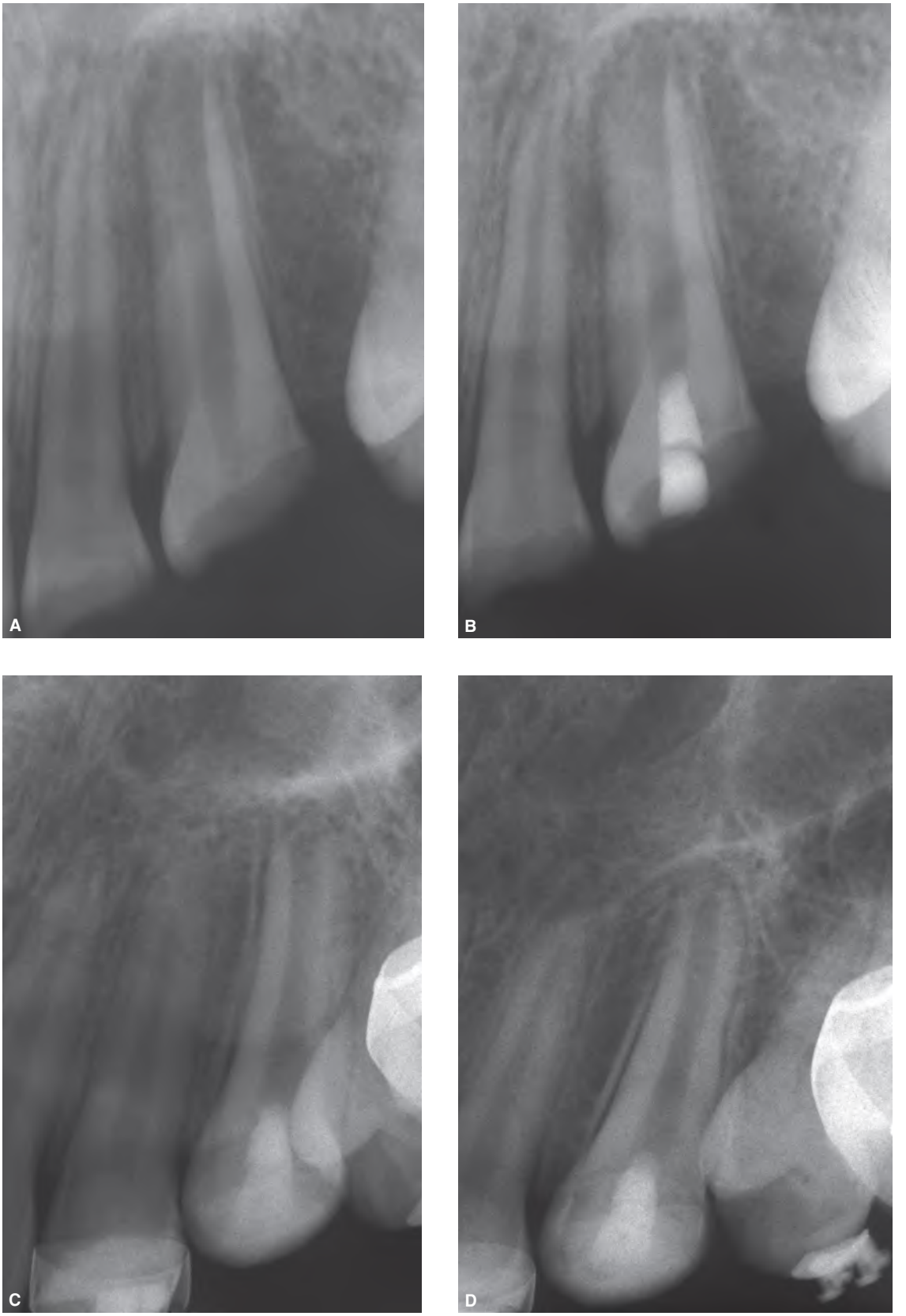

FIGURE 27-6 ***A.*** 12-year-old patient presents 24 hours after oblique coronal fracture of his maxillary right canine exhibiting a large open apex. ***B.*** Shallow pulpotomy (Cvek) completed with MTA after 5.25% NaOCl hemostasis. ***C,D.*** Six-month and one-year radiographic recalls showing apical closure. The tooth tested within normal limits to cold testing at both time periods. (Reproduced with permission from Bogen and Chandler.[2])

INDICATIONS FOR VITAL PULP THERAPY

Vital pulp therapy is indicated whenever the remaining pulp exhibits reversible pulpitis. It can be induced to generate a reparative hard tissue barrier that protects the dental pulp from further microbial invasion. In order to potentiate long-term pulpal preservation, direct pulp capping is recommended for teeth with deep caries, mechanical exposures, or traumatic injuries. The initial diagnosis, based on radiographic evaluation, pulp testing, patient history and clinical evaluation, will determine the suitability and probable outcome for direct pulp capping and pulpotomy procedures. The prime objective is to maintain pulp vitality, and therefore, postpone more aggressive endodontic and restorative therapies that may decrease the long-term prognosis. Teeth receiving orthograde root canal therapy (RCT), post and core placement, cuspal or full coverage, will exhibit lower long-term survival rates than teeth with vital pulps.[52–57] Moreover, teeth that have undergone RCT and are restored with prosthetic crowns show a higher vulnerability for root fractures, caries, and other plaque-related complications.[58–60]

VITAL PULP THERAPY MATERIALS

To identify ideal biocompatible and bioactive materials, researchers have, for decades, investigated a multitude of vital pulp therapy agents. These have included CH compounds,[61–66] zinc phosphate and polycarboxylate cements, zinc oxide, calcium phosphate, calcium-tetracycline chelate, calcium phosphate ceramics, antibiotic and growth factor combinations, Bioglass, Emdogain, cyanoacrylate, hydroxyapatite, resin-modified glass-ionomers, hydrophilic resins and recently MTA and other CSCs.[67–78] Similar research has focused on materials such as Ledermix, dimethyl isosorbide, glycerrhetinic acid/antibiotic mix, and potassium nitrate.[79]

Advanced methods to eliminate caries progression include laser and ozone technology and also strategies that activate pulpal defenses using bioactive agents.[80–82] Outcomes from former human retrospective studies, evaluating direct pulp capping in permanent teeth, are variable and depend on techniques and materials that show a wide range of success rates between 30% and 85% over 5- to 10-year periods.[61–66]

The ideal pulp capping material should be sterile and display the following characteristics[83]: stimulate hard tissue repair, maintain pulpal vitality, release fluoride to prevent secondary caries, should be bactericidal or bacteriostatic, adhere to dentin, adhere to restorative material, resist forces during restoration placement, resist forces under the restoration during its lifetime, be radiopaque, and provide a bacteria-tight seal.

Calcium Hydroxide

Calcium hydroxide has long been considered the "standard" material in vital pulp therapy. Although the compound has been shown to have some desirable properties, investigations in humans have shown that success rates decrease as observation periods increase.[63–66] The high alkaline pH provides a favorable bactericidal characteristic that also promotes pulp tissue irritation, which in turn stimulates pulpal defense and repair.[70] However, CH exhibits disadvantages that include cell culture cytotoxicity, pulp cell apoptosis, poor marginal adaptation to dentin, and inconsistent stimulation of reparative hard tissue formation.[84–87] The material can also degrade and absorb beneath restorations, is associated with primary tooth resorption, and can encounter interfacial failure during amalgam condensation.[88–90] When used in conjunction with bonding resins, the compound demonstrates gap formation at the dentin interface.[91] Moreover, hard tissue bridges beneath CH exhibit tunnel defects and the material is ineffective at providing a long-term seal against microleakage when used as a pulp capping agent.[84,90] The instability and disintegration of CH under permanent restorations, coupled with defects in the hard tissue bridge, afford microorganisms a pathway for penetration into vulnerable pulpal tissue and the subsequent activation of circulating immune cells, thus promoting pulpal irritation, pulpal calcification and potential canal obliteration (Figure 27-7).

Adhesive Resins and Resin-Modified Glass-Ionomers

In the early 1980s, researchers in Japan first introduced hydrophilic resin systems for direct pulp capping.[92–94] Encouraging results were seen in preliminary research based on ISO standards using non-human primate models.[95–99] Favorable biocompatibility and reparative bridge formation was documented in exposed pulps directly capped with various resins and evaluated histologically for pulpal reaction, microbial presence and reparative hard tissue formation.

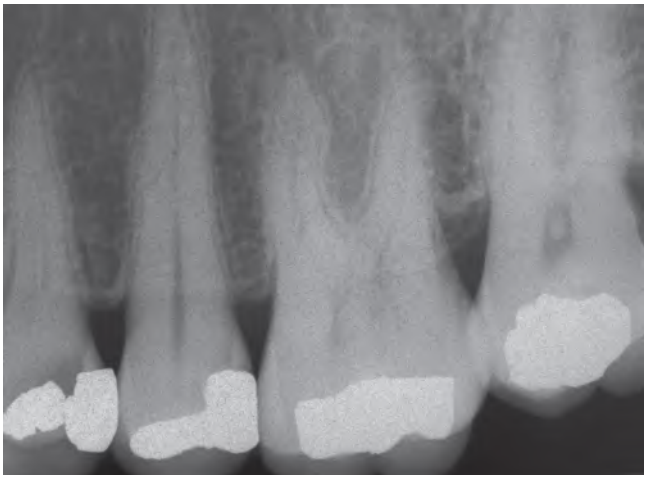

FIGURE 27-7 Periapical radiograph of a maxillary left molar in a 46-year-old female. The patient received a direct pulp cap in the second molar with hard setting calcium hydroxide (Dycal®) approximately 10 years before. The molar exhibited a delayed response to cold testing. Dystrophic calcification can be seen including a large pulp stone.

However, although these favorable results were observed in non-human primates, the transitional use of these materials was not mirrored in human subjects.[100–104] Unfavorable histological reactions to adhesive systems have been observed in clinical investigations when directly placed against pulp tissue in humans. Histological sections from various studies reveal cellular incompatibility characterized by mononuclear inflammatory infiltrates, PMN's, macrophages, multinuclear giant cells, blood extravasation between the layers of the bonding resin, and an absence of calcific tissue formation.[100,101,104,105]

Direct pulp capping with either a hydrophilic resin or resin-modified glass-ionomer cement has been compared in two clinical studies in human subjects.[106,107] Histological results from one study showed that both Clearfil Liner Bond 2 (Kuraray Co., LTD, Osaka, Japan) and Vitrebond (3M Espe Dental Products, St. Paul, Minnesota) initially generated a moderate to intense inflammatory cellular response failing to stimulate reparative bridge formation after 300 days. The unpredictable nature of reparative hard tissue formation using hydrophilic resins, and the potential contamination of calcific bridges by microorganisms, has been confirmed in other non-human investigations. One contemporary study measured the degree of bacterial contamination, associated with "tertiary dentin" bridge formation in primates, revealing contamination in 18.6% of resin-based composite, 22.2% of resin-modified glass-ionomer, and 47.0% of CH pulp-capped specimens.[108]

These investigations suggest that some pulp capping materials do not promote predictable pulpal healing, nor do they provide a hospitable environment for reparative bridge formation and the exclusion of microorganisms.[109,110] However, repair should proceed successfully beneath the material when bacterial microleakage is prevented and hydrophilic resin capping can be successful in some cases (Figure 27-8). Progressive studies, incorporating growth factors and hard tissue promoting agents, coupled with adhesive resin systems to encourage repair and wound healing are emerging.[111,112]

Mineral Trioxide Aggregate

Mineral Trioxide Aggregate was the first CSC, introduced as a root canal repair material in the early 1990s.[113] The initial composition of this bioactive CSC included tricalcium

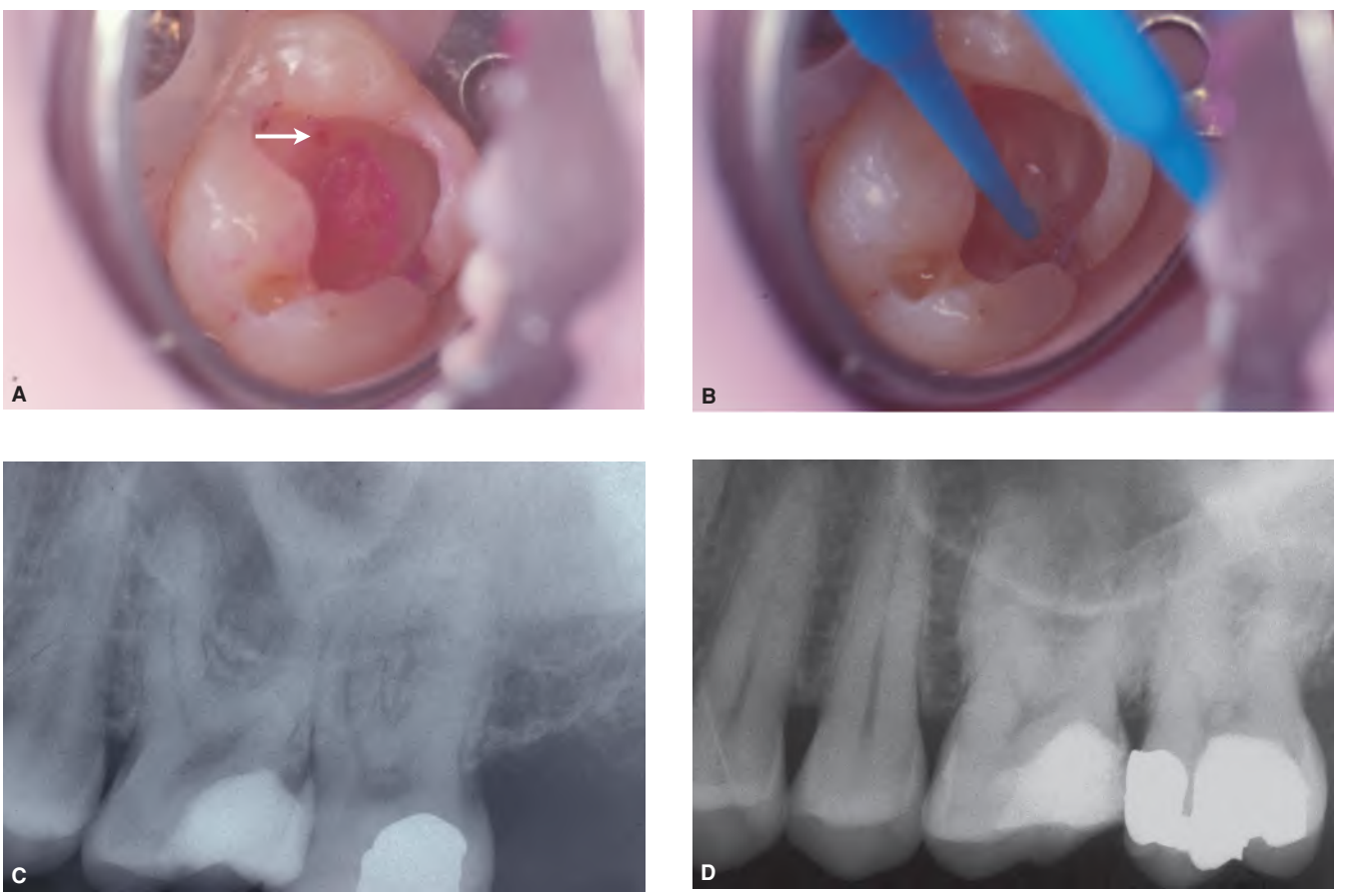

FIGURE 27-8 16-year-old female patient with a deeply carious maxillary left first molar diagnosed with reversible pulpitis. ***A.*** Pulp exposure (0.5 mm) after caries detector dye staining (arrow). ***B.*** Direct hydrophilic resin pulp cap after NaOCl hemostasis. ***C.*** Two-year radiographic recall showing reparative bridge formation. ***D.*** 15 years and 6 month radiographic review. The patient was symptom free and the tooth responded to cold testing within normal limits. Note presence of pulp stone in second molar after indirect pulp cap with calcium hydroxide eight years previously. (© Dr. George Bogen. All rights reserved.)

silicate, tricalcium aluminate, tricalcium oxide, silicate oxide, and other mineral oxides.[114] The composition of the original cement, ProRoot MTA®(Tulsa/Dentsply, Tulsa, OK), has changed since its introduction with the substitution of dicalcium silicate for tricalcium silicate and the addition of tetracalcium aluminoferrite, calcium sulfate dehydrate and bismuth oxide; the latter was added to impart radiopacity.[115] White MTA (WMTA) was later produced to resolve esthetic concerns by ferrite (Fe_3O_3) reduction with no detectable change in clinical performance compared to the original gray powder.[115–118] However, staining from WMTA is still an undesirable characteristic, and innovative methods have been recommended to overcome this drawback.[119]

The favorable physicochemical properties exhibited by MTA make it a superior material when used as a cement for partial or complete pulpotomy and direct pulp capping in both primary and permanent teeth.[120–122] Portland cement is structurally similar to MTA, and shares MTAs favorable properties that include the ability to harden in the presence of blood and moisture.[123] Set MTA exhibits superior marginal adaptation and the cement is non-resorbable. It forms a reactionary interfacial layer with dentin, resembling hydroxyapatite in structure when setting in the presence of calcium ions and tissue fluids.[124,125] The slow release of calcium ions, small particle size, and sustained alkaline pH also characterize this bioactive cement.[126] On a cellular level, MTA stimulates cytokine release, induces pulpal cell proliferation, and promotes hard tissue formation.[127,128] The slow calcium release and combined alkaline pH of 12.5 may contribute to the reduction of microorganisms remaining after caries excavation.

When curing, MTA and other CSCs exhibit crystal formation within dentinal tubules, in the presence of alkaline phosphate, that are responsible for microbial entombment and neutralization.[129] The high pH also encourages the extraction of growth factors nested in the adjacent dentin that contribute to calcific bridging.[127,130] Direct pulp capping using MTA demonstrates a strong capacity to generate reparative calcific barriers in primate and canine models.[74,131–134] Current investigations in humans using MTA for partial or complete pulpotomies and direct pulp capping have shown favorable short and long-term outcomes.[135–141] These and other studies confirm that the material does provide a biocompatible, noncytotoxic and antibacterial environment, and a favorable surface morphology that allows for cell attachment and proliferation.[142] The soluble components of MTA, released during setting at the dentin interface that form hydroxyapatite, may also encourage the release of growth factors and other bioactive molecules such as transforming growth factor beta (TGF-β1) and adrenomedullin.[143] Increased levels of dentin extracellular proteins are stimulated by the presence of MTA and culminate in hard tissue bridge formation after stimulating calcific repair mechanisms.

Recent investigations in humans comparing the efficacy of MTA with CH for direct pulp capping have revealed the distinct biological advantage of MTA in promoting repair and maintaining prolonged pulp vitality.[140,141] Mineral trioxide aggregate direct pulp capping was examined using a two-visit protocol in permanent teeth, diagnosed with reversible pulpitis.[144] Forty-eight of 49 teeth (97.96%) with an average observation period of 3.94 years showed favorable outcomes, based on subjective symptomology and cold testing. Pulpal calcifications were seen in 10.6% (5/49) of cases and all immature permanent teeth (15/15) exhibited complete root-end closure (apexogenesis) when open apices were present initially.

A practice-based, randomized clinical trial evaluated 376 teeth for up to two years comparing the success of direct pulp capping in permanent teeth using either MTA or CH. It revealed that the probability of failure at two years for CH was 31.5% versus 19.7% for MTA, confirming the superior clinical performance of MTA.[140] Another controlled cohort study examined 229 teeth and compared MTA direct pulp capping with CH during 2 to 10 year recall period using logistic regression and equation logit models; 80.5% of teeth in the MTA group showed better long-term results compared to only 59% of teeth in the CH group.[141]

The remarkable outcomes for direct pulp capping using MTA are attributable to its inherent bioactive and bioinductive properties.[145] The anti-bactericidal characteristic of the set cement, due to the sustained alkaline pH, coupled with sodium hypochlorite (NaOCl) application, contribute to the elimination of the majority of residual microorganisms remaining at the dentin/pulp interface after caries removal and pulp exposure. MTA is hygroscopic and hardens in the presence of moisture, so direct contact with blood, or tissue fluids, does not significantly affect its curing properties. Moreover, microleakage and bacterial growth are impeded due to the small particle size and the close adaptation of the CSC to dentin.[125,129] Hard tissue formation is encouraged by the slow release of calcium ions that stimulate growth factors from the dental pulp and promote signaling molecules such as TGF-β, IL-1α, IL-β and MCSF.[143,146] The surface texture and compressive strength of the set cement also allows for intimate chemical bonding with adhesive restorations and provides minimal compression under heavy occlusal loading when a final restoration is provided.

Calcium Silicate-Based Cements (CSCs)

Since the introduction of MTA, a broad assortment of newly formulated CSCs have become available for applications in endodontics and specifically vital pulp therapy.[78,147–150] The major constituents of these new cements include dicalcium and tricalcium silicate, the central components of both MTA and Portland cement. Due to their hydraulic properties, calcium silicate compounds attain immediate strength on hydration and investigations have shown these aggregates to up-regulate angiogenic and transcription factors as well as pulp fibroblasts and stem cells. Other characteristics of

CSCs include bioinductive capacities that positively influence cell proliferation, differentiation and induction of hard tissue barriers while producing minimum inflammation. Although clinical investigations in humans using the new CSCs are minimal, they all appear to have suitable properties for use as pulp capping and pulpotomy agents.[151–155]

There are currently over 25 MTA-like materials available and initial investigations demonstrate comparable physiochemical and bioinductive properties that appear beneficial for vital pulp therapy. Often referred to as bioceramic materials, some of these CSCs include MTA Angelus, MTA BIO and MTA Branco (MTA-Angelus, Londrina PR, Brazil), Biodentine™ (Septodont, Cambridge, Ontario, Canada), BioAggregate® and iRoot® BP Plus (Innovative Bioceramix, Vancouver, BC, Canada), Calcium Enriched Mixture (CEM) (Bionique Dent, Iran), EndocemMTA (Maruchi Inc., Wonju-si, Gangwon-do, South Korea) and Endosequence root repair material (Brasseler USA, Savannah, Georgia). Recently introduced hydraulic silicate cements also include RetroMTA and OrthoMTA (BioMTA, Seoul, Korea), Grey MTA Plus (Avalon Biomed Inc., Bradenton, Florida), MTA plus (Prevest-Denpro, Jammu City, India), Tech Biosealer Endo (Isasan SRL, Revello Porro, Italy), and Micromega® MTA (MicroMega, Besançon, France). Ongoing laboratory and clinical investigations of other calcium silicate–based compounds are currently confirming their safety and efficacy.[151]

MTA-Angelus is composed of 75% Portland cement and 25% bismuth oxide and has strong potential in vital pulp therapy. The removal of calcium sulfate in the formulation allows for a shorter setting time of 10 minutes, making the CSC superior for one-visit pulp capping or pulpotomy procedures.[156] The crystalline structures of MTA-Angelus are similar to gray and white ProRoot MTA, and when used as a pulp capping material in humans, MTA-Angelus produces hard tissue bridges histomorphologically comparable to those of ProRoot MTA.[155,157,158] The cement has demonstrated exceptional results when used in primary tooth pulpotomies[159] and as a pulp-capping agent on direct exposures in caries-free teeth.[158]

Biodentine™ also features a short setting time of 10 minutes and shows remarkable bioactive properties that can have beneficial applications for pulptomies and both indirect and direct pulp capping procedures. The cement does not alter human fibroblast cytodifferentiation and when measured with the Ames' mutagenicity test shows an absence of genotoxic/cytotoxic effects.[160–162] The sealing ability of Biodentine™ at the dentin interface appears to be similar to that of MTA when analyzed by SEM.[163] This compound exhibits pulp tissue compatiblity that induces fibroblast (odontoblast-like) cell recruitment and encourages calcific bridge formation comparable to MTA when used as a pulp capping or pulpotomy agent.[134,164,165]

BioAggregate® is another bioactive CSC cement that shows an exceptional capacity to stimulate hard tissue bridge formation. The cement has been shown to promote mineralization in osteoblast cells, inhibit osteoclast differentiation, and inhibit inflammatory bone resorption *in vivo*, similarly to MTA.[166–168] When in contact with human dental pulp cells, both MTA and BioAggregate promote cellular adhesion, migration, and attachment demonstrating excellent cytocompatibility.[169,170] BioAggregate, Biodentine, and MTA all stimulate hard tissue formation by advancing odontoblast-like cell differentiation and nodule formation during the mineralization process. The favorable properties of these new CSCs suggest applications in regenerative endodontic procedures as well as vital pulp therapy. (Figure 27-9). For more details, see also Chapter 29, "Regenerative endodontics."

Endosequence root repair material shows a high potential as a pulp capping and pulpotomy agent demonstrating low cytotoxicity, exceptional bioactivity, and impressive healing characteristics when used in endodontic surgery.[171–173]

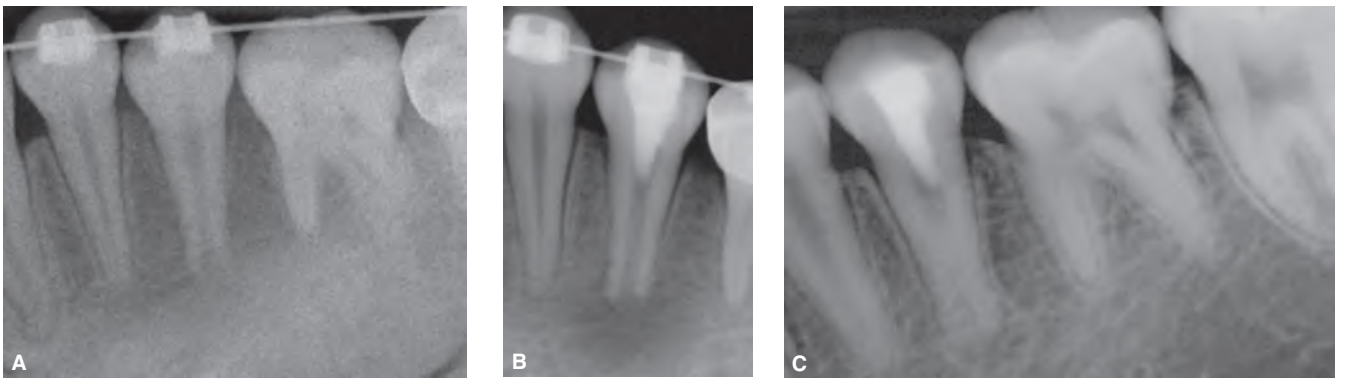

FIGURE 27-9 Regenerative endodontic procedure in a mandibular left second premolar associated with a dens evaginatus and necrotic pulp. The 15-year-old patient presented with acute swelling and full orthodontic banding. ***A.*** Preoperative radiograph showing open apex and presence of periradicular pathosis associated with the affected tooth. ***B.*** Post-treatment radiograph after initial treatment with 1% NaOCl and calcium hydroxide, followed by blood clot induction and placement of Bioaggregate® restored with bonded composite. ***C.*** Radiographic review after two years demonstrating continued root growth, wall thickening and apical closure. (Courtesy of Dr. Mark Olesen, North Vancouver, BC, Canada.)

Calcium enriched mixture (CEM) cement has demonstrated excellent bioactivity and remarkable efficacy when used in vital pulp therapy.[174] In a 5-year study CEM, when used as a pulpotomy agent in permanent molars diagnosed with irreversible pulpitis, showed similar outcomes to conventional root canal treatment in a multicenter randomized clinical trial.[42] The findings of this study could have a strong impact on treatment options for patients in economically challenged geographical areas worldwide. The future potential for the new generation of CSCs as beneficial agents in vital pulp therapy and regenerative endodontics will accelerate as research expands and innovative CSCs are further developed.

DIAGNOSTIC CRITERIA FOR SUCCESSFUL OUTCOME

Treatment for the immature permanent tooth with advanced carious lesions include several options that preserve the remaining and identifiable healthy tissue. The coronal pulp tissue can be preserved *in toto*, partially removed, or removed entirely to the base of the pulpal floor, as long as the critical radicular pulp tissue is preserved to allow continuing development and apical maturation (apexogenesis) in teeth with open apices. Induction of apexogenesis should be the clinician's primary goal, with the pulp protected and encouraged to remain vital in cases of trauma where tooth development may be interrupted.[175–177] For more details, see Chapter 28, "Management of teeth with immature apices."

The clinician must make a careful assessment of all available information before treatment can be initiated. Critical aspects of vital pulp therapy include a differential diagnosis based on medical history, radiographic evidence, and patient report combined with pulp sensitivity (cold) and percussion testing. Clinical assessment should include the degree of mobility, periodontal probing and the presence of localized swelling or sinus tracts. Both periapical and bitewing radiographs must be evaluated for evidence of caries, periapical disease, internal/external resorption defects, furcation radiolucencies, and pulp calcification due to trauma or prior restorations.

Subjective symptomology must be considered after clinical and radiographic assessments have established an absence of disease. The majority of patients with deep carious lesions often experience sensitivity to heat, cold, and certain acidic or sweet foods. However, the subjective response to cold testing can be inconsistent and a short lingering response (one–two seconds) may not be a signal that the pulp is irreversibly involved. Moreover, cold testing responses have been shown to be unpredictable, especially in some individuals with immature permanent teeth.[178,179] The clinician must also recognize that pain to percussion is most often associated with irreversible pulpitis. In teeth diagnosed with irreversible pulpitis and open apices, promotion of root-end closure (apexogenesis) is recommended using MTA or other CSC with pulpectomy or regenerative procedures.[177,180,181] (Figure 27-10). For more details, see Chapter 28.

Teeth with initial caries have a higher probability for repair and calcific bridge, or tertiary dentin formation, than teeth that have a history of previous restorations or trauma.[182,183] Direct pulp capping may not be indicated for mature permanent teeth planned for full coverage restorations, or with a prior history of extensive restorative care. However, from a technical standpoint, full crown preparations with occlusal pulp exposures may have a more favorable prognosis. Pulp exposures on the axial walls of full coverage preparations are extremely difficult to treat clinically due to the handling properties of MTA and other CSCs. Considering the technical challenge of placing the material against an exposure on a vertical wall, orthograde RCT can be recommended as a more predictable treatment option in permanent teeth.

Selecting an appropriate vital pulp treatment should be based on the extent of the remaining tooth structure. Pulpotomy rather than direct pulp capping is recommended for teeth in young patients exhibiting advanced caries and severe coronal breakdown, or requiring full coverage. In patients with rampant caries, pulpotomies are recommended as opposed to direct

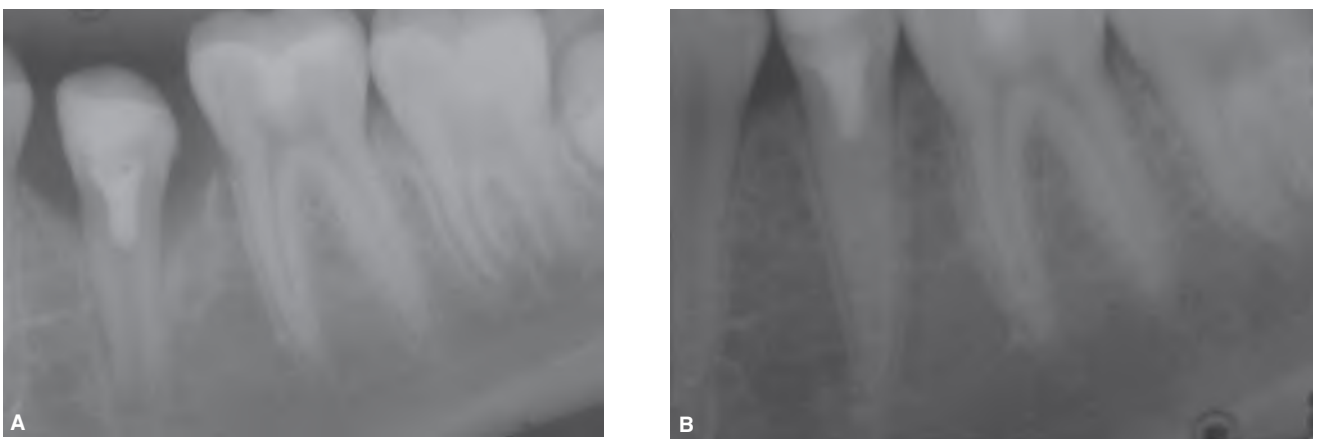

FIGURE 27-10 ***A.*** Post-operative radiograph of a hypocalcified mandibular left second premolar in an 11-year-old female patient. The tooth received a full pulpotomy after a large carious exposure. Hemostasis obtained with NaOCl followed by a 5 mm MTA plug, glass ionomer coverage and restoration with composite. ***B.*** Radiographic recall at 2.5 years showing root maturation (apexogenesis). (Courtesy of Dr. David Taylor, Rolling Hills Estates, CA.)

pulp capping, since the majority of these patients will exhibit recurrent caries at a higher rate.[184–186] However, young patients who have caries associated with reversible pulpitis on all first molars can still be considered excellent candidates for direct pulp capping. It is also evident that the prognosis for vital pulp therapy diminishes with the increasing age of the patient.[64,186] Nevertheless, clinical success can be seen in older patients when diagnosed with reversible pulpitis (Figure 27-11).

When the pulp exposure is visualized under magnification, and hemorrhage control assessed, the clinician can reconfirm the initial diagnosis. If non-hemorrhaging tissue is discovered, it is most likely necrotic, and must be removed until bleeding tissue is encountered. After achieving hemostasis, a partial or miniature pulpotomy can be completed with a high-speed round diamond bur using MTA or a CSC placed directly against the entire wound and surrounding dentin. When bleeding cannot be controlled on direct pulp exposures with 5.25% to 8% NaOCl after a 10- to 15-minute contact period, the diagnosis must be changed to irreversible pulpitis and pulpotomy or pulpectomy is recommended. The option that offers the greatest benefit to the patient and secures the optimum prognosis for the long-term retention of the tooth determines which type of vital pulp therapy to be selected (Figure 27-12).

A greater risk of an unexpected and sizeable pulpal exposure may possibly occur since pulp sizes are underestimated on radiographs.[187] However, it is known that the size of an exposure has no influence on the outcome in direct pulp capping.[188] Some clinicians erroneously assume that larger exposures have a poorer prognosis and may consider size in their decision-making process. In a study involving the examination of simulated exposures (0.5–0.9 mm), dentists overestimated exposure size by a mean of 26% when not using a ruler or prior calibration.[189] Pulp dimensions between the genders and among racial groups may also vary considerably.[190]

CARIES REMOVAL

Thorough caries removal under dental dam isolation, aided by optical magnification and a caries detector dye, are essential tools in attaining favorable outcomes for direct pulp capping and pulpotomy procedures. Our understanding of the carious process was redefined in the mid-1970s when an objective

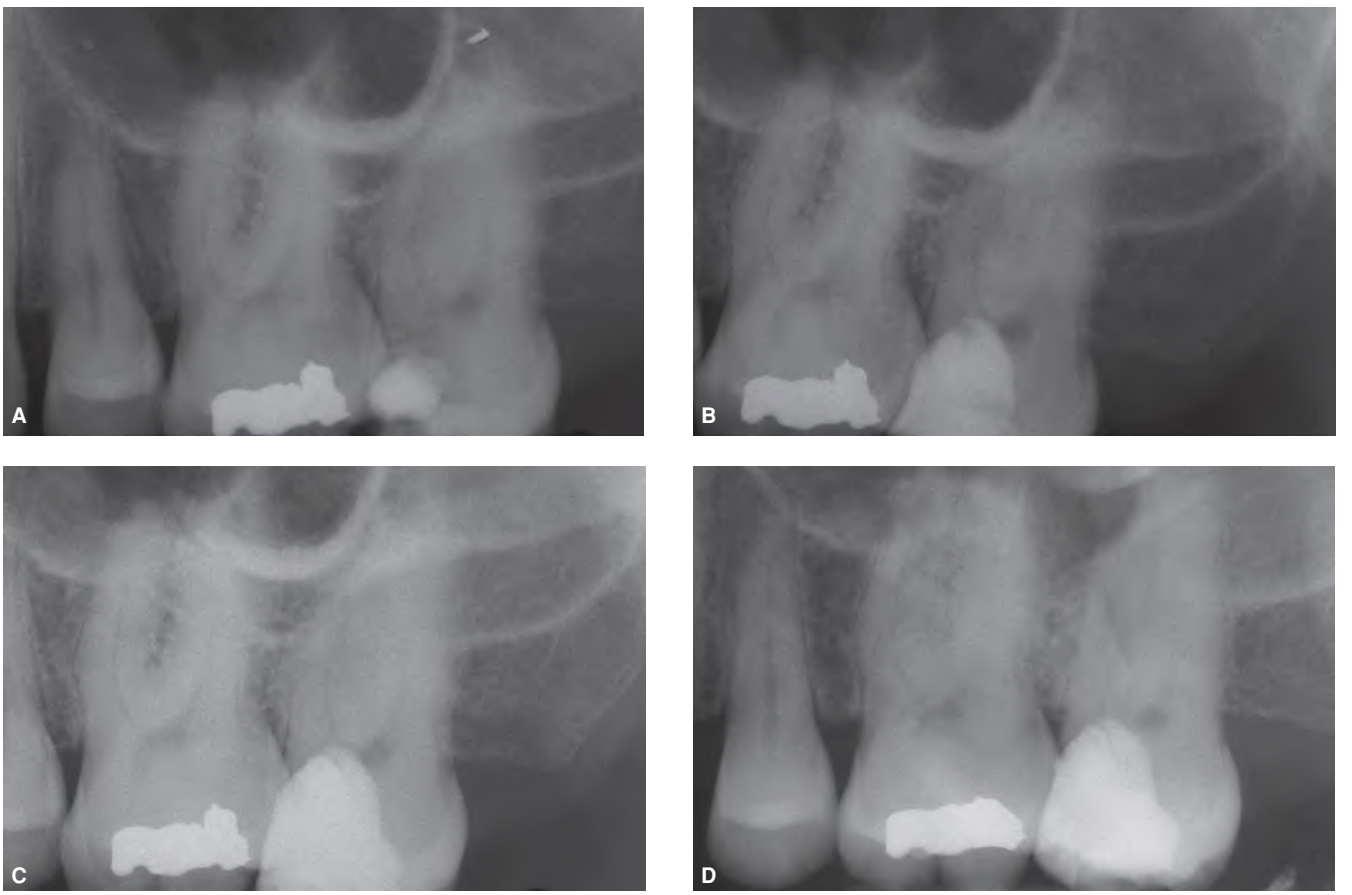

FIGURE 27-11 48-year old male presents with a deeply carious and asymptomatic maxillary left second molar. ***A.*** Pre-operative radiograph reveals extensive mesial caries, pulp calcification and ZOE provisional restoration. ***B.*** Post-operative radiograph after 1.0 mm exposure, NaOCl hemostasis, MTA direct pulp cap, wet cotton pellet placement and Photocore® provisionalization. ***C.*** Radiograph taken one year after pulp capping and placement of a permanent bonded composite restoration. The patient was asymptomatic and the treated tooth responded to cold testing within normal limits. ***D.*** Three-year radiographic follow-up. The treated tooth responded to sensitivity tests within normal limits. (© Dr. George Bogen. All rights reserved.)

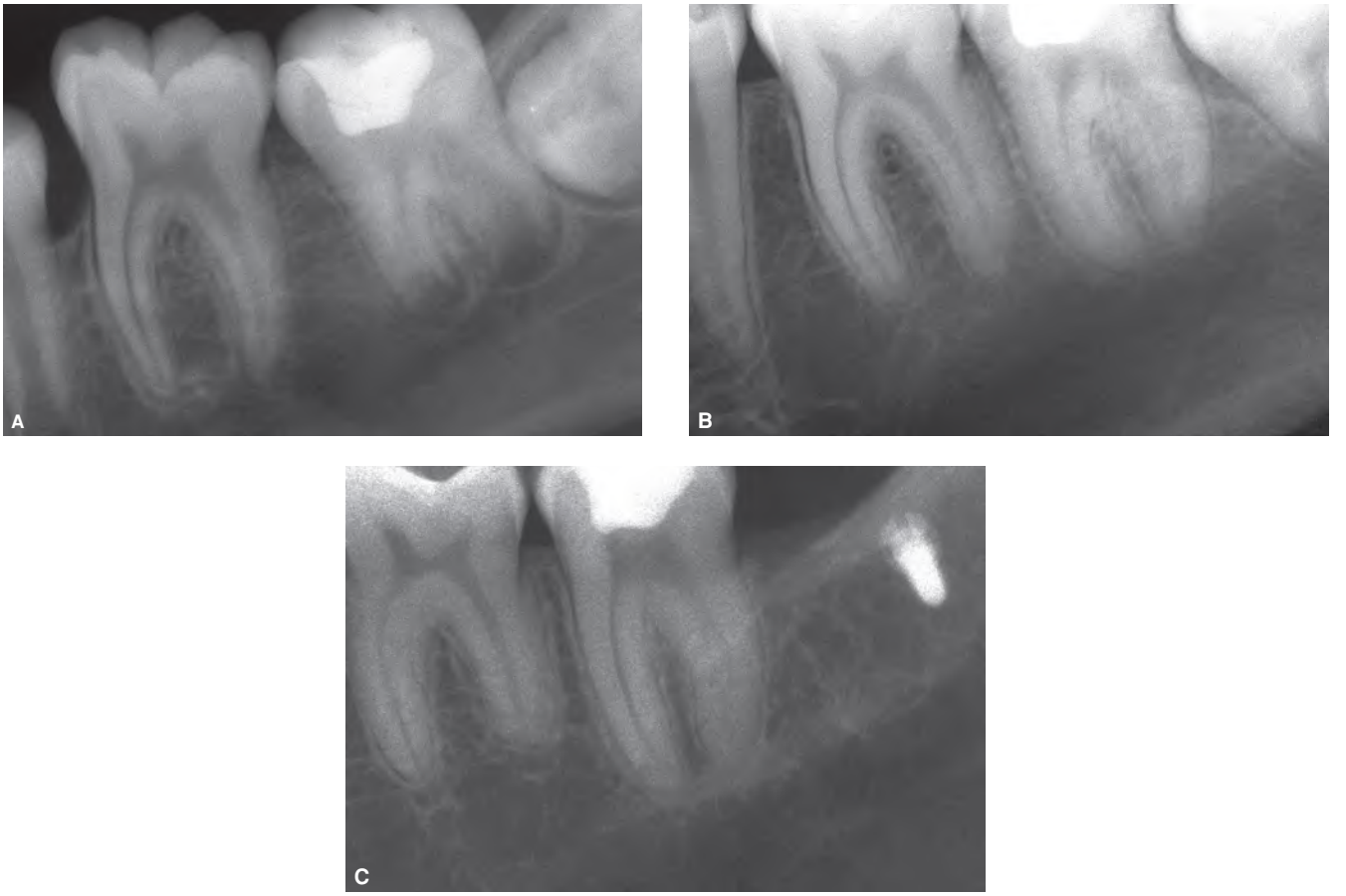

FIGURE 27-12 ***A.*** Nine-year-old male after second molar direct pulp capping with MTA and bonded composite restoration completed in a two-visit sequence. ***B,C.*** Radiographic recalls at 4.5 years (B) and 10 years (C). Normal root maturation can be seen at the 4.5 year recall period. Titanium screw in third molar position placed after jaw fracture three years earlier (C). (Reproduced with permission from Bogen and Chandler.[2])

technique for removing the outer infected carious layer was discovered. This technique, using a caries detector dye, preserved the remineralizable inner carious layer, thus preserving tissue critical for continued pulpal protection and survival.[191,192] The staining process of carious dentin is based on the principle that acidogenic Gram-positive bacteria, whose main byproduct is lactic acid, first break down hydroxyapatite and subsequently collagen in the outer carious layer, but spare bound collagen in the second layer, where the intermolecular crosslinks are still intact.[193,194] When the outer layer of two distinctive carious layers can be selectively stained and carefully removed, repair and pulpal preservation can proceed normally when teeth are sealed with advanced bonded composite systems.

The invading bacteria at the furthest point of microbial penetration are responsible for the production of proinflammatory mediators at the pulp/dentin interface. This challenge to the pulp is minimized by removing the necrotic dentin in the outer layer and by preserving the second (caries affected) layer that will remineralize. This remineralization process occurs when whitelockite crystals are formed in the presence of calcium and phosphate ions that block the tubules. Evidence of caries-affected inner layer dentin remineralization has been demonstrated in both non-human primate and dog models.[195,196] Contributing further to pulpal survival are adhesive composite restorations that are no longer dependent on retentive cavity preparations, hence sparing more tooth structure and generating less trauma to the dental pulp. Conservative objective caries removal promotes pulpal preservation by not injuring the remaining nonstaining ***caries-affected dentin***. The use of caries detector dyes allows the operator to visually inspect infected dentin using the dental operating microscope (DOM) that may have been overlooked and potentially compromise the treatment outcome.[197–200]

The efficacy of caries detector dyes has been controversial, since the possibility for residual acidogenic bacteria exists in unstained dentin while non-infected dentin can potentially be stained.[197,201,202] It has also been shown by atomic force microscopy and transverse digital microradiography that heavily stained carious dentin still contains mineralized tissue and may be worth retaining.[203] These drawbacks to using caries detector dyes could serve as a strong argument for indirect pulp capping strategies. However, the clear advantage during direct pulp capping procedures is reducing the probability of leaving active carious tissue that may have been otherwise gone undetected, together with improved caries removal guidance while using the DOM.

TISSUE HEMOSTASIS

Many antimicrobial materials and hemostatic agents have been recommended for use in vital pulp therapy. These include ferric sulfate, disinfectants such as Tubulicid ® (Global Dental Products, North Bellmore, New York) and Concepsis ® (Ultradent Products Inc., South Jordan, Utah), epinephrine, varying concentrations of hydrogen peroxide (H_2O_2) and NaOCl. Direct pressure at the exposure site with cotton pellets moistened in saline, or sterile water, is the most universally accepted method to control hemorrhaging of exposed pulps. Lasers and electrosurgery have demonstrated limited efficacy in hemorrhage control.[204] However, NaOCl is an accepted antimicrobial irrigant used in orthograde endodontic therapy and has been promoted since the late 1950s as an effective hemostatic agent in direct pulp capping and pulpotomy.[205,206]

When used in vital pulp therapy, NaOCl solutions provide several advantages that demonstrate its superiority over other agents. This agent contributes to the chemical amputation of the fibrin and blood clot, biofilm removal, clearing of dentinal chips, cavity interface disinfection, removal of damaged cells at the exposure site, and provides excellent hemostasis.[204,207,208] Concentrations of 5.25% to 8.0% NaOCl primarily affect the peripheral pulp cells without impairing underlying pulp tissue.[188,209] Although NaOCl exhibits a strong organic dissolving capacity, the reaction toward the the pulp tissue appears relatively benign.[210] However, when used for care of burn victims, as a fluid dressing in concentrations greater than 0.025%, it has shown to be detrimental in wound healing.[210]

The importance of hemostasis in vital pulp therapy was demonstrated in a landmark study where teeth were directly pulp capped in a carious field, using a fast set CH.[188] The 2-year success rate was 81.8% using a caries detector dye and hemorrhage control, completed with 10% NaOCl. Statistical analysis demonstrated that responses to thermal stimuli and percussion, diameter of the pulp exposure, age of the patient and types of teeth, had little influence on the success rate. However, the ability to arrest bleeding at the exposure site was the foremost variable and prognosticator for a successful outcome. This observation accentuates the relevance of complete hemostasis when employing an effective agent and how it affects the outcome of direct pulp capping and pulpotomy procedures.

Inflammatory mediators including IgA, IgG, IgM, prostaglandin E_2, and elastase are present at higher levels in clinically inflamed pulps.[211] Greater levels of mediators during neurogenic inflammation can affect the level of intrapulpal pressure and, therefore, the probability of securing pulpal hemostasis. Observational data suggests that if pulpal hemostasis cannot be attained within 10 to 15 minutes, the diagnosis should be revised to ***irreversible pulpitis*** and pulpotomy or pulpectomy should be initiated.

Exposed vital pulp tissue was investigated histologically in beagle dogs by studying freshly cut dentin prepared to depth of 2 mm and treated with 5.25% NaOCl. No inflammatory cells were detected at one and four week periods after the cavities were sealed with Cavit®. Teeth with vital dentinal tubules and recently prepared dentin do not appear to show any further pulpal damage when rinsed with undiluted 5.25% NaOCl.[212] In addition, pulps of non-human primates, directly capped with adhesive resins, were not adversely affected by NaOCl at varying concentrations.[207,208]

One investigation examined human primary teeth pulp capped with either CH or various adhesive bonding agents.[213] The teeth were clinically and radiographically evaluated for a period of 24 months after 1.25% NaOCl was used for 60 seconds, to ensure hemorrhage control. Results revealed a 93% survival rate when exfoliations were excluded. Sodium hypochlorite hemostasis did not impair biological repair and subsequent calcific barrier formation when used as a hemostatic agent. This finding was again demonstrated in a study where the outcomes for pulpotomies were compared using either ferric sulfate ($FeSO_4$) or NaOCl on primary teeth restored with an IRM base/stainless steel crowns.[214] A short-term one year evaluation revealed a 100% retention rate of teeth in the NaOCl group with 79% radiographic success, superior to the $FeSO_4$ group. Ferric sulfate has been recommended as a replacement for formocresol in pulpotomies on deciduous teeth due to its effective hemostatic characteristics. However, the agent interferes with the bond strength of adhesive resins and its use is not recommended when bonded restorations are intended.[215]

Direct pulp caps were also examined in a histological study completed on human third molars 30 and 90 days after pulp capping with either CH or a self-etching adhesive system and using 2.5% NaOCl for hemostasis.[216] The study revealed that CH performed better biologically than the adhesive resin and that pulpal repair was not compromised by the use of NaOCl. At 90 days, neither exposure to 0.9% saline, 5.25% NaOCl, or 2% chlorhexidine digluconate incapacitated healing after direct pulp capping with CH when examined in healthy human pulp tissue.[217] In an investigation that measured parameters of success in direct pulp capping in a carious field, 10% NaOCl was used without any marked affect on continued pulpal vitality and repair.[188] It is clearly evident that the use of ***NaOCl*** in concentrations of 5.25% to 8.0% for direct pulpal exposures **can be recommended** and is a relatively safe and practical method to predictably achieve hemostasis (Figure 27-13).[213,214,216–218]

Another effective hemostatic agent is MTAD (Biopure®, Tulsa/Dentsply, Tulsa, Oklahoma), an antimicrobial agent and irrigant introduced for smear layer removal during root canal treatment and retreatment.[219] The solution is a commixture of a detergent (Tween 80), tetracycline isomer (doxycycline), and acid (citric acid). The irrigant exhibits many favorable characteristics and may be a useful replacement for EDTA in conjunction with NaOCl. The agent cleans the dentin/pulp tissue interface without affecting the flexural strength and modulus of elasticity of dentin. It also shows a notable antimicrobial effect against some strains of *Enterococcus faecalis*.[220–224]

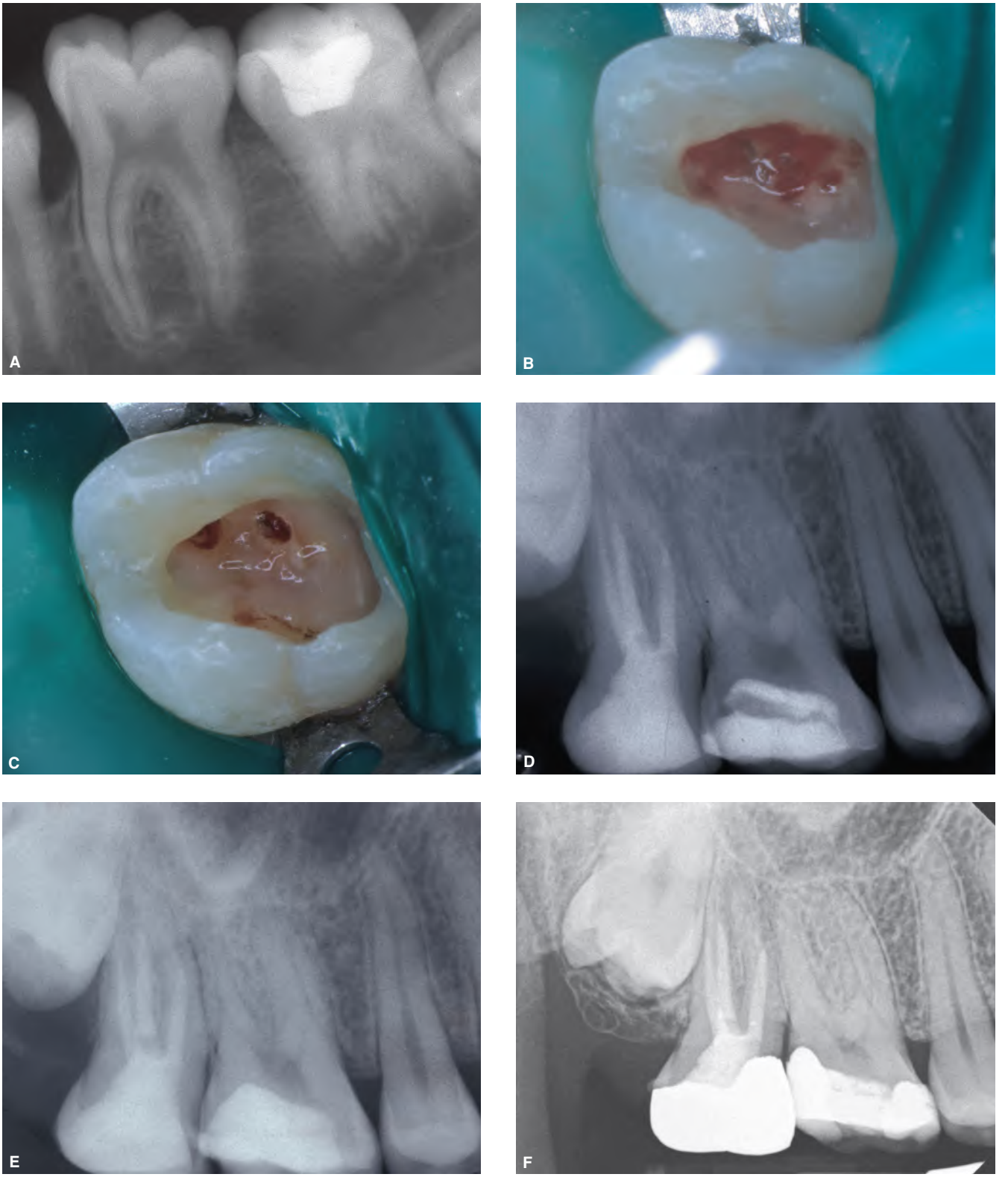

FIGURE 27-13 ***A.*** Symptomatic maxillary first molar in 14-year-old female. ***B.*** Hemorrhage visible after exposure of three pulp horns following caries removal. ***C.*** View of pulp exposures after five minutes of 5.25% NaOCl contact showing stable hemostasis. ***D.*** Radiograph showing MTA pulp capping material placed over entire pulpal roof and surrounding dentin with flat wet cotton pellet and Photocore® provisional restoration. ***E.*** Radiograph showing the affected tooth restored with bonded composite restoration. ***F.*** 17-year radiographic follow-up with second composite restoration. The molar was asymptomatic and tested within normal limits to carbon dioxide cold testing. (Reproduced with permission from Bogen and Chandler.[2])

Direct Pulp Capping

Treatment Recommendations

Recommended steps for direct pulp capping with MTA using a two-visit format:

1. Following diagnosis, the tooth has been identified as having either reversible pulpitis or a normal healthy pulp. After profound local anesthesia, the tooth is isolated with a dental dam, further sealed with an agent such as Oraseal® (Ultradent Products Inc., South Jordan, Utah), or comparable product, if required, and the crown disinfected with either chlorhexidine solution or NaOCl. Illumination and magnification (DOM) of the area is highly recommended. The undermined enamel is removed with a diamond or carbide bur and soft debris is removed with a spoon excavator.
2. After the carious dentin is air dried, a caries detector dye is applied for 10 seconds, and the tooth washed and dried. Caries removal is completed with slow speed #4-2 carbide round burs and spoon excavators until minimal (light pink) or no profound stained dentin is evident. The caries detector is again reapplied on air dried dentin for 10 seconds, and the process carefully repeated (possibly five to seven applications) until no or only a light pink staining is evident.
3. If a pulp exposure occurs during caries removal, the bleeding can be controlled by placement of a cotton pellet moistened with 5.25% to 8% NaOCl for 20 to 60 seconds and the staining and caries removal continued carefully around the exposure site until little or no staining is visible. Areas that are identified as reparative dentin with little or no staining should not be removed.
4. After caries removal, the exposure(s) should be hemorrhaging to some degree. A cotton pellet moistened with 5.25% to 8% NaOCl is placed directly against the exposure(s) for a contact time of 5 to 10 minutes. After hemostatsis is achieved, the cavity is gently rinsed with water and dried with air using a two-way syringe. However, if no exposure is encountered, it is still recommended to clean the dentin with the application of NaOCl for five minutes. When hemostasis is not achieved within 10 to 15 minutes, the diagnosis is changed to irreversible pulpitis and more aggressive treatment is indicated. Conversely, if bleeding is not evident after pulpal exposure, the tissue is most likely necrotic, and a miniature, partial or complete pulpotomy, or pulpectomy, must be initiated using a high speed round diamond bur. If during the course of caries removal, the entire pulpal roof or axial wall is removed, a pulpotomy must be considered.
5. The MTA or CSC is mixed according to the manufacturer's instructions (3:1, MTA:H_2O) and will have the consistency of wet sand. The cement is brought to the site in bulk with either a hand instrument (Glick or spoon excavator) or an MTA carrier gun. The MTA or CSC should be placed directly over the exposed pulp tissue and all surrounding dentin. The material is gently patted down with a small moist cotton pellet (or a dry pellet if the mixture is too wet) and when in place, it should be a minimum of 1.5 mm in thickness. If MTA is pushed inadvertently into the pulp chamber, it will not impact the outcome negatively. A circumferential region of dentin and enamel measuring approximately 1.5 mm, or more, should be cleared around the MTA with a small (2–3 mm) moist cotton pellet placed at the end of an explorer. This will allow an adequate area for the future bonded restoration to provide an effective seal.
6. A custom fabricated, flat (1–2 mm thickness), moist, cotton pellet, or gauze, is then placed over the entire area of the MTA. If the area involves a Class II preparation exposure including the axial wall, then the moist covering may require placement in two sections. If the patient is willing to return within four hours, a large moist cotton pellet, or gauze, can be placed with the patient instructed not to eat or chew during the interim, since this may dislodge the slow setting cement.
7. After cotton pellet placement, a strong interim restoration is provided, preferably an unbonded composite material that will facilitate removal during the second visit (e.g., Photocore®, Kuraray Co., LTD, Osaka, Japan). Unless amalgam is the designated restorative material, eugenol-based interim restorations such as IRM, or ZOE, that may interfere with adhesion and bond strengths of adhesive resins should be avoided.[225]
8. The second appointment can be scheduled 5 to 10 days after MTA or CSC placement. Before attaining profound anesthesia, the patient is questioned about sensitivity, mastication comfort, or presence of pain. The tooth is then cold tested (CO_2 ice or Endo-Ice®, Hygienic Corp., Akron, Ohio) to confirm continued normal vitality. After injection of a local anesthetic, the tooth is isolated as before. The interim material is removed with a high-speed diamond or carbide bur, under water spray. The cotton pellet, or gauze, is removed and the embedded cotton fibers removed with a spoon excavator, or similar hand instrument. Working with high magnification (DOM) is strongly recommended. The MTA or CSC is checked to ensure proper curing and a bonded (self-etching) composite restoration placed following the manufacturer's recommendations.
9. After the permanent restoration is placed, the occlusion is checked and adjusted as required. The patient is then recalled at 6 weeks and subjective symptomology and cold testing evaluated. If it appears that the procedure was successful, radiographic follow-up, cold testing, and subjective symptomology can be evaluated at 6 and 12 months. The patient, if compliant, should then be evaluated on a yearly basis.

One-Step Pulp Capping [115]

According to one manufacturer of MTA (ProRoot® MTA, Dentsply, Tulsa, Oklahoma), pulp capping can be completed

in one visit. In some situations, treatment of the immature permanent tooth can be difficult, especially in young patients with challenging medical or behavioral problems that may require treatment under sedation. When one step is indicated, the manufacturer recommends the following protocol:

1. Complete a cavity preparation outline under dental dam isolation, using high-speed burs under constant water cooling.
2. Excavate any carious tooth structure using a round bur at low speed or use hand instruments.
3. Rinse the cavity and exposure site(s) with 2.6% to 5% NaOCl. Heavy bleeding may be controlled with a cotton pellet moistened with NaOCl.
4. Prepare ProRoot® MTA according to listed instructions.
5. Apply a small amount of MTA over the exposure using a small ball applicator or similar device.
6. Remove the excess moisture at the site with a dry cotton pellet.
7. Apply a small amount of Dyract Flow™ flowable compomer (Dentsply International, York, Pennsylvania) (or an equivalent light-cured resin, glass-ionomer liner) to cover the MTA material, and light cure according to its instructions.
8. Etch the remaining cavity walls with 34% to 37% phosphoric acid gel for 15 seconds. Rinse thoroughly.
9. Dry the cavity gently, leaving the dentin moist, but not wet. Apply Prime and Bond NT material, or an equivalent bonding material. Cure according to its instructions.
10. Place TPH Spectrum (Dentsply/Caulk, Milford, Delaware) composite material or an equivalent composite resin to complete the restoration.
11. Assess the pulp sensibility (responsiveness to tests) every six months and evaluate the tooth radiographically every three to six months or as needed.

The authors recommend that caries removal be completed under magnification (DOM) with the aid of a caries detector dye (Step 2). Also, during MTA placement against the exposure site (Step 5), a larger bulk of MTA can be placed that includes the majority of surrounding dentin at a thickness greater than 1.5 mm, to ensure bacterial neutralization. With newly introduced fast setting CSC products, manufacturers recommendations must be followed. However, caries removal under DOM using caries detector dyes and NaOCl hemostasis will provide superior results. Unfortunately, these adjunctive procedures are often overlooked by many operators and instructional brochures published by manufacturers.

THE FINAL RESTORATION

The long-term maintenance of pulp vitality and continued normal function of the pulp capped/pulpotomized tooth is largely dependent on the quality and seal of the final restoration. Bacterial microleakage around restorations is directly associated with the degree of pulpal inflammation and the frequency varies according to the material used.[226] The pulp will have the highest probability for wound repair and survival if microleakage is minimized or eliminated.[227–229] It has even been proposed that cavity preparations that are unfilled and left open, are less harmful than leaking restorations.[230] The appropriate restorative material for each case must be carefully selected and the delivery executed with a high level of skill to provide the best likelihood of a predictable seal against microleakage. Since the delivery of adhesive restorative materials can be technique sensitive, manufacturer's guidelines must be followed and specific composite materials matched with their respective and recommended bonding resins.[231]

Restorative options for immature permanent teeth include full coverage restorations, composite resins, and bonded or unbonded amalgam restorations. The probability of pulp survival increases with conservative restorative treatment that best preserves the remaining healthy tooth structure.[194] The practice of placing stainless steel crowns on immature permanent teeth with adequate remaining tooth structure should be avoided. The age of the patient, the size and depth of the cavity preparation, and the choice of restorative material are all factors affecting repair mechanisms within pulp tissue.[232]

Although amalgam has proven to be a reliable material because it is inexpensive and relatively simple to place, it has disadvantages that include esthetic concerns and potential health risks for dental providers.[233–236] Amalgam restorations can also be associated with coronal infractions or cuspal fracture, particularly if cusps are not protected.[237,238] In structurally compromised immature teeth, the difference in the modulus of elasticity between amalgam (28–60 GPa) and dentin (12–18 GPa) can contribute to a greater risk of coronal and radicular fractures.[239–241] Also, it may not provide an impenetrable seal against microleakage and bacterial contamination.[242,243] However, amalgam restorations, when elected, may exhibit less microleakage if placed in conjunction with a bonding resin or resin-modified glass-ionomer liner.[227,244,245] Survival rates for composites are improving as material technology advances and tooth isolation requirements are acknowledged.[246–249] Even though amalgam restorations remain a reliable and predictable long-term treatment option, the clinical transition to adhesive systems is advancing as technology improves and amalgam use declines.

POST OPERATIVE FOLLOW-UP

Accurate predictors for measuring survival rates in vital pulp therapy include clinical recall and radiographic evaluation. Recall compliance of asymptomatic cases is generally more challenging than teeth that become irreversibly inflamed or progress to symptomatic apical periodontitis after treatment. Since the presence of recurrent caries, restoration failure, poor oral hygiene, or other conditions requiring attention can be identified during a recall appointment, the follow-ups after vital pulp therapy can be beneficial and provide a stronger impact. Unfortunately, some parents and guardians lacking basic oral health knowledge do not practice

preventive measures and may not appreciate the importance of follow-up care thus compromising recall rates.[250,251] Proper follow-up assessment is based on the common practice of the six-month recall. The recall period is related to the frequency of oral prophylaxis; this practice has recently been challenged.[252] It is now recommended that it be based on the patient's need and thus optimal recall rates should be established individually. Factors such as caries index, periodontal status, symptomology, and assessment of cranio-facial development in younger patients should be considered.[253]

An adequate time period for a tentative diagnosis of pulp survivability was determined to be 3 months in an investigation where direct pulp capping was completed with CH.[188] An observation period of 21 to 24 months was shown to establish the prognosis for long-term pulp survival in the same study. The two-visit protocol (described earlier in this chapter) allows clinicians the opportunity to examine the treated tooth at 5 to 10 days after initial treatment. If the procedure appears favorable at the second appointment, the provider can confidently schedule the future follow-up at six weeks, and then at 6 and 12 months if possible.[144]

Apexogenesis of the immature adult tooth is the essential objective in vital pulp therapy. Radiographic confirmation of root-end closure in ***immature permanent teeth*** treated with direct pulp capping, or pulpotomy procedures, is the most reliable indicator of successful treatment. The physiological development and formation of the root-end should advance uneventfully in healthy patients at a constant rate after the application of MTA or other CSC, whether direct pulp capping or pulpotomy was performed (Figure 27-14).[136,137,254–256]

The natural succession of tooth development in vital teeth should follow a predictable pattern when viewed chronologically and compared radiographically to contralateral untreated

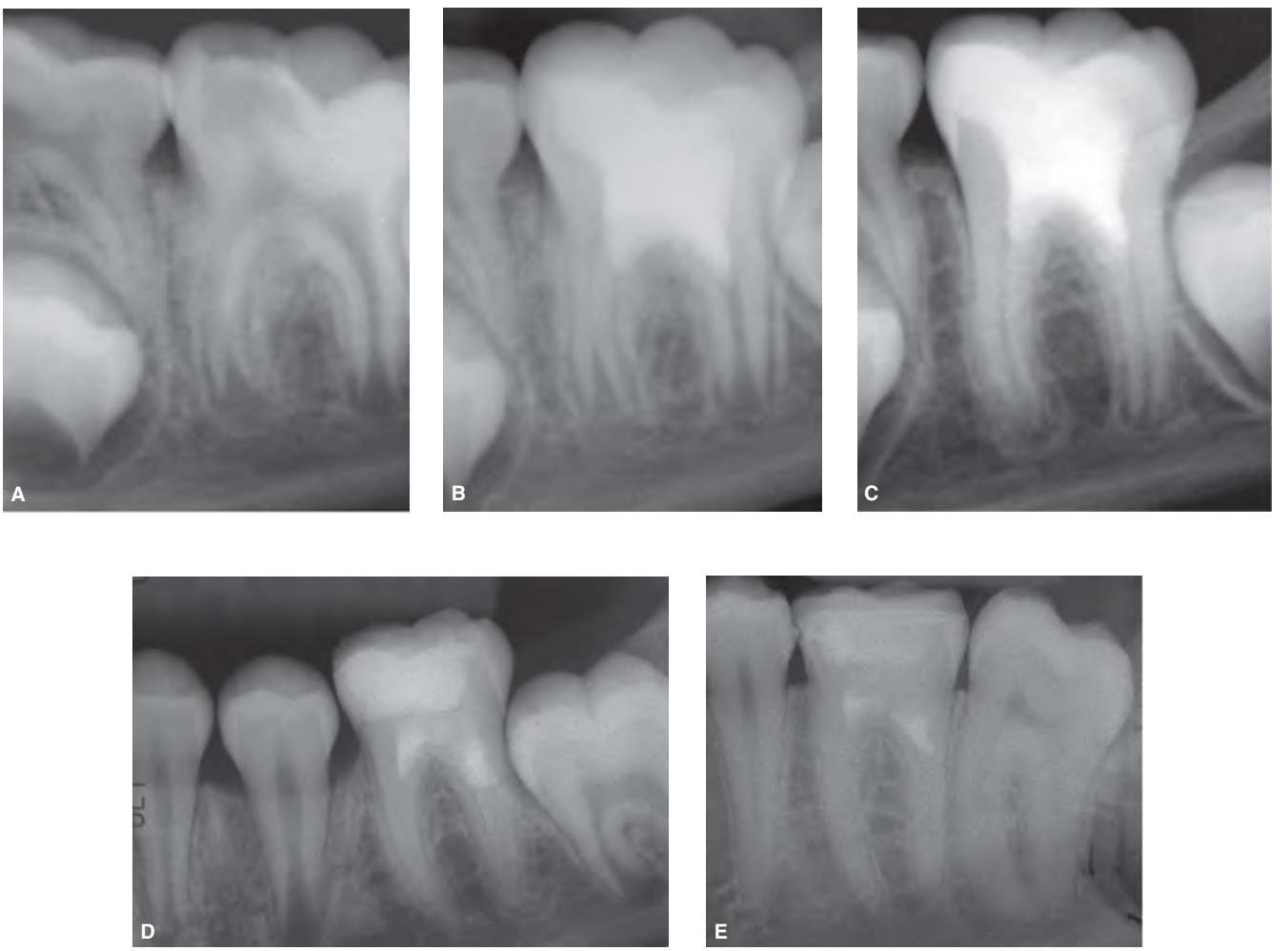

FIGURE 27-14 Seven-year-old patient presenting with moderate to severe pain associated with a dens evaginatus of the mandibular left first molar. ***A.*** Preoperative radiograph showing immature apices and presence of a coronal defect that was clinically filled with hyperplastic tissue. ***B.*** Postoperative radiograph after complete pulpotomy with MTA. The access cavity was sealed with glass ionomer cement and bonded composite. ***C.*** 15-month follow-up radiograph demonstrating continued root development and apical closure. ***D.*** Five-year recall radiograph showing completed root development. ***E.*** 12-year radiographic follow-up. The patient was asymptomatic and the molar in full function. Note, no cuspal coverage restoration was placed. (Courtesy of Dr. Mark Olesen, North Vancouver, BC, Canada.)

teeth.[257] Comparison of contralateral tooth development at the same time intervals, is therefore a superior way to assess the success of vital pulp therapy. Teeth with necrotic pulps and periapical pathosis that have sustained trauma have traditionally been treated with CH to stimulate apical barrier formation and augment root-end closure.[258] Variable times from 5 to 20 months can be required to complete barrier formation.[259]

Mineral Trioxide Aggregate and other CSCs are now widely accepted as replacements for CH in direct and indirect pulp capping, pulpotomy, and apexogenesis procedures and can be expected to promote apical maturation at coinciding time periods.[260] The dental pulp and surrounding tissue have an exceptional regenerative capability when a ***microbe free environment*** is implemented.[231,261,262] The recent introduction of these advanced bioactive materials that reliably promote pulp repair and healing have made a progressive and substantial contribution to our understanding and ability to protect and maintain the living pulp.

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Chapter 28

Management of Teeth with Immature Apices

Shahrokh Shabahang

ROOT APEX AND CORRECT LEVEL OF OBTURATION

Grove, in 1930, proposed that root canals should be filled to the cemento-dentinal junction (CDJ).[1] Based on his observations, the CDJ is on average 0.5 mm–0.75 mm from the apical foramen (Figure 28-1). In 1958, Kuttler[2] confirmed that the CDJ is the ideal terminus of a root canal filling, and that over-extension of the obturation material is not desirable because these materials constitute foreign bodies, that prevent deposition of new cementum. In his paper, he further cited that following extirpation of pulp tissue, the periodontal membrane that remains in the cemental (most apical) portion of the canal is capable of giving rise to new cementum. In examination of the clinical correlation of the root apex and its surrounding structures, Moodnik[3] concluded that the root apex must be viewed as a vital dynamic tissue that is capable of development, growth, and repair.

TOOTH DEVELOPMENT

Prior to manipulation of immature permanent teeth, an understanding of tooth development is paramount.[3–7] This holds true whether devising an accurate diagnosis and appropriate treatment plan in the clinic or developing new materials and techniques in laboratories. For more details, see Chapter 2, "Structure and function of the pulp–dentin complex."

During tooth development, the inner and outer dental epithelia fuse and form the cervical loop.[5] From this region root formation begins, initiated as an apical proliferation of the two fused epithelial structures forming a double layer of cells known as the Hertwig's epithelial root sheath (HERS). The sheath is similar to the inner enamel epithelium and acts as the stimulus and template for the differentiation of odontoblasts and formation of root dentin.[7] The HERS is located at the end of the developing root (Figure 28-2), with its cells lining the pulp space and it is responsible for increasing root length and closure of the apical foramen.[8] The root sheath determines the number, size, and shape of the roots and the future cementoenamel junction.[5] Fragmentation of the HERS allows the cells of the investing follicle to pass through and contact the newly formed dentin surface where the cells differentiate into cementoblasts and initiate cementum formation. In many teeth, a network of the fragmented root sheath persists in the periodontium in close proximity to the root after root development has been completed to form the epithelial cell rests of Malassez (ECRM).

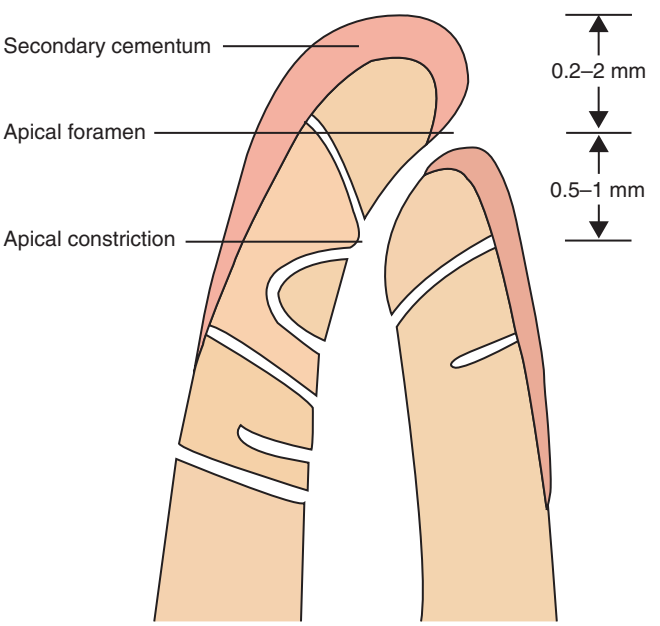

FIGURE 28-1 Illustration of apical root anatomy depicts location of the apical foramen, that is typically short of the root apex and exits on the lateral border of the root apex.

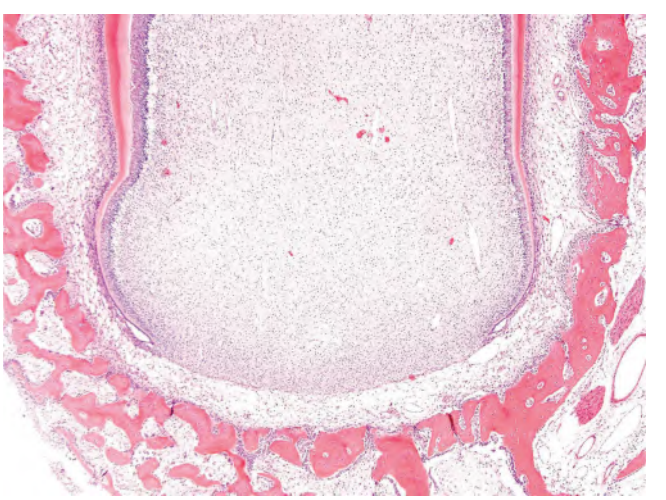

FIGURE 28-2 Photomicrograph of the apical segment of an immature root and its surrounding periradicular tissues. The inner and outer enamel epithelia join to form the HERS.

Facial injuries between the ages of 6 and 14 years have the potential for interrupting, altering, or arresting root development.[8] In general, trauma to the teeth can cause either the complete arrest of odontoblast function or accelerate their activity, resulting in pulp calcification (Figure 28-3).

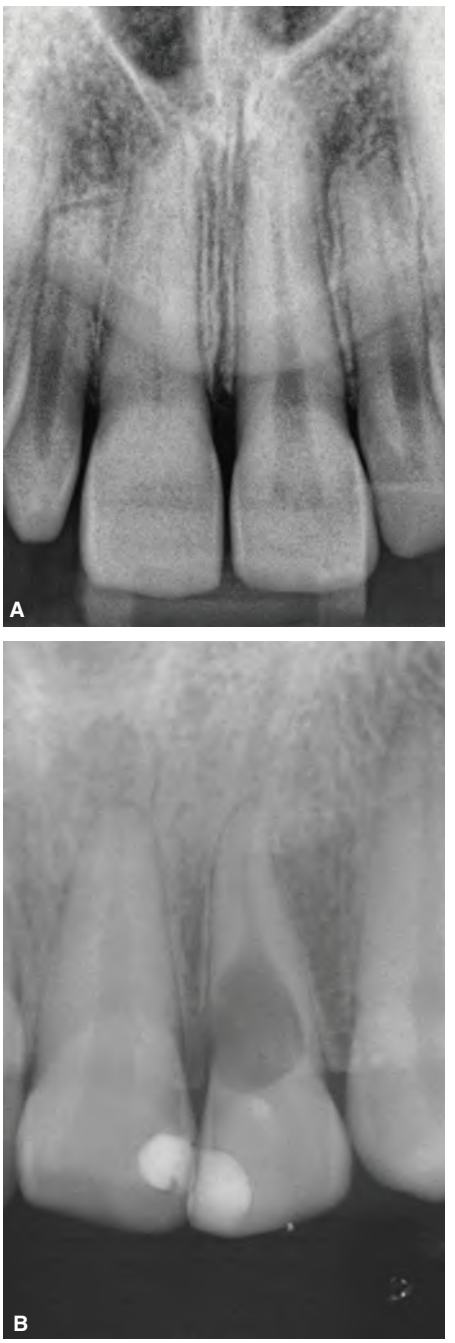

FIGURE 28-3 Radiographic images illustrating that, subsequent to traumatic injuries, the pulp may either increase dentin deposition or initiate dentinoclastic activity. ***A.*** The canal space in the maxillary right central incisor has undergone calcific metamorphosis due to an increase in dentin deposition in reaction to trauma sustained by the tooth. ***B.*** Large internal resorptive defect in the maxillary left central incisor, which is another possible scenario resulting from increased dentinoclastic activity due to trauma.

Complete destruction of the HERS can arrest any further lengthening of the root, but previously differentiated odontoblasts and cementoblasts can complete apical closure. HERS forms the outline for the root and apex, but without a vital pulp, root development cannot continue to completion.[9]

BONE AND DENTIN

Bone consists of three different components: (1) a mineral component, (2) collagen and (3) other organic components including growth factors. The growth factor component has bone morphogenetic activity and can be extracted after demineralization of bone.[10] Mechanisms that generate new bone formation include: Osteogenesis, the transfer of viable bone-producing cells within the graft to a new anatomic location; osteoconduction, creeping substitution or bony ingrowth; and osteoinduction, the stimulation of mesenchymal cells to differentiate into osteogenic cells that produce new bone.[11]

Implantation of devitalized decalcified bone, following hydrochloric acid treatment in extra-skeletal sites, stimulates host cells to differentiate and produce bone through a transient inflammatory reaction.[12] Differentiation of the osteoprogenitor cells is elicited by local alterations in cell metabolic cycles. Chondroprogenitor cells produce cartilage, that later becomes vascularized, to set up a second or delayed induction system for bone formation. Whether progenitor cells produce bone or cartilage is determined by the microenvironment or local factors. When implanted in experimental periodontal bony defects in dogs, decalcified allogeneic bone matrix results in enhanced bone formation.[13] Inoue and associates[14] evaluated the effects of demineralized dentin and bone matrix in rats. They reported that the implants induced formation of cartilage most rapidly and abundantly in muscle, and most slowly and in the smallest quantity in periodontal ligament.

Smith et al.[15] implanted dentin matrix components in exposed pulps in ferret teeth. Significant deposition of new extracellular matrix was observed at the pulp/dentin interface after 14 days. The dentin matrix expressed a local response as there was no deposition of reactionary dentin at any other sites in the teeth. In the absence of implanted proteins, there was no evidence of deposition of reactionary dentin beneath the cavities at any time.[15]

BONE MORPHOGENETIC PROTEINS

Bone morphogenetic proteins (BMPs) can be divided into subgroups based on the primary amino acid sequence in the mature region of the molecule. BMP-2 and BMP-4 are closely related proteins with 92% identity in the cysteine region.[10] BMP-5, BMP-6, BMP-7 (also known as osteogenic protein-1), and BMP-8 (osteogenic protein-2) form another subgroup with approximately 75% amino acid homology.[16] These two subgroups are interrelated by nearly 60% amino acid homology, while BMP-3 is in a subgroup of

its own. Additionally, a sub-group of BMP-related molecules known as growth and differentiation factors (GDF) have been identified.[16] Most BMPs that have been cloned share structural similarities to members of the transforming growth factor-β (TGF-β) superfamily.[17] TGF-β belongs to a family of disulfide-linked homodimers, which are involved in the migration, proliferation, and differentiation of mesenchymal cells during embryogenesis and tissue repair.[18,19]

To study the effect of BMP on reparative dentin formation, pulps of teeth in young dogs were amputated and 3 milligrams of crude BMP were implanted in the cavity on the surface of the amputated pulp.[20] Canine serum albumin was implanted in other teeth to serve as control. Pulp responses were categorized in four stages:

Stage 1 (Immune response): BMP evoked a cell mediated response within one week.

Stage 2 (Proliferated response): BMP was almost completely absorbed; spindle-shaped mesenchymal cells migrated and proliferated between the first and second week.

Stage 3 (Osteodentin formation): Between two and four weeks, in some parts of the cavity, especially adjacent to the dentinal walls and just beneath the cement-filling surface, osteodentinoblasts and/or osteodentinocytes differentiated to form osteodentin.

Stage 4 (Tubular dentin formation): Between four and eight weeks, odontoblasts differentiated to form tubular dentin. In the control sites containing serum albumin implants. The cavity was filled with pulp tissue much more slowly than in the experimental group.

On the basis of these results, the author concluded that BMP has desirable properties as a pulp-capping agent because it is completely absorbed and can produce a large amount of reparative dentin on the amputated pulp without affecting the radicular pulp.

Lianjia et al.[21] implanted 200 μg of BMP directly onto the surface of amputated pulps in the premolar teeth of adult dogs and compared these findings with the control group that was treated with the same amount of calcium hydroxide. Their results showed that in the bell stage of tooth development, the outer and inner enamel cells and differentiated odontoblasts demonstrate a positive reaction to BMP-monoclonal antibodies. Incorporation of [^{3}H]thymidine into pulp cell nuclei and alkaline phosphatase activity in the pulp cell cytoplasm were much higher in the BMP group when compared with the control group. These results suggest that BMP not only stimulates DNA synthesis of dental pulp cells, but also promotes calcification. After one week, all pulps capped with BMP showed minimal signs of inflammation and by the second week, the pulp tissues were free of inflammation. Starting at week three, dentin formation was induced and the dentin bridge was complete and had begun to calcify. In the control group, only some osteodentin formation was present at the fourth postsurgical week and a dentin bridge did not form.

OP-1 may recruit mononuclear phagocytes and stimulate proliferation of mesenchymal cells that differentiate into osteogenic cells that ultimately lead to ectopic bone formation.[22] Transcripts of *bmp-2* and *bmp-7* have been identified at bud and cap stages in the area of the enamel knot in the dental epithelium. Postlewaite et al.[23] compared the ability of recombinant human OP-1 (rhOP-1) and recombinant human TGF-β1 (rhTGF-β1) to induce migration of fibroblasts and to affect a fibrogenic phenotype in these cells *in vitro*. The results demonstrated that although both are potent chemoattractants for neutrophils, monocytes and fibroblasts, only TGF-β1 upregulates fibroblast functions associated with a fibrogenic phenotype (i.e., stimulation or proliferation and synthesis of collagen). In contrast to rhTGF-β1, rhOP-1 neither affected the rate of mitogenesis of fibroblasts nor enhanced synthesis of collagen or hyaluronan by fibroblasts (functions that may be important in soft tissue repair). Neither protein stimulated the production of prostaglandin E2 (PG-E2).

The extent of osteogenic activity was histological examined in implants containing 25 mg of rat collagen carrier with varying amounts of OP-1.[24] Alkaline phosphatase activity and calcium content of the implant have been used as biochemical markers for osteogenesis (calcium content of the implant is directly proportional to the total amount of bone formation). Results indicate that the carrier alone does not induce bone, whereas implants containing both carrier and OP-1 induce endochondral bone formation in a dose-dependent manner. Furthermore, OP-1 samples show evidence of hematopoietic bone marrow recruitment. Histological evaluation of hOP-1 implants in rats has similarly shown that as little as 5–10 ng of the protein in 25 mg of matrix carrier is sufficient to induce endochondral bone formation in a dose-dependent manner.[22] At a concentration of 1 μg/25 mg of matrix carrier (that is the level at which rhOP-1's activity plateaus), the bone-forming activity of the implant is four times greater than the activity elicited by intact demineralized bone matrix.

OP-1 has also been used to induce bone formation in close apposition to titanium dental implants placed in tooth sockets of monkeys. Rutherford et al.[25] used a bovine protein preparation in combination with monkey bone collagen carrier. They found that at the three-week observation period, OP-1 induces bone formation in close apposition to at least some portion of the titanium surface, while similar findings are not observed in the absence of OP-1. Substantially more new bone formation is also evident in sockets filled with OP-1 implants compared to those filled with the collagen matrix alone or those left empty.

In a comparative study with calcium hydroxide, OP-1 in a collagen carrier was used as a pulp-capping agent in the premolars of adult monkeys using 1.5, 3.0, and 6.0 mg of the material per tooth.[26] Calcium hydroxide was used in a setting paste (Dycal). In a third group, teeth were filled with the collagen carrier alone, while a fourth group did not receive any material. The animals were sacrificed six weeks after the surgical procedures and the samples were histologically evaluated. Reparative dentin formation was present in all teeth treated with hOP-1/collagen carrier in which the material had been retained in the teeth for the duration of the six-week healing phase. The 6.0-mg group induced significantly more reparative dentin. Three of the five pulps treated with calcium hydroxide contained partial dentin

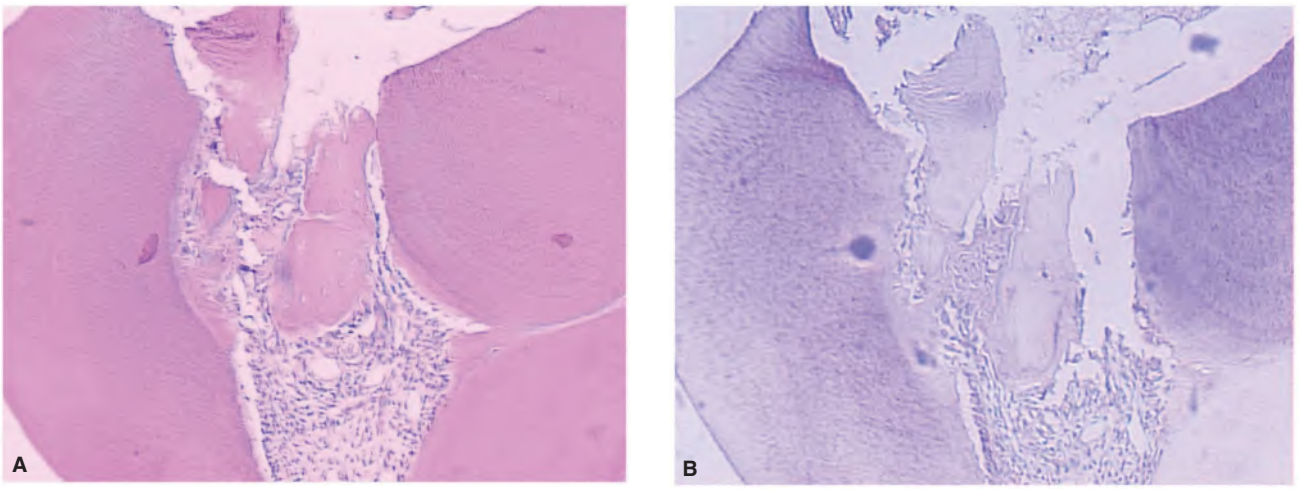

FIGURE 28-4 Photomicrographs showing ectopic bone formation in the pulp chamber of a rat molar following pulp capping with OP-1. ***A.*** H&E staining shows bony morphology with lacunae. ***B.*** Represents an immunohistochemical section stained with antibody against dentin sialoprotein (DSP), that is found approximately 200-fold more concentrated in dentin than in bone. The figure shows heavy staining with antibody to DSP in the dentin area and lighter staining in the ectopic bone produced in the pulp.

bridges, while no dentin bridge formation was noted in the collagen carrier and sham control groups. In the experimental groups, the pulps appeared normal with an intact odontoblastic layer. In the calcium hydroxide group, the dentin bridges formed at the expense of the pulp chamber. Furthermore, calcium hydroxide remained in the tooth superficial to the dentin bridge while the hOP-1/collagen was resorbed and replaced with a mineralized fibrous connective tissue. Conflicting results have been reported by other investigators.[27] In their study, Andelin et al. found ectopic bone formation rather than dentin (Figure 28-4). This finding is, in fact, consistent with Sampath's findings with ectopic bone formation observed when OP-1 was subcutaneously implanted in rat dermal tissues.[24]

In 1999, Shabahang and coworkers examined whether OP-1 has superior abilities to calcium hydroxide in induction of root-end formation.[28] Immature premolars in dogs were infected to allow periapical lesion formation. All root canals were debrided using sodium hypochlorite as an irrigant and then dressed with calcium hydroxide for one week to disinfect the canals.[29] The canals were then filled with calcium hydroxide, OP-1 in a collagen carrier, collagen carrier alone or MTA. At the end of the 12-week observation period, OP-1 showed similar results to calcium hydroxide with 5 of 13 roots in each group demonstrating complete barrier formation at the root-end while the remaining eight roots remained open. When a barrier was formed, the mean thickness was significantly greater in the OP-1 group compared to the calcium hydroxide group (Figure 28-5). Interestingly, when the collagen carrier alone was used, none of the 11 evaluable samples showed barrier formation (Figure 28-6). This finding suggests that the collagen carrier may inhibit root-end induction or that root canal disinfection alone may not be adequate and an active material is required to aid in induction of root-end closure.

To induce root-end formation mesenchymal cells must be stimulated to differentiate into odontoblasts and cementoblasts

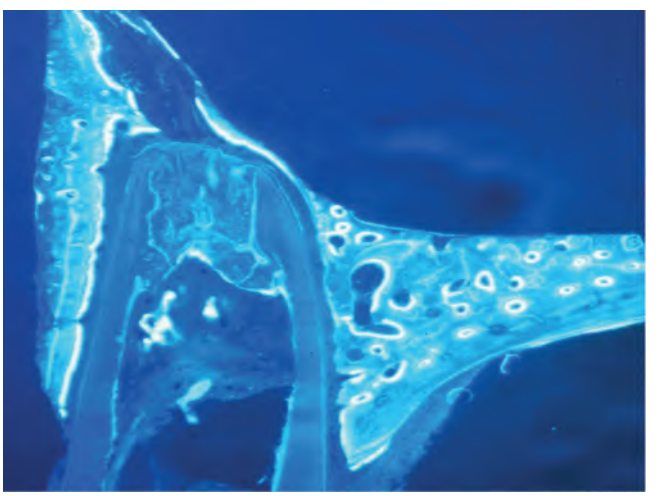

FIGURE 28-5 A fluorescent photomicrograph of a specimen showing formation of a thick layer of mineralized tissues in the apical region of an immature tooth that was treated with OP-1.

in order to produce dentin and cementum, respectively. This inductive process will require proper signaling to ensure that the pluripotent cells differentiate into the appropriate cells to produce the desired tissues.

DIAGNOSIS AND CASE SELECTION

When the pulp is vital and the apex is not formed, it is imperative to make every attempt to maintain the vitality of the pulp in order for the apex to complete its formation.[9] Only odontoblasts, the specialized cells in the pulp, are capable of producing dentin. In addition to interruption of root apex formation, in the absence of a vital pulp, thickening of dentin will not occur along the entire root length. Thin dentin in the cervical region teeth is more susceptible to fracture if subsequent traumatic injury occurs (Figure 28-7).

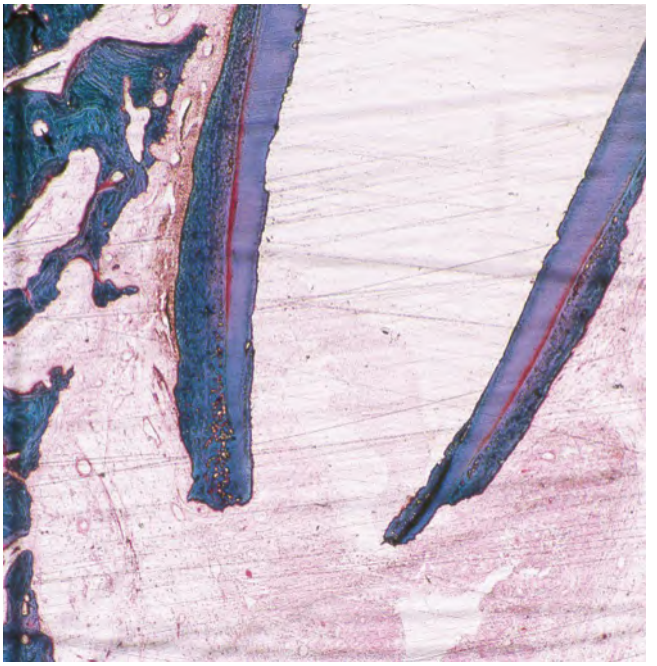

FIGURE 28-6 Specimen stained with Masson's Trichrome showing absence of apical closure in an immature tooth that was treated with collagen carrier alone. Figure illustrates cementum covering the inner and outer surfaces of dentin walls.

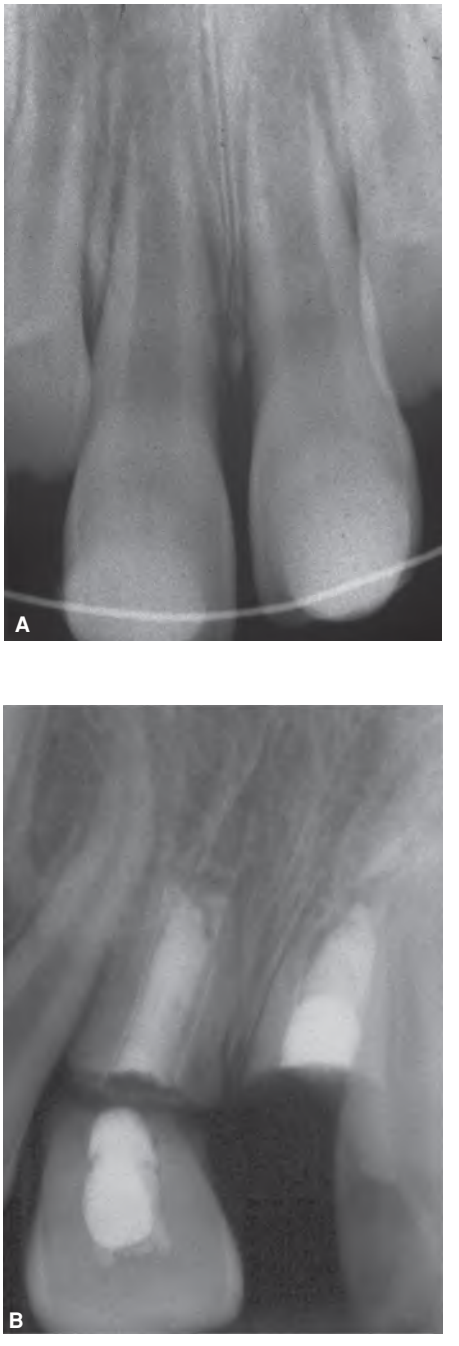

FIGURE 28-7 Radiographs show maxillary central incisors that were subjected to a traumatic incident. ***A.*** Teeth are splinted shortly after injury. ***B.*** Eighteen-month follow-up radiograph shows cervical root fractures in both teeth subsequent to apexification. (Courtesy of Dr. Leif K. Bakland, Loma Linda, CA, U.S.A.)

Pulp tissues should be removed only when irreversibly inflamed or necrotic. In young patients, the pulp has been typically exposed to less irritation and restorative treatment, is larger and more cellular, and has a higher likelihood to recover from injury. Cvek et al.[30] found that teeth that have been subjected to crown fractures and pulp exposure maintain a vital pulp for up to seven days post injury. Inflammation in these fractured teeth is limited to the surface 2 mm of the pulp just beneath the exposure level.

Accurate diagnosis is imperative prior to determining the treatment plan for teeth with underdeveloped roots. For this, careful assessment of pulp status is required. Radiographs are used to assess the maturity of the developing root and presence or absence of periradicular pathoses. Clinical evaluation should be based on history and clinical testing. Less reliable pulp testing data pose a special challenge in children. Verbal communication may be impacted by the young patient's linguistic development and apprehension.[31–33] Assessment of the true histologic status of the pulp is complicated by unpredictable and subjective symptomology reported by the patient.[34] The response to pulp testing is also less predictable than in adults and therefore less accurate. The most reliable test may be cold testing while[35,36] electric pulp testing in teeth with immature apices can result in less accurate results.[37] While assessment of nerve impulses is less reliable, determination of pulpal blood flow may be a more accurate means to determine the condition and prognosis of an injured young pulp in an underdeveloped tooth. Strobl et al.[38] used laser dopplers to evaluate changes in pulpal blood flow (PBF) in teeth that were subjected to various types of traumatic injuries. They were able to show that, immediately after removal of the stabilizing splint, while lateral and extrusive subluxation injuries do not significantly impact PBF, intrusive injuries consistently lead to diminished PBF, and ultimately, pulp necrosis (Figure 28-8). Thus, teeth that have a history of intrusive injuries will most likely require endodontic therapy due to a high incidence of pulp necrosis. A potential shortcoming with laser

Doppler measurement of PBF is interference from the blood circulation in the gingival tissues.[39] For a more accurate measurement, placement of dental dam may be indicated to block out any interference from gingival tissues.

The flow chart in Figure 28-9 can serve as a guide to aid in the decision-making process when treating permanent teeth with incomplete root development. As the chart shows, in immature teeth with vital pulps, pulp vitality must be preserved. If the pulp is irreversibly inflamed or necrotic, the decision to perform conventional root canal treatment or a root-end closure procedure is based on the level of apical root development.

Whilst pulp testing is important to obtain an accurate diagnosis of the condition of the pulp, determination of the degree of root development is primarily achieved radiographically. Conventional 2-dimensional periapical radiographs have significant limitations because they primarily illustrate the mesio-distal view of the root. In developing roots, the buccal-lingual dimension of the root canal is typically greater than the mesio-distal dimension (Figure 28-10). Thus, while a periapical view of an incisor may show a closed apex in the

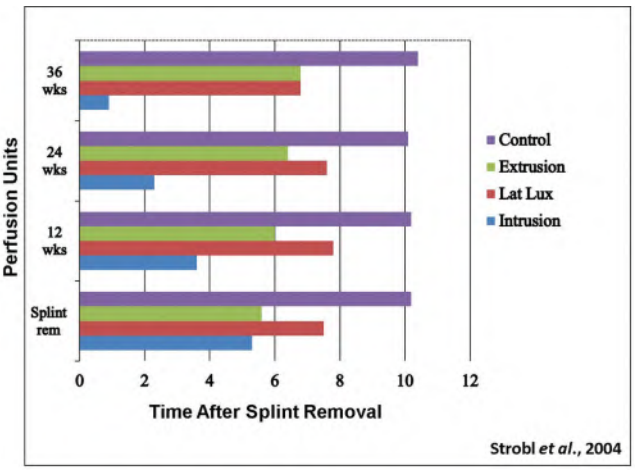

FIGURE 28-8 Data from Strobl et al.[38] show that intrusive injuries result in the greatest amount of damage to teeth. While pulps in teeth that sustain lateral luxation and extrusive injuries are associated with minimal changes in pulpal blood flow (PBF), intrusive injuries result in rapid decrease in PBF.

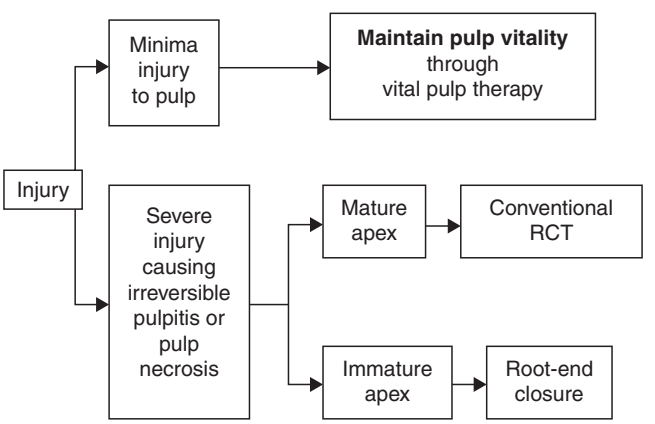

FIGURE 28-9 Decision-making for permanent, immature teeth. Algorithm to aid in the decision making process for immature permanent teeth that sustain injuries affecting the pulp.

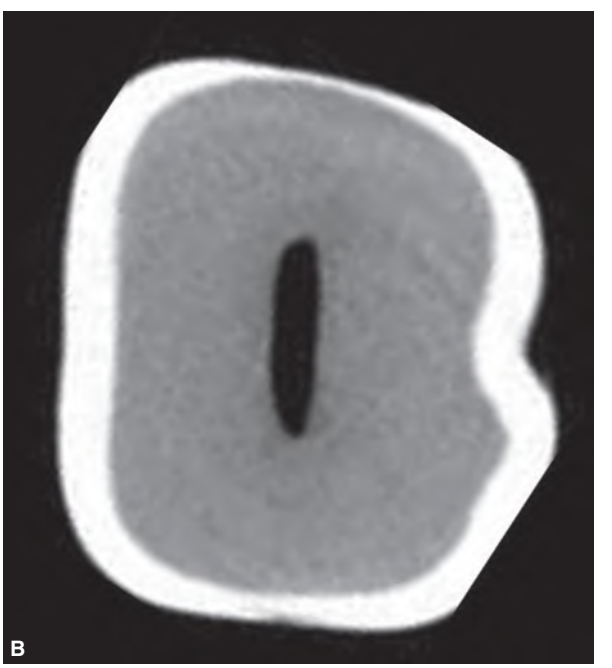

FIGURE 28-10 Illustrations of the apical appearance of immature teeth showing a wider diameter in the bucco-lingual dimension compared to the mesio-distal dimension. ***A.*** Photograph of the open apex of a molar. (Courtesy of Dr. Mahmoud Torabinejad, Loma Linda, CA, U.S.A.) ***B.*** Electron microscopic view of an open apex clearly showing a wider bucco-lingual width compared to its mesio-distal dimension.

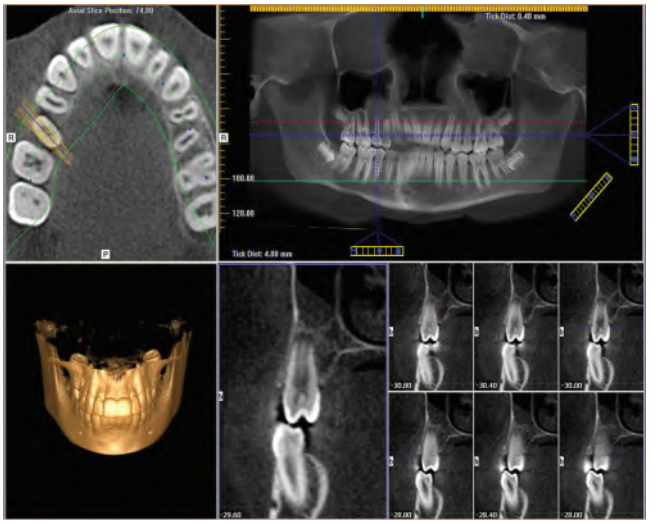

FIGURE 28-11 3-D imaging of a maxillary second premolar clearly shows presence of an open apex from lateral sections through the long axis of the tooth. (Courtesy of Dr. Mahmoud Torabinejad, Loma Linda, CA, U.S.A.)

mesio-distal dimension, the obscured bucco-lingual angle may be open. A root with apparently parallel walls mesio-distally may have divergent walls and greater width bucco-lingually; a canal with tapering walls mesio-distally tends to have parallel walls and greater width bucco-lingually. This developmental pattern can be misleading with respect to convergence and divergence. Since the bucco-lingual aspect of the root canal is the last dimension to become convergent as the root develops, a radiograph may be showing an apically convergent root canal, that in the bucco-lingual plane is divergent.[40] A more complete and accurate visualization of anatomic structures is now possible with the use of 3-dimensional CT imaging.[41] Figure 28-11 demonstrates the utility of 3-dimensional radiographs for a full view of a premolar with open apices.

INFECTION CONTROL

Immature permanent teeth present unique challenges during conventional root canal treatment not only because of a blunderbuss root apex, but also because of thin and fragile dentin walls (Figure 28-12). In order to maintain maximum root dentin, debridement should be achieved primarily through chemical means to remove any remaining pulp remnants and for disinfection. Minimal mechanical instrumentation is required because the canals are already wide. An added difficulty is determination of accurate root length measurement. Accurate canal length measurement is required to ensure complete canal debridement without over-instrumentation and over-extension of the obturation material. Extrusion of foreign materials might damage the periapical tissues including the remaining and vital HERS. Electronic apex locators are not accurate in immature teeth or teeth with wide open apices and radiographic determination of root length is recommended (Figure 28-13).[42,43]

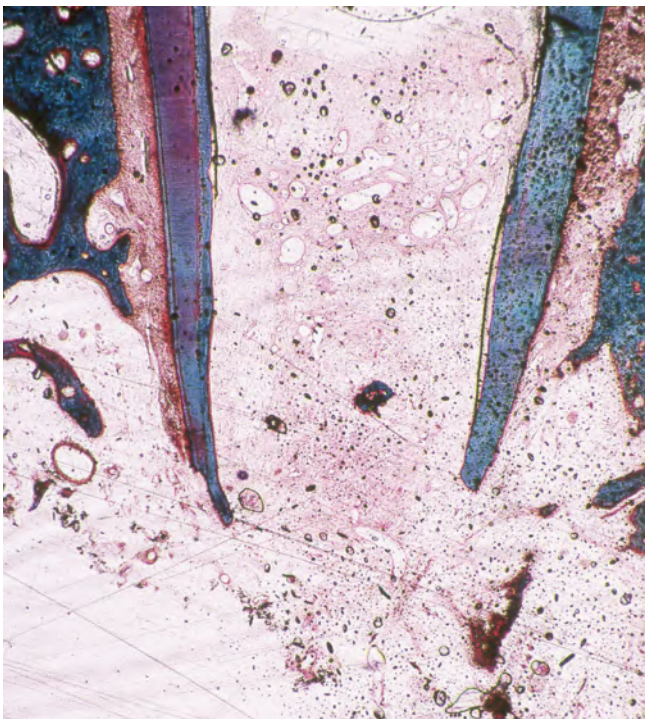

FIGURE 28-12 Ground section of an immature tooth stained with Masson's Trichrome showing thin and fragile root dentin.

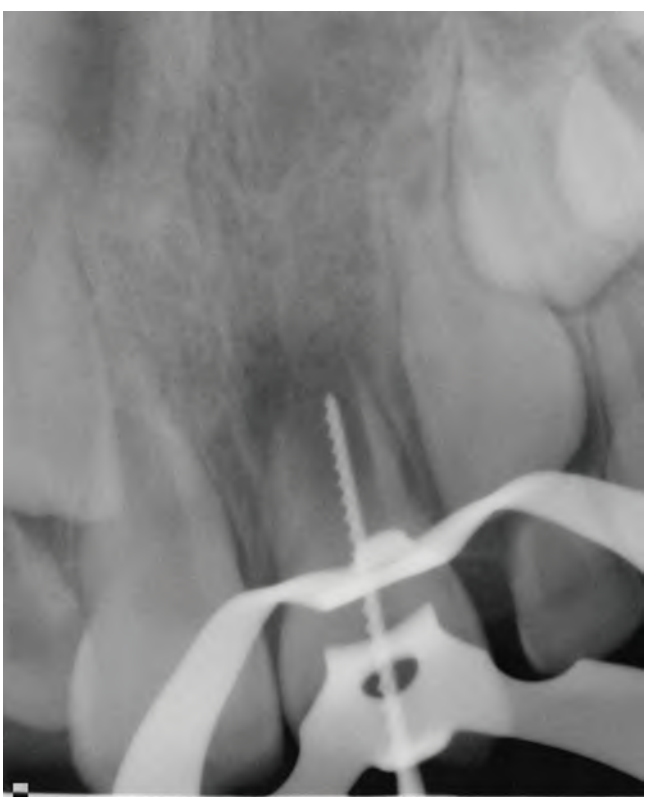

FIGURE 28-13 Radiograph of a maxillary left central incisor with an endodontic file illustrating radiographic root length determination in a tooth with a wide open apex. (Courtesy of Dr. Atosa Mehrfar, Loma Linda, CA, U.S.A.)

Sodium hypochlorite (NaOCl) and calcium hydroxide have tissue dissolving and antimicrobial properties.[44–53] NaOCl possesses more potent activity and exerts its activity during chemo-mechanical debridement. Calcium hydroxide, on the other hand, requires increased exposure times. Obturation of the canal space with calcium hydroxide allows disinfection along with dissolution and removal of pulp remnants.[29,50] While these properties of NaOCl and calcium hydroxide are required and important for canal debridement, neither may be effective in completely disinfecting the root canal system.[53–56] NaOCl can be readily inactivated by heat, light, and exposure to air.[57,58] It is most effective when mixed into solution just before use.[59]

Increased exposure times to these chemicals may also have detrimental effects on dentin structure. Machnick et al[60] showed that when dentin is exposed to NaOCl for periods exceeding 20 minutes, structural changes occur that may ultimately result in weakening of the root dentin. Similarly, exposure to calcium hydroxide for periods exceeding one month may result in structural changes in dentin that leading to significantly higher susceptibility to root fracture.[61–68] This finding may be especially significant in immature permanent teeth that have thin dentin walls in the cervical region of the tooth.

Due to these shortcomings, investigators have searched for more effective irrigants and medicaments to disinfect root canals. Use of antibiotics for root canal disinfection is not a new concept in endodontics. Ball,[69] in 1964, used antibiotic pastes for root canal disinfection. Das[70] used oxytetracycline HCl ointment after root canal debridement and found that disinfection of the root canal system can successfully induce root-end closure in a tooth with an open apex.

More recently, there has been renewed interest in adding antibiotics to root canal debridement protocols. BioPure MTAD was introduced in 2003 as a final root canal rinse for smear layer removal and disinfection. Torabinejad et al.[53,54,71–73] published a series of papers to report findings from systematic studies that were designed to demonstrate the advantages of a mixture of doxycycline, citric acid, and detergent over NaOCl and other commonly used root canal irrigants. The benefits presented include safe removal of smear layer (Figure 28-14) without damage to the underlying dentin[71,72]; antimicrobial activity including contact killing of resistant strains[53,54]; and substantivity.[73]

Iwaya and associates[74] reported the effectiveness of a double antibiotic paste to disinfect a premolar with a necrotic pulp that exhibited a large periradicular lesion. Three years later, Banchs and Trope[75] published a similar case. In their studies, Trope's group recommended the use of a triple antibiotic paste composed of metronidazole, ciprofloxacin, and minocycline. This antibiotic combination has proven effective in disinfecting the root canal system *in vitro*.[76,77] Animal experimentation has confirmed that significantly better disinfection can be achieved when root canals are disinfected with the triple antibiotic paste compared with biomechanical debridement using 1.25% NaOCl.[78] One potential drawback to the use of this mixture is staining of the tooth structure caused by longer exposure times to minocycline. For this reason, some have recommended omission of minocycline and use of a double antibiotic mix.[79,80]

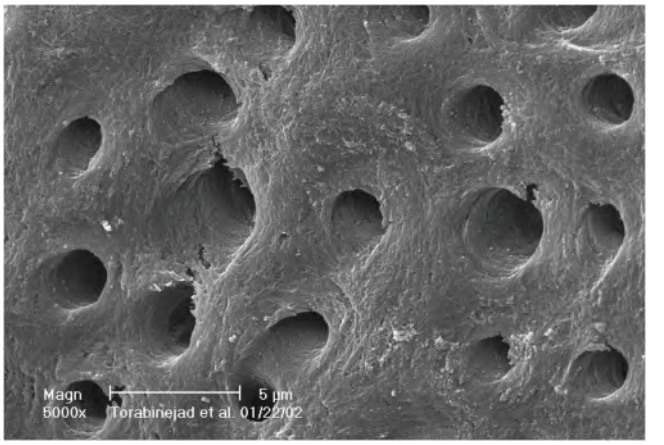

FIGURE 28-14 Scanning electron photomicrograph of root dentin after removal of smear layer with BioPure MTAD. The image shows complete removal of the smear layer without damage to the underlying dentin. The open dentinal tubules are clearly open and free of debris.

Another important factor to consider when selecting an appropriate root canal disinfectant for root-end induction procedures is their potential impact on viable cells (including stem cells of the apical papilla) in the periapical tissues. The double and triple antibiotic pastes can significantly reduce the survival of these cells in contrast to calcium hydroxide.[81] In a similar study, unlike NaOCl and EDTA supported cell survival, 2% chlorhexidine did not support survival of stem cells derived from apical papilla.[82]

Complete disinfection of the infected root canal system is a critical step during root-end induction procedures. Regardless of the protocol employed to induce apical closure, remaining bacteria in the root canal system will lead to continued irritation that can interfere with root-end development.

APEXIFICATION

In young teeth with incomplete root-end formation, severely inflamed or necrotic pulp tissues must be removed and the root canal system must be debrided in the conventional manner.[5] The young pulpless tooth, frequently, has thin and fragile walls making it difficult to adequately clean and to obtain the necessary apical seal.[83] Completion of endodontic therapy should therefore be delayed until root-end closure has been completed by apexification.

Torneck and associates[84] examined the effect of injury and oral contamination on the dental pulp of monkeys. Pulp injuries were induced in incisors of young *Macaca irus* monkeys with incompletely formed roots and observed for 7–95 days. Periapical radiolucencies were visible as early as 14 days following injury. As early as 28 days postoperatively, varying

degrees of inflammatory cell infiltration were visible in the residual portion of the apical pulp. At the end of the observation period (95 days), the specimens displayed post-operative root formation with some attempt at apical closure. The apical closure was a result of deposition of tubular and cellular dentin as well as cementum along the inner walls of the root canal. This phenomenon was attributed to the potential for portions of the dental pulp, including the peripheral layer of odontoblasts, to remain vital and active despite the presence of severe pulpal and periapical pathoses.

Some investigators believe that continuation of root apex development in nonvital teeth may be resumed by removal of the nonvital tissue and control of infection alone. In his 1980 publication, Das[70] reported a case of a 12-year-old child who was seen two years after a traumatic injury to a maxillary central incisor. The root canal was debrided, irrigated with sodium hypochlorite, and medicated with oxytetracycline hydrochloride ointment. Two months following initial treatment, the patient presented with resolution of symptoms. Continued root development had ensued.

Cameron[85] reported two cases in which he attributed the continuation of root-end closure to infection control using ultrasonically activated sodium hypochlorite. Calcium hydroxide was also used in these teeth as intra-canal dressing.

Lieberman and Trowbridge[86] reported a case in which apical closure of nonvital permanent incisors was observed in the absence of intervention. An 11-year-old patient had presented with maxillary central incisors that had been traumatized four years previously without any treatment. Teeth were examined and determined to be nonvital with radiographic evidence of large root canal spaces and periapical lesions. Radiographic evaluation also revealed closed apices in both incisors. The incisors were extracted due to severe malocclusion and general restorative difficulties. Histological examination indicated that the mineralized tissue that had formed at the apices of the teeth was composed of atubular dentin and cementum. The authors concluded that hard tissue formation occurred in the presence of vital tissues at the apices following which the pulps became inflamed and eventually underwent necrosis.

Various procedures and medicaments have been recommended for apexification including Tricresol and Formalin,[87] antibiotic pastes,[69] tricalcium phosphate,[88] and calcium hydroxide. Calcium hydroxide has been used in different mixes such as camphorated parachlorophenol (CMCP),[83,84,89–91] iodoform,[92] water,[93,94] local anesthetic solution, isotonic saline solution, glycerol, and other liquids.[40,95–98]

Ham[91] and associates evaluated the apical closure of immature pulpless teeth following experimentally induced blood clots. Seventeen teeth in three young monkeys were subjected to pulp exposures, contaminated with saliva and sealed with zinc phosphate cement. Radiographs were taken to confirm the presence of periradicular lesions. Teeth were then instrumented and medicated with CMCP and beechwood creosote. Eight samples were filled with calcium hydroxide mixed with CMCP and the remaining nine were treated with induced blood clots. The coronal halves of the latter group were filled with gutta-percha and sealer. None of the teeth in either the calcium hydroxide or blood clot group demonstrated complete bridging of the apex with calcified hard tissue.

Nevins[99] reported a case of a pulpless maxillary lateral incisor with an open apex in an eight-year-old boy. The young patient had developed thermal, percussion, and palpation sensitivity following traumatic intrusion of the incisor. The tooth underwent an apexification procedure using a mixture of collagen-calcium phosphate gel. The six-month postoperative radiograph showed continued root formation that was similar to the unaffected contra-lateral tooth. In a follow-up study, Nevins[100] concluded that the collagen-calcium phosphate gel provides a suitable medium for hard tissue formation. In a conflicting study, Citrome and associates[97] found severe inflammatory lesions with no evidence of apexification or cementogenesis in two 5-month-old dogs whose premolars were treated with collagen-calcium phosphate gel. In contrast, five of seven teeth treated with calcium hydroxide showed no evidence of inflammation and all teeth had apexification and apical cementogenesis. The hard tissue bridge was identified as cementum or amorphous calcified matter.

Regardless of the vehicle used, calcium hydroxide has historically been the material of choice for apexification procedures. Numerous studies have reported successful induction of apical closure or the formation of a hard tissue barrier against which gutta-percha and sealer may be condensed. In 1966, Frank[83] proposed placement of a thick paste of calcium hydroxide and CMCP and recommended follow-up evaluations at three- to six-month intervals until closure of the apex. He also recommended confirmation of a calcified barrier radiographically or clinically with an endodontic instrument (Figure 28-15). Frank further proposed that with

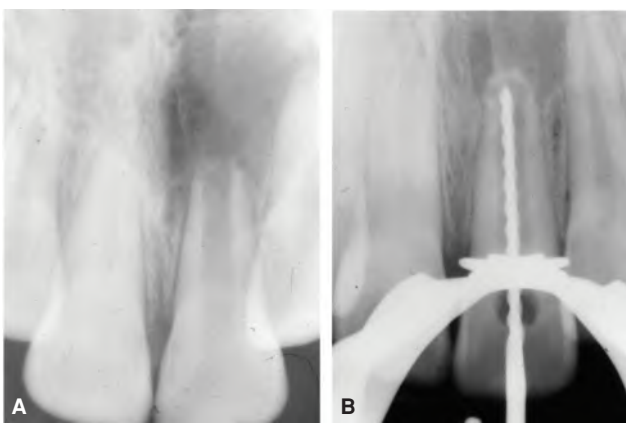

FIGURE 28-15 Radiographic images of a maxillary left central incisor in an 8-year-old patient with a history of trauma. ***A.*** initial radiograph shows an open apex and a large periapical lesion. Calcium hydroxide was replaced two to three times over a six-month period. ***B.*** Radiograph obtained at follow-up shows formation of an apical barrier that is visible radiographically and detectable with an endodontic instrument. (Courtesy of Dr. Leif K. Bakland, Loma Linda, CA, U.S.A.)

this procedure, the clinical results might vary. Outcomes may include any of the following: (1) An apex that closes with a definite though minimal recession of the root canal (the apical aspect continues to develop with a seemingly obliterated apex); (2) the obliterated apex may develop without any change in the root canal space; (3) there may be no radiographic evidence of continued root and apex development, however, an instrument inserted into the canal encounters a definite stop that would allow for obturation of the canal space; or (4) a calcific bridge may form just coronal to the apex that can be visualized radiographically.

Feiglin[101] similarly classified apex formation following apexification procedures with calcium hydroxide and listed them as follows: (1) The apex appears bridged over with hard tissue that is assumed to be osteocementum or osteodentin; (2) a perfectly normal apex is formed from the point of trauma to the apex of the tooth; (3–4) variable amounts of vital tissue remain above the point of trauma, that allow the root canal to heal or bridge over, and the apex to continue forming and to calcify; and (5) apex appears lifted off during tooth movement.

Dylewski[89] evaluated histologically the efficacy of calcium hydroxide-CMCP paste in incisor teeth of a young rhesus monkey with artificially induced open apices. In three of the teeth, instrumentation and obturation were limited to within the canal, and in the remaining teeth, the files were manipulated 1–5 mm beyond the apex, in an attempt to disrupt the epithelial diaphragm. His findings showed that instead of continued normal root development, a state of repair is observable at the apices characterized by proliferation of connective tissue. The connective tissue differentiates into a calcified material identified as osteodentin. Presumably, the bridging that occurs across the apex is not via the formation of typical dentin from odontoblastic activity but rather from the proliferation of connective tissues at the apical area. In this report, the HERS was not detectable in any of the sections and complete root-end closure could not be demonstrated in any of the teeth studied.

Harrison and Rakusin[102] also noted the presence of a cellular, atubular cementum bridge at the root apex during histological examination of a maxillary central incisor treated with calcium hydroxide (Figure 28-16). In their fourth paper in a series of publications, Torneck[84] et al. used a thick paste of calcium hydroxide-CMCP-mix in root canals of partially developed permanent teeth in monkeys. Injury to the pulp and periradicular tissues was induced and all teeth were left open to the oral environment for a period of 39–196 days to allow root canal contamination and periradicular lesion formation. Prior to the placement of the intra-canal dressing, the root canals were cleaned, shaped, and irrigated with sterile saline. Radiographic findings at the end of the observation period demonstrated reduction in the width of the root canal space and increase in root length. Closure of the foraminal opening was also observed as a result of deposition of calcified tissues. The hard tissue bridge was composed of a mixture of osteodentin, tubular dentin, and cementum or

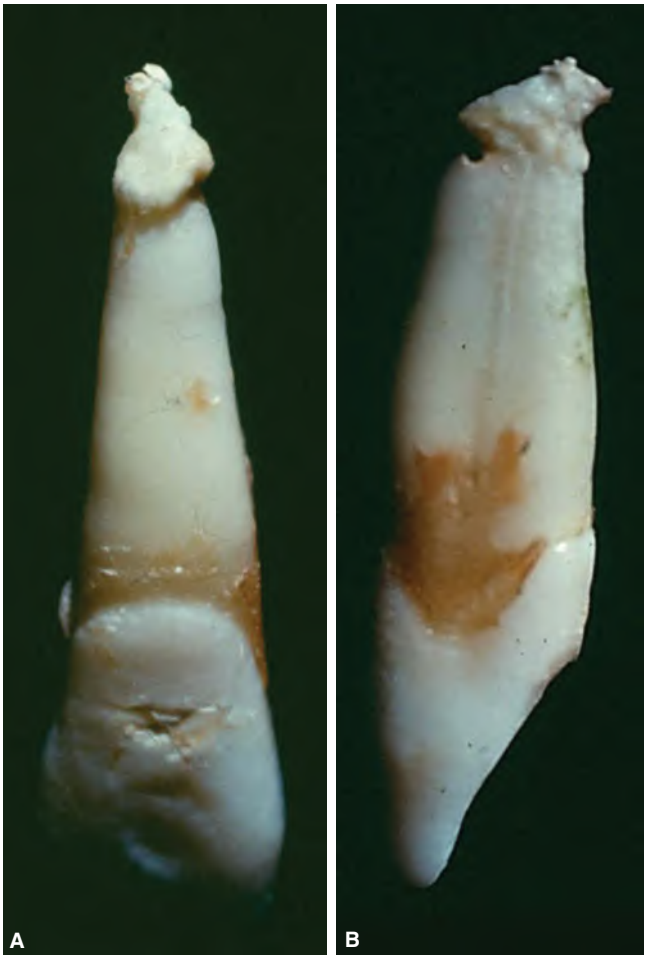

FIGURE 28-16 The hard tissue barrier that forms at the root apex following root-end closure procedures is typically an amorphous material that appears to be comprised of dentin, cementum, and bone. ***A.*** Buccal view of a maxillary central incisor showing formation of an irregularly shaped hard tissue barrier at the root apex. ***B.*** Lateral view of the same tooth showing a different angle. ***C.*** Fluorescent photomicrograph of a tooth following root-end closure shows a hard tissue barrier that is a mixture of various mineralized tissues. (Courtesy of Dr. Leif K. Bakland, Loma Linda, CA U.S.A.)

bone (Figure 28-16). Despite the absence of control teeth, the authors concluded that the use of a calcium-hydroxide-containing paste as a root canal dressing appears to accelerate the rate at which apical growth and foraminal closure occur in treatment of experimentally injured, partially developed incisor teeth.

Heithersay[95] studied a series of clinical cases that were treated with calcium hydroxide over a 14- to 75-month observation period. He used Pulpdent paste (Pulpdent, Pulpdent Corp., Watertown, MA, U.S.A.), that is dispensed in a methyl cellulose carrier. Results showed that in teeth with continued apical development, a definite fine apical canal is visible radiographically, but there is no definite apical barrier.

Calasept (Nordiska Dental, Ängelholm, Sweden) is another commercially premixed calcium hydroxide paste (calcium hydroxide, calcium chloride, sodium chloride, sodium bicarbonate, potassium chloride, and Aqua Steril). Ghose et al[103] used Calasept in traumatized, partially developed permanent central incisors of 43 patients between the ages of 8 and 12 years. All of the teeth had crown fractures with pulp exposures. The pulps had been exposed to the oral environment for one month to three years and were necrotic. Forty-nine of 51 teeth developed an apical barrier that could be confirmed clinically and radiographically. The required time for hard tissue formation ranged from 3 to 10 months.

Leonardo et al.[104] compared two commercially available calcium hydroxide pastes (Calan and Calasept) in dogs. Over an observation period of 187 days, they found that both pastes are able to induce closure of the apex with mineralized tissue. While apical closure did occur even in the presence of inflammation in the periradicular tissues, when inflammation was severe, hard tissue formation was interrupted.

Rationale for Calcium Hydroxide Therapy

Calcium hydroxide has been advocated for apexogenesis and apexification procedures for various reasons. When implanted subcutaneously in the dorsal tissues of adult rats, calcium hydroxide pellets induce heterotopic ossification in the soft tissues around the implants with a high level of predictability. The earliest sign of ossification appeared 10 days after implantation.[105] In this study, ossification was seen more frequently around calcium hydroxide pellets. When other materials were implanted, only 3 of 11 materials demonstrated any evidence of calcification.[105] The high pH of calcium hydroxide may activate alkaline phosphatase activity, and the presence of a high calcium concentration may increase the activity of calcium-dependent pyrophosphatase, which plays an important role in the mineralization process.[106]

Impact of pH

Tronstad and associates[107] studied the effects of calcium hydroxide on the dental tissues of incompletely formed roots of maxillary and mandibular incisors in monkeys. After induction of pulp necrosis, endodontic treatment was performed on replanted and non-replanted teeth. The root canals were filled with a paste of calcium hydroxide mixed with Ringer's solution and the animals were observed over four weeks. Changes in pH were registered in the periodontal ligament, cementum, in four zones of the dentin, and in the root canals. The results indicated that teeth with incomplete root formation had pH values in the normal physiologic range, while the root canal of teeth with complete root formation had a pH in the range of 10–12.2. Control teeth had pH levels ranging from 6.4 to 7.0. In another study, Javalet and associates[108] evaluated the role of the hydroxyl ions in calcium hydroxide by comparing the effect of calcium hydroxide (pH = 11.8) and calcium chloride (pH = 4.4) on the induction of apical closure. Periradicular lesions were induced in immature incisors of monkeys by repeated inoculation of the teeth with *Staphylococcus aureus*. Three of six animals were sacrificed after three months and three after six months. In the three-month samples, calcium hydroxide-treated teeth were the only ones exhibiting apical closure. In the six-month specimens, all roots filled with calcium hydroxide showed evidence of partial or complete apical closure. In contrast, only two of the calcium chloride-filled roots showed partial apical closure and two showed no evidence of hard tissue formation. The results of the study suggest that the pH of calcium hydroxide plays a more significant role in the induction of root-end closure than the presence of exogenous calcium ions.

Although there is clearly a change in intra-canal pH levels when root canals are dressed with calcium hydroxide, changes in the periradicular pH may not be as consistently reported. Some studies have shown no appreciable effect from intra-canal calcium hydroxide on the pH of the periradicular tissues[109,110] while a rise in the pH on the root surface has been reported by others.[111] When calcium hydroxide paste is placed inside root canals of extracted teeth, the pH in cavities prepared on the surface of the roots increases.[111] There is a rapid rise in the pH by day three and the increased pH value remains at approximately 10 (120-day observation period). Whether these results can be duplicated *in vivo* or whether the physiologic buffering would neutralize the pH remains to be investigated.

Impact of Calcium Ions

The action of calcium hydroxide has also been linked to its high calcium ion concentration. To substantiate this hypothesis, radioactive calcium has been used to determine whether a diffusion of calcium ions occurs from the calcium hydroxide placed over amputated pulps into the dentin bridge or pulp tissues in dogs.[112] These investigators exposed pulps and covered them with a paste of calcium hydroxide containing 120 μCi of radioactive calcium chloride per gram of calcium hydroxide powder. No evidence of radioactivity was observed within the area of new dentin formation. Almost 30 years following this experiment, Kawakami and associates[113] attempted to determine the fate of radioactively labeled calcium hydroxide paste that was embedded

in rat subcutaneous tissues. ^{45}Ca-labeled calcium hydroxide was mixed with an unlabeled root canal filling paste containing calcium hydroxide. An incision was made and the paste was embedded subcutaneously in the abdominal connective tissue. Each animal received 500 mg of the paste with 1,000 μCi of ^{45}Ca. The animals were sacrificed 7 or 13 days after the procedure. The osseous tissues demonstrated the presence of the label in the animals. Evaluation using electron microscopy confirmed that the material was calcium salts that presumably originated from the paste. Using whole-body autoradiography, movement of ^{45}Ca was also recognized. The authors concluded that the calcium component moved into bone through blood circulation. This conclusion was made based on the visualization of ^{45}Ca in the capillaries as shown by microscopic autoradiography. While these experiments demonstrate movement of calcium ions, they do not prove that the calcium ions cause mineralization or ossification of tissues.

Outcomes

Several studies have evaluated traditional apexification using calcium hydroxide to determine the time required for successful closure of the apex and the overall outcome of this procedure in general. Heithersay[95] evaluated calcium hydroxide delivered in a methylcellulose carrier (Pulpdent, Pulpdent Corp., Watertown, MA, U.S.A.) for its ability to induced root-end closure in 21 patients. Teeth were followed between 14 and 75 months. Results showed that when continued apical root development occurs, a fine canal space is visible radiographically in the apical region rather than a well-defined apical barrier. When Calasept was used in partially developed permanent central incisors with pulp exposures resulting from crown fractures, 49 of 51 teeth treated developed an apical barrier.[103] The pulps in these teeth had been exposed to the oral environment for one month to three years rendering them necrotic. Apical barriers developed in 3–10 months and were confirmed clinically and radiographically.

Morfis and Siskos[114] used calcium hydroxide powder mixed with anesthetic solution to treat 34 teeth in 12 patients ranging from 8 to 40 years in age. Root development continued in six cases; in three cases, the investigators observed formation of an apical bridge in addition to continued root development; bridge formation alone was reported in 21 cases; and, in four cases there was a complete absence of root-end closure. Lee and associates[115] evaluated the time required for calcium hydroxide to induce apical closure in immature permanent incisors of patients ranging in age from 7 to 10 years. All 32 incisors were necrotic due to traumatic injury with some exhibiting radiographic evidence of a periapical lesions. The mean duration for apical closure was 10–14 weeks. In a retrospective study designed to evaluate the efficacy of calcium hydroxide to cause apical closure in non-vital immature incisor teeth, a 100% success rate was reported.[116] Apical closures developed at variable times but within one year. The time variability was attributed to several factors: (1) Older children with narrow open apices required shorter treatment times than younger children; and, (2) the necrotic teeth that did not have evidence of periradicular infection demonstrated more rapid root development and apical closure compared with teeth with periradicular infections.

Dominguez-Reyes et al.[117] also examined calcium hydroxide treatment of necrotic permanent incisors with open apices in young patients to determine the required time for apical closure. While all 26 teeth responded well with confirmed apical closures, 23 required three to four sessions of calcium hydroxide replacement in order to obtain apical closure. In this study, the average time needed for apexification was slightly more than 12 months. In contrast, preoperative symptoms and presence of a periradicular lesion did not affect the outcome of treatment.[117] In a more recent study, incisors with necrotic pulps and open apices were treated in children ranging in age from 6 to 13 years.[118] Apical closure was reported in all 28 cases with mean treatment duration of 8.6 months (ranging from 3.24 to 13.96 months). All patients were observed for additional two years following completion of endodontic treatment. Root canal re-infection occurred in 7.1% of the cases highlighting the importance of a good root canal seal to maintain long-term success.

While these outcomes studies demonstrate a high level of success, traditional apexification using calcium hydroxide therapy may be unpredictable. The shortcomings of apexification with long-term calcium hydroxide therapy include: (1) Variability in the time required for open apices to close; (2) multiple visits necessary to replace and refresh the calcium hydroxide paste; (3) potential for coronal leakage and canal re-infection; (4) variable and undefined types of tissue laid down as an apical barrier; and (5) potential coronal fracture, if the patient doesn't adhere to a strict follow-up protocol.

Apical barrier formation may take from 3 months to 2 years.[83,119,120] This time period frequently necessitates multiple re-applications of the calcium hydroxide paste.[121–127] To date, there has been no consensus on the optimal timing to change the dressing material and the impact of calcium hydroxide replacement on the rate and quality of apical barrier formation. Recommendations by researchers for re-application of calcium hydroxide varies from once every month, once every three months, once every six to eight months, or not at all.[120,124,125,128,129]

While coronal leakage and canal re-infection intuitively should impact outcomes of apexification procedures, a clear impact of infection on the success of apexification procedures has not been established. Some studies report that treatment time is increased in the presence of infection,[130,131] and other studies have found no statistically significant differences in treatment outcomes when infection is present versus when there is an absence of bacteria.[103,119,122,126]

The type and quality of bridge formed at the apex following apexification with calcium hydroxide therapy may also be unpredictable. The barrier may be an incomplete bridge that does not cover the entire apex.[132] While a traditional two-dimensional periapical radiograph may indicate

the appearance of a complete bride in one plane, a view from another angle may show incomplete bridge formation. The calcified bridge formed following apexification is a porous structure[116] that may be permeable to microbial irritants. Furthermore, the pores themselves may contain remnants of necrotic tissue and can result in persistent periradicular irritation and inflammation.[88]

Recently, another potential issue has been identified with the long-term exposure of dentin to calcium hydroxide. While in the past, susceptibility of cervical root fracture was primarily attributed to the thin dentin walls, researchers have found that long-term application of calcium hydroxide has deleterious effects on the structural integrity of root dentin.[61–68] These studies have demonstrated that placement of calcium hydroxide in root canals for longer periods of times (typically required for apexification) changes the structure of dentin and significantly reduces the tooth's ability to resist forces that may lead to fractures.

APICAL BARRIERS

Based on the shortcomings of apexification with long-term calcium hydroxide therapy, there has been continued research to identify alternative means to promote apical closure in immature permanent teeth. Long-term therapy increases the risks of patient attrition; repeated appointments may be disruptive to patients' schedules and difficult for young children who may be apprehensive about dental visits, and therefore, compliance may become an issue; geographic relocation renders difficulties in following patients to continue therapeutic replacement of calcium hydroxide; and, calcium hydroxide therapy may be ineffective in some cases and root-end closure may never occur (Figure 28-17).

Kuttler[2] stated that radiographic and clinical signs of a more rapid periapical regeneration can be observed when dentin shavings are implanted in the terminal portion of the root canal. Teeth with apical plugs of calcium hydroxide have demonstrated mean leakage values that are significantly less than those obturated without plugs.[133] Pitts and associates[134] compared histologically calcium hydroxide to dentin to determine which material is more effective as an apical plug. They used a cat model and over-instrumented the cuspid teeth to simulate open apices. Based on their findings, both materials are effective in controlling the filling materials within the confines of the canals. Formation of a calcified tissue barrier is more rapid, however, when using dentin plugs (one month in the majority of specimens containing dentin plugs in contrast to three months in samples plugged with calcium hydroxide).

Tricalcium phosphate has also been proposed as an apical barrier in a single-appointment technique.[135] When compared to calcium hydroxide-CMCP paste used in multi-appointment treatment of open apices, tricalcium phosphate shows no overfills in the apices of permanent human teeth. In contrast, calcium hydroxide treatment can result in overextensions of the obturation material. Because of its larger particle size, tricalcium phosphate may offer improved resistance against compaction, and therefore, less material is extruded.

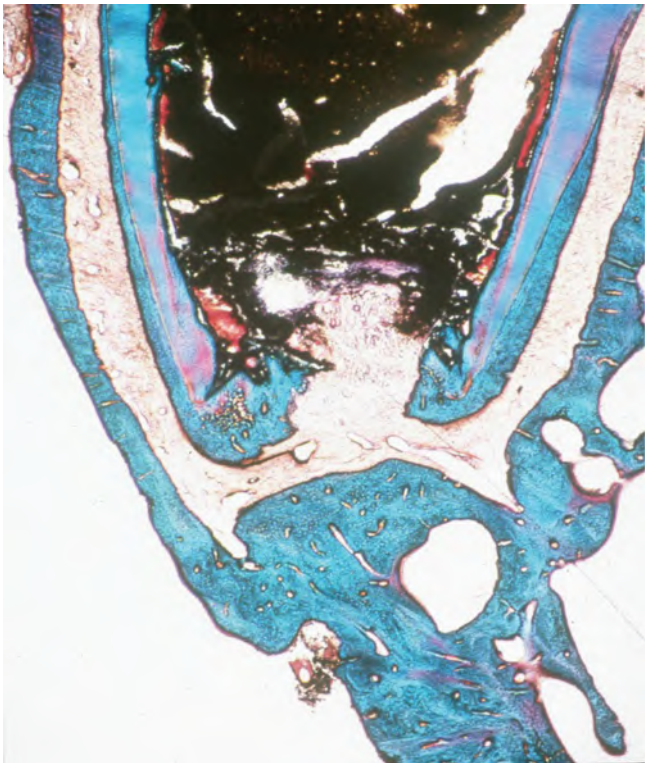

FIGURE 28-17 Photomicrograph of a specimen stained with Masson's Trichrome. This tooth was treated with calcium hydroxide for three months. Figure shows presence of an open apex.

Brandell and associates[136] created open apices in incisor teeth of monkeys by over-instrumenting canals with a size #50 K-file to a level 0.5 mm beyond the radiographic apex. Then they evaluated apical closures produced by three different materials. Teeth were divided into four groups and the apical 2 mm of each root canal was packed with: (1) Demineralized dentin (organic component); (2) hydroxyapatite (a substitute for inorganic dentin); or (3) dentin chips. Teeth in group 4 did not receive an apical plug and served as negative controls. Four of the monkeys were sacrificed after three months. Of the 24 samples evaluated in this time period, only one (packed with demineralized dentin) demonstrated complete closure of the apex. At the six-month observation period, none of the demineralized dentin plugs showed complete apical closure. In contrast, four of six roots filled with hydroxyapatite exhibited complete apical closure, and three of six roots with dentin chips showed similar findings. The authors concluded that the organic component of dentin may not be effective in inducing apical hard tissue formation.

Mineral trioxide aggregate (MTA) is another material that has been used as an apical barrier. The principle components of MTA include tricalcium silicate, tricalcium aluminate, tricalcium oxide and silicate oxide. MTA powder consists of fine hydrophilic particles, that set in the presence of moisture and

result in a colloidal gel that solidifies to a hard structure.[137] Microanalysis of the powder has shown that calcium and phosphorous are the main ions present. Dye leakage[138] and bacterial leakage[139] studies have demonstrated that MTA leaks significantly less than amalgam and Super EBA. Mutagenicity and cytotoxicity experiments using the Ames Test have shown that MTA does not produce higher reversion rates[140] and that set MTA is significantly less toxic (Figure 28-18) than set amalgam, composite, Super EBA or set IRM.[141] MTA has been successfully used as root-end filling during apical surgery[142] and repair of root perforations.[143] A significant benefit of MTA when placed in contact with periradicular tissues is its ability to induce cementum formation around the repair area.[142] The cementum layer may provide an additional biological seal to enhance the apical resistance to microbial leakage into the periapical tissues. The high pH of MTA after mixing (reported to be 10.2, rising to 12.5 at three hours and thereafter), similar to that of calcium hydroxide,[137] may be one of the mechanisms by which induction of hard tissue formation may occur in its presence.

The use of MTA as an apical plug in the treatment of immature non-vital teeth has potential as well. In 1999, Shabahang et al. [28] compared MTA plugs to calcium hydroxide apexification and induction of root-end formation with osteogenic protein-1. In this study, MTA induced hard tissue formation with the most degree of consistency. Similar to previous findings in other applications of MTA, a continuous band of cementum formed (Figure 28-19). In two cases, MTA was extruded beyond the confines of the root apex. In one, cementum formed around the extruded MTA (Figure 28-20). Again, the benefit of this cementum layer is an added biological seal that can enhance healing of the periradicular tissues.

Ham et al.[144] compared the healing of immature non-vital teeth in monkeys. Teeth were treated with an apical plug of MTA or underwent traditional apexification with calcium hydroxide. Based on their findings, MTA-treated teeth display less inflammation and a greater amount of hard tissue formation. In a dog model, investigators examined the effects of calcium hydroxide paste used before placement of an MTA apical plug. It was discovered that MTA promoted apical root formation and periradicular healing. The initial use of calcium hydroxide paste was not necessary for apical root formation to occur. Furthermore, the use of calcium hydroxide was directly proportional to an increase in extrusion of

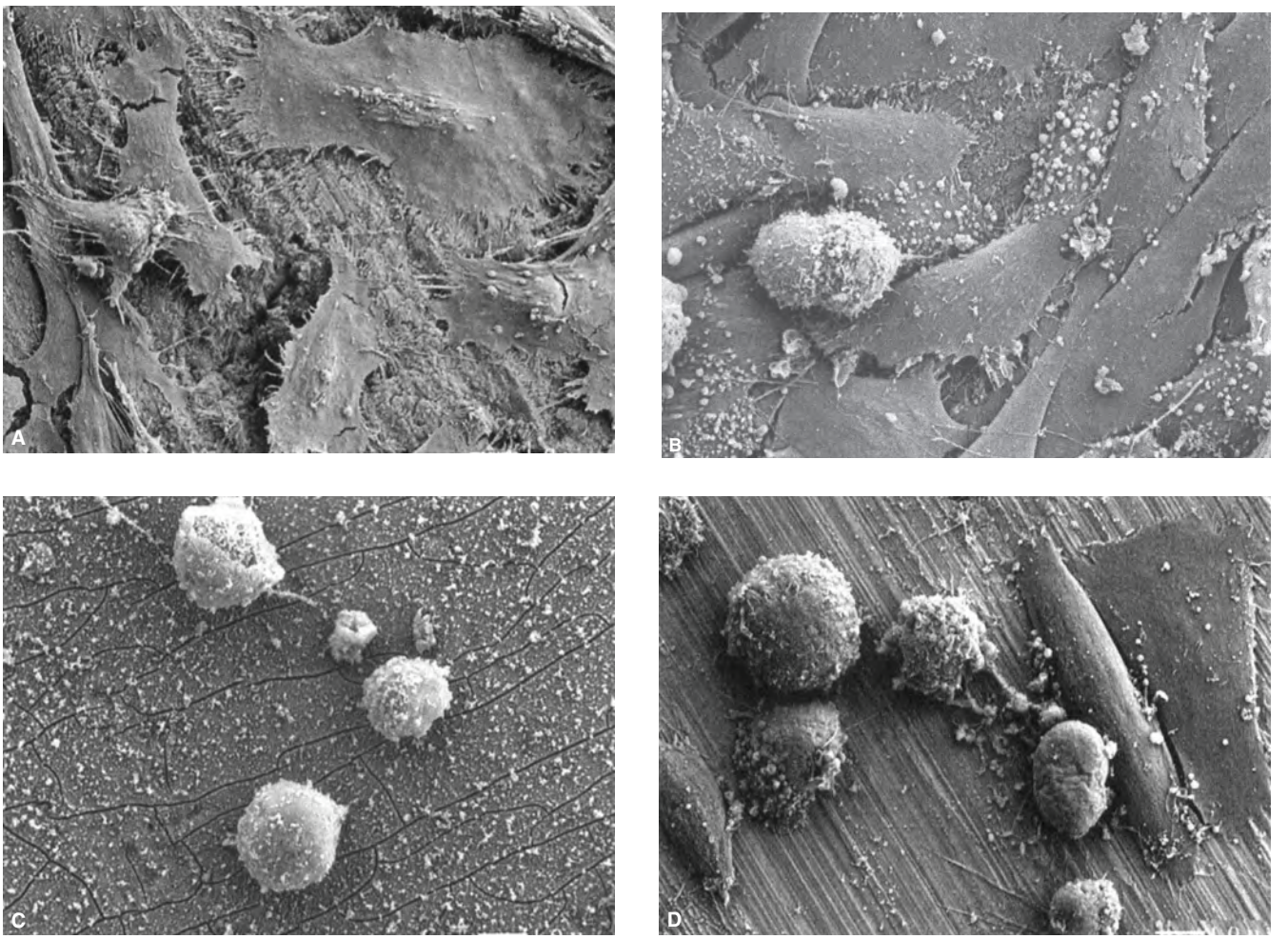

FIGURE 28-18 Scanning electron photomicrographs of cells cultured on various materials. The images show that cells grow nicely on MTA (A) and composite (B) as depicted by expanded cells but poorly on amalgam (C) and IRM (D).

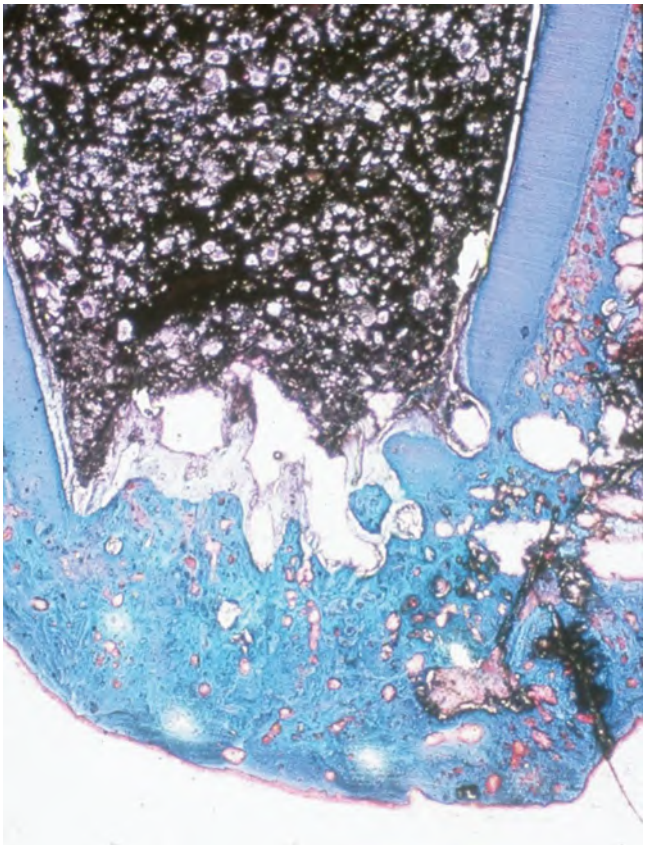

FIGURE 28-19 Photomicrograph of a ground specimen stained with Masson's Trichrome demonstrating apposition of a thick band of cementum at the apex of an immature tooth treated with MTA.

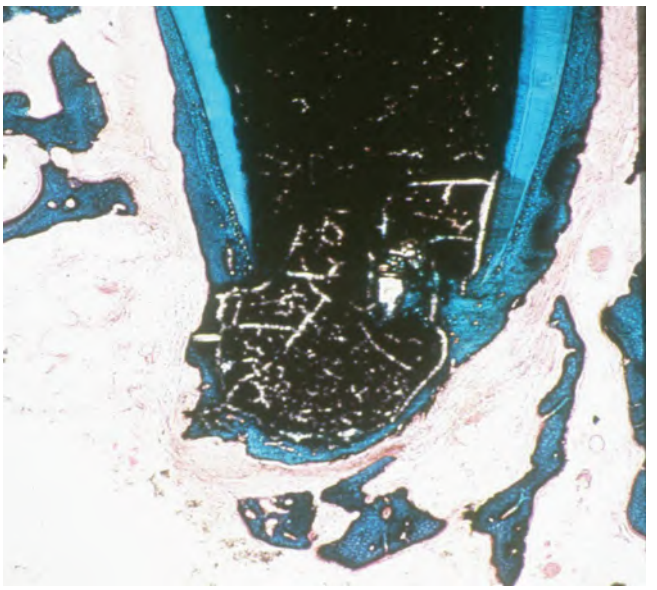

FIGURE 28-20 Specimen demonstrating small extrusion of MTA beyond the confines of the root apex. A complete band of cementum has formed around the extruded MTA.

MTA and formation of barriers beyond the limits of the root canal walls.[145] In contrast to the previous study, other investigators have found that intra-canal medication with calcium hydroxide for one week results in better marginal adaptation of an apical plug of MTA.[146] This finding may be related to the ability of calcium hydroxide to eliminate pulp tissue remnants or organic components remaining on the dentin surface thus allowing a closer adaptation of MTA to dentin. Hachmeister et al.[147] examined the bacterial leakage patterns in a simulated model of immature non-vital teeth and concluded that the technique of placing MTA rather than the intrinsic characteristics of the MTA material itself has the greatest impact on the degree of bacterial leakage.

Technique

Once root canal debridement is completed, pluggers that are commonly used for warm vertical compaction of gutta-percha are sized to fit sequentially in the root canal space. The smallest plugger should fit loosely at a level ~ 0.5 mm from the working length. A small increment of MTA is then placed in the middle to apical third of the root canal system using one of several commercially available MTA carriers (Figure 28-21). The MTA is compacted with the pre-fitted pluggers. Ultrasonic vibration can be used on the pluggers to help advance MTA towards the apical extent of the canal.[148–151] The use of a matrix material is generally not required to limit the movement of MTA into the periradicular tissues. The movement of MTA into the apical area of the canal can be facilitated

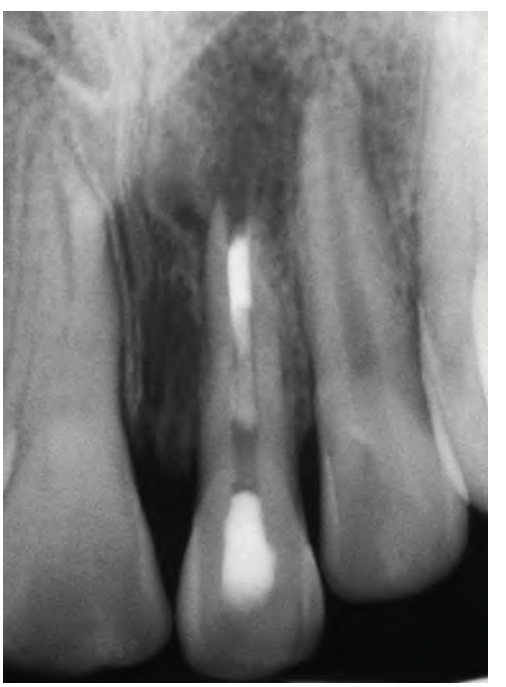

FIGURE 28-21 Radiographic image of a maxillary left lateral incisor with incremental placement of MTA in the mid to apical third of the root canal. The MTA is then further advanced with endodontic pluggers and if necessary, via ultrasonic vibration. (Courtesy of Dr. Douglas H. Snider, Chico, CA, U.S.A.)

by mixing the first increments of MTA in a moist consistency and by using smaller increments initially. The moist material will require light pressure application.

Once an adequate apical plug of MTA is compacted to the working length and confirmed radiographically, excess material can be removed from the coronal and middle thirds of the root canal system with the use of moist paper points. Thickness of the MTA plug should be approximately 3–5mm (Figure 28-22) to minimize the possibility of future apical leakage.[148,150–152,153–156] In many instances, MTA can be compacted to a level just below the cemento-enamel junction. If the canal space is long, gutta-percha and sealer may be used to obturate the coronal segment of the canal space. This may necessitate an additional appointment to allow setting of the MTA prior to obturation of the remaining segment of the canal space with gutta-percha and sealer. The restorative core material can then be placed over the gutta-percha. Extension of the core layer into the coronal third of the canal may enhance fracture resistance of the tooth.[153]

Outcomes

Following the publication of an animal study proving the utility of MTA as a suitable material for root-end closure procedures,[28] several clinical cases were published to report successful management of non-vital immature teeth with MTA[157–165] including a case where calcium hydroxide apexification had failed.[166]

Pace et al.[167] published a report on 11 teeth with immature root apices that were treated with MTA apical plugs.

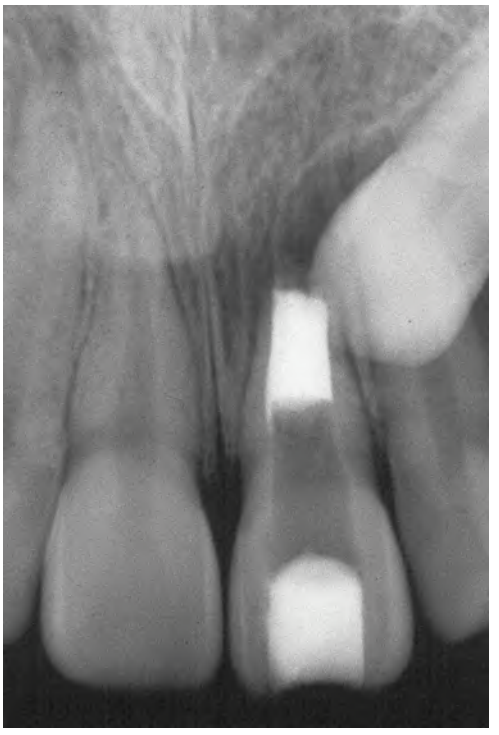

FIGURE 28-22 Radiographic image of maxillary left central incisor demonstrating placement of a 3- to 5-mm thickness of MTA. (Courtesy of Dr. Marshall E. Gomes, Lodi, CA, U.S.A.)

Each tooth received intra-canal calcium hydroxide for one to two weeks followed by a 3- to 5-mm apical plug of MTA. At the two-year posttreatment follow-up, 10 of 11 cases had completely healed while the remaining tooth showed incomplete healing. Based on these results, the authors concluded that MTA apical plugs present a viable treatment protocol for root-end induction. In a similar case series, five immature teeth with necrotic pulps were treated with an apical plug of MTA after one to six weeks of calcium hydroxide dressing for canal disinfection. Four of five teeth showed clinical and radiographic signs of healing after two years. MTA had extruded beyond the root apex in the case that did not heal.[168] Extrusion of MTA more than 2 mm beyond the root apex may retard healing and cementum formation (Figure 28-23).

Sarris et al.[169] treated teeth of children with an MTA plug. The mean age in the study population (n = 15) was 11.7 years and all 17 teeth were diagnosed with a necrotic pulp and an immature apex. Following initial canal debridement, all teeth were dressed with calcium hydroxide for one week. Each canal then received a 3–4 mm thick plug of MTA at the root apex. Patients returned after one week for final obturation of the root canal space with thermoplastisized gutta-percha. MTA placement was considered adequate in 13 of 17 teeth treated. Teeth were followed from 6 to 16 months with a mean follow-up time of 12.53 months. A clinical success rate of 94.1% and a radiographic success rate of 76.5% was reported. At the final follow-up visit, 17.6% of the teeth had uncertain healing radiographically.

In a retrospective study, 20 teeth with open apices were evaluated in 19 patients.[150] Calcium hydroxide was placed in the canals for at least one week prior to placement of a 4 mm thick MTA apical plug. Of the 20 teeth evaluated, 16 were of young patients with under-developed apices. The follow-up period ranged from 12 to 44 months (26.7 month average). Based on periradicular index scores of 1 or 2 and absence of clinical signs or symptoms at the follow-up examination, 93.75% were considered healed.

In a case series study, Nayar et al.[170] published the outcomes of 38 permanent teeth with immature apices that were treated with an apical plug of MTA. Presence of pre-operative periradicular radiolucency did not impact the healing outcome. Based on a 100% clinical and radiographic success rate at the 12-month follow-up examination, the authors concluded that treatment of immature permanent teeth with an MTA apical plug is a predictable procedure. These findings were corroborated by Annamalai and Mungara[171] a year later. In their report, all 30 teeth tested were clinically and radiographically successful at the 12-month follow up visit. Complete root-end closure was documented in 86.6% of cases and root growth in 30% of cases. Moore et al.[172] treated 22 necrotic immature permanent incisors with an apical plug of white MTA. The mean age in the 21 children was 10 years. Again, calcium hydroxide was used as intra-canal medication to disinfect the canals prior to placement of the MTA plug. The mean follow-up time in this study was 23.4 months. Based on clinical and radiographic evaluations, a success rate of 95.5% was reported. A side effect that was noted by the authors was coronal discoloration in 22.7% of the teeth that were treated.

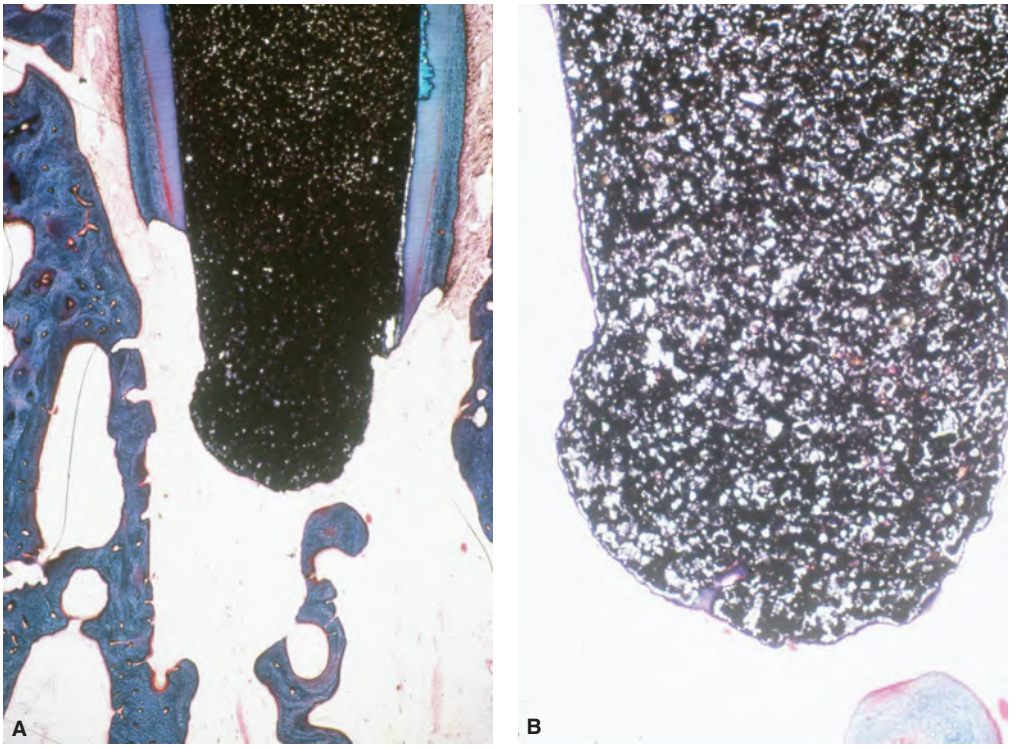

FIGURE 28-23 Photomicrograph illustrating extrusion of MTA further beyond the root apex. ***A.*** Low magnification shows no cementum formation around the extruded MTA. ***B.*** Higher magnification of the same specimen shows a thin but continuous band of cementum around the extruded MTA material.

Since the introduction of MTA, more apical plug cases have been completed with MTA and have become available for evaluation. Two studies with larger sample sizes examined the outcomes of MTA in non-vital immature teeth. In a prospective study, the outcomes of 57 teeth with open apices were studies in 50 patients. In this report, no interim medications were used and MTA apical plugs were placed in a single appointment. Forty-three cases provided a minimum follow-up period of 12 months. Using the Periapical Index score to determine decrease in the size of apical lesions, healing was recorded in 81% of cases. The authors concluded that MTA can be used in a single appointment protocol in teeth with open apices with predictable results as an alternative to calcium hydroxide apexification.[173] The second study was a retrospective analysis of 144 teeth in 116 patients.[174] All teeth were treated in the same private endodontic office between 1999 and 2006. Fifty-two teeth were medicated with calcium hydroxide for three weeks prior to placement of the MTA apical plug. Treatment in the other 92 teeth was completed in one visit. Of the 144 teeth that received treatment, 78 (54%) were available for follow-up evaluation (approximately 60% of the one-visit cases and 40% of the two-visit cases) with a mean follow-up period of 19.4 months. Overall success rates were 93.5% in the single visit cases and 90.5% in the two visit cases. In this study, while all teeth had open apices, 119 of the 144 teeth treated were considered immature teeth. The remainder were mature teeth that had open apices. Forty-five of immature teeth were medicated with calcium hydroxide and treated with an apical plug on the second appointment and 74 were treated in a single appointment. Sixty-eight of the 119 teeth with immature apices (57%) were available for follow-up examination (same distribution of approximately 60% one visit and 40% two visits). Of the 74 teeth that had at least one year follow-up, 96.5% healed in the single visit group and 89% healed in the two visit group. Only one case in each treatment category failed with respect to apical healing. Four patients that were initially treated with calcium hydroxide did not return to complete treatment in a timely manner and when they did, the teeth were no longer restorable.[174]

Studies have also directly compared the outcomes of MTA used as an apical plug to calcium hydroxide apexification. In one of these two armed studies, El-Meligy and Avery[175] selected 15 children each with at least two necrotic permanent teeth requiring root-end closure procedures. At the 12-month follow-up, two of the teeth treated with calcium hydroxide became re-infected. In contrast, all MTA-treated teeth showed absence of infection and appeared clinically and radiographically intact. In the same year, Pradhan et al.[176] conducted a comparative study to determine the success rates and length of time required to achieve closure of open apices using a calcium hydroxide apexification protocol versus the MTA plug protocol. Twenty non-vital permanent maxillary incisors with immature apices were assigned to either the calcium

hydroxide treatment or MTA treatment groups. The MTA protocol consisted of 7 days of disinfection with a calcium hydroxide dressing followed by placement of an MTA plug that was placed in the apical third of the root canal. The calcium hydroxide apexification protocol involved placement of intra-canal calcium hydroxide until an apical barrier was evident. In both groups, the remaining portion of each canal was obturated with gutta-percha and sealer. While no significant difference was observed in the healing of the periradicular lesions, completion of treatment was significantly more rapid when MTA was used as an apical plug (0.75 ± 0.5 months) compared to traditional calcium hydroxide therapy (7 ± 2.5 months). This significant reduction in treatment time is clinically important because it reduces chair-side time for clinicians and enhances convenience for patients.

In a systematic review and meta-analysis comparing the outcomes of calcium hydroxide apexification and MTA apical plugs, the authors concluded that the two protocols have similar success rates.[177] When combining the data in the two studies discussed earlier by El Meligy and Avery[175] and Pradhan et al.,[176] the difference in clinical success between the two treatment regimens was not *statistically* significant. Yet 100% of the MTA cases were successful at the final follow-up evaluation in comparison to a 92% success rate in the calcium hydroxide treated cases.[177]

Based on the existing body of evidence, treatment of immature permanent teeth with an apical plug is a good alternative to long-term calcium hydroxide therapy. The advantages include more predictable closure of the apical opening, reduced treatment time, and fewer appointments. With the availability of materials such as MTA that provide a good seal and biocompatibility, placement of an apical plug provides a more predictable treatment option. The plug presents a barrier that provides a matrix for immediate obturation. Histologic evaluation of MTA in periradicular regions has shown consistent apposition of hard tissues including cementum and bone. The formation of this added layer of biologic seal confirms the biocompatibility of MTA.

Another possible benefit of single visit root-end closure procedures is minimized exposure to medicaments that may have deleterious effects on the dentin structure. In addition to their antimicrobial activity, sodium hypochlorite and calcium hydroxide have tissue dissolving properties. This characteristic is valuable when removing pulp remnants; however, dissolution of organic components of dentin during longer exposure times by these chemicals can also alter the structural integrity of dentin. These changes may be, at least partially, responsible for fractures, that have been previously reported in the cervical region of immature teeth that had undergone apexification procedures.

The requirement for fewer appointments is especially valuable in young patients. Children are frequently apprehensive and intolerant of longer appointments. Therefore, techniques that can minimize return visits enhance the patient and doctor experience. In addition, failure to return for completion of treatment can have a significant impact on outcomes and ultimately restorability of these teeth.[86]

Despite its advantages, treatment of immature teeth with apical plugs has certain shortcomings. Most importantly, placement of an apical plug only addresses the apical opening. Generally, failure in immature teeth with a history of apexification has been due to fractures in the cervical region of the tooth rather than the root apex. The primary causes of cervical root fracture are thin dentin walls in these immature teeth and long exposure times to chemicals that can alter dentin structure. Substitution of apical plugs in lieu of long-term calcium hydroxide therapy to achieve root-end closure can eliminate the latter issue. Nevertheless, while treatment of an immature permanent tooth with an apical plug can address the apical opening, continued root development ceases once a vital pulp is removed. The remaining thin dentin walls of the root structure stay weak and susceptible to fracture if subjected to future traumatic injuries. Another potential drawback is the possibility of tooth discoloration resulting from MTA placement in the canal. This issue is clearly significant in anterior teeth. To minimize color changes in the coronal portion of anterior teeth, MTA plugs can be limited to the apical 4–5 mm of the root canal space. The remaining segment of the canal space may be restored with bonded resin in shorter roots. In longer roots, a layer of gutta-percha and sealer can be used to obturate the coronal two-thirds of the root canal space prior to restoring the coronal area with bonded resin. Placement of bonded resin below the cemento-enamel junction may also increase the fracture resistance of the tooth.[178,179]

Future efforts in pulp revascularization and pulp regeneration protocols will provide a more complete solution for treatment of permanent teeth with immature roots that have sustained traumatic or carious injuries to the pulp. The benefit of restoring circulation or regenerating pulp include re-establishment of viable tissues that allow the affected tooth to mount an immune response against microleakage of microorganisms into the root canal system, and continued root development along the entire root length. Achievement of this objective requires proper disinfection and seal to prevent re-infection; proper signaling for stem cells to promote regeneration of pulp rather than ectopic formation of tissues that are not desirable in the canal space (e.g., bone); and a suitable scaffold that allows timely release of the signaling proteins while serving as a barrier for guided tissue regeneration to prevent ingress of bone (Figure 28-24). Timely release of signaling proteins requires a scaffold material that is absorbed over a suitable time period thereby gradually releasing the signaling material. This gradual release of the protein would allow differentiation of the stem cells into the desired specialized cells and ultimately regeneration of the desired tissue. For a more detailed discussion of this topic, see Chapter 29, "Regenerative endodontics."

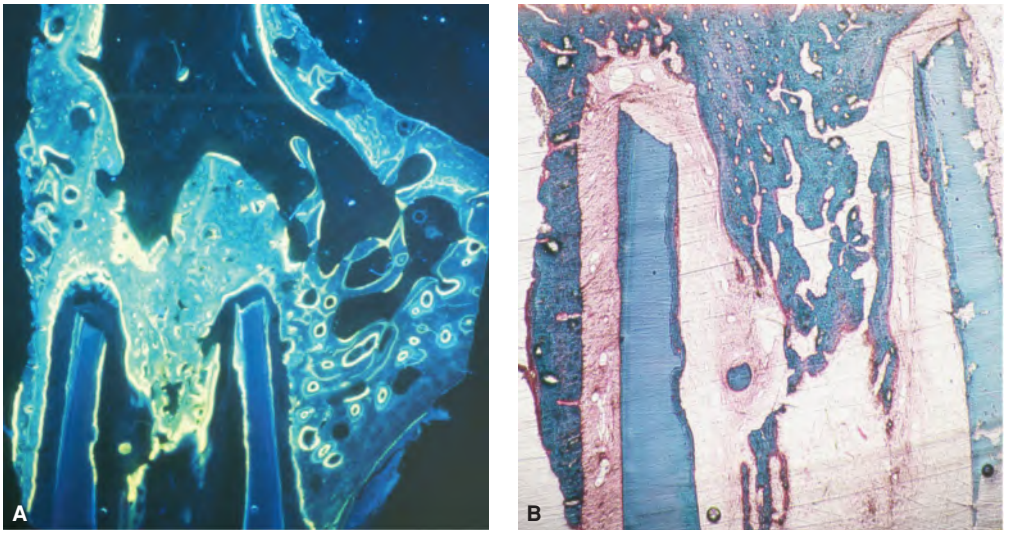

FIGURE 28-24 Photomicrograph of an immature tooth with a wide open apex that was subjected to root-end closure. ***A.*** Fluorescent microscopy shows take-up of tetracycline by the newly deposited cementum on the inner surface of the root dentin and bone. ***B.*** Masson's Trichrome staining shows ingrowth of bone into the root canal space.

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Chapter 29

Regenerative Endodontics

Anibal R. Diogenes, Anthony J. Smith

INTRODUCTION

The concept of the regeneration of organs or organisms can be traced back to ancient Greece, and regenerative medicine has a rich history,[1–3] providing a foundation for our vision of the field today. The Human Genome Project together with exciting advances in stem cell biology, tissue engineering and biotechnology, over the last couple of decades, have all contributed to "the coming of age" of regenerative medicine and its clinical translation.

Dentistry has played an important role in the history of regenerative medicine, including early attempts at dental transplantation by Charles Allen in 1687[4] and pulp capping by Pfaff in 1756 and others[5] subsequently. The introduction of calcium hydroxide[6] and, more recently, the widespread use of Mineral Trioxide Aggregate (MTA)[7] and parallel products such as the calcium trisilicates[8] have all emphasized the central role for biologically-based therapies in endodontics. In a review of the current status of regenerative research in endodontics, there was a call for action, to exploit recent progress in stem cell and other areas of biology, to clinically translate regenerative endodontic therapies.[9] A wide range of strategies are now emerging, from which to exploit the biological responses within the pulp for tissue regeneration.

This chapter discusses the biological principles upon which the development of future regenerative endodontic therapies will be based and the progress and challenges at the present time.

BIOLOGICAL PRINCIPLES ASSOCIATED WITH REGENERATIVE ENDODONTICS

Odontoblasts show an intimate relationship with the other cells of the pulp, and their extracellular matrices reflecting its pivotal role in determining the structure and functional activities of the dentin-pulp complex. While the odontoblast has long been recognized for its synthetic/secretory actions leading to dentin deposition,[10] its environmental sensing functions, and their contributions to the innate and immune defence responses of the tooth, are being increasingly identified.[11,12] Thus, cellular activity within dentin-pulp is important not only for its development but also, its subsequent homeostasis and responses to environmental challenges.

A long tradition of vital pulp therapy in endodontics emphasizes a common goal of developing effective biological approaches, to address the effects of more aggressive carious challenge to the tooth. Historically, empirical pulp capping protocols have provided the foundation for these biological approaches. However, the parallel deepening of our understanding of the biological behaviour of dentin-pulp in health and disease has provided a platform for exciting developments with biologically-based therapies in endodontics. These developments encompass a broad spectrum of approaches, ranging from simple exploitation of existing biological activities within dentin-pulp, to ambitious tissue engineering approaches at both the tissue and whole tooth levels.[13]

Tissue engineering has been defined as "an interdisciplinary field that applies the principles of engineering and life sciences toward the development of biological substitutes that restore, maintain, or improve tissue function or a whole organ."[14] Importantly, tissue engineering relies upon "understanding the principles of tissue growth, and applying this to produce functional replacement tissue for clinical use", and depends upon the basic supposition that "the employment of natural biology of the system will allow for greater success in developing therapeutic strategies aimed at the replacement, repair, maintenance, and/or enhancement of tissue function."[15]

Thus, any biological consideration of dentin-pulp in the context of repair and regeneration and maintenance/facilitation of vitality requires an understanding of the cells and their extracellular matrices in dentin-pulp and the molecular signaling networks, that influence the behavior of these cells in health and after injurious challenge, whether of microbial or traumatic origin. Of particular importance in the context of regenerative endodontics is the presence of stem and progenitor cell populations in pulp and the surrounding tissues.

Oral Stem and Progenitor Cells

During embryonic development, migration of cranial neural crest cells to the mesenchyme of the first branchial arch results in generation of the ecto-mesenchymal cells giving rise to the various cell populations of the dental papilla and dental follicle of the tooth germ. Genetic-based cell lineage analysis has confirmed that physiological primary odontoblasts are derived from these cranial neural crest cells.[16] After the last division of the peripheral cells of the dental papilla in the bell stage of tooth development, the daughter cell closest to the dental basement membrane undergoes terminal differentiation to an odontoblast, while the other daughter cell is not exposed to this morphogenic influence and contributes

to the cell-rich layer of Höhl underlying the odontoblast layer. Thus, as tooth development is completed, the mature tooth will contain undifferentiated cells within the cell-rich and other regions of the pulp.

These various undifferentiated cells in the mature pulp contribute to the different stem cell populations reported to be present in dental pulp. These cells all demonstrate mesenchymal stem cell (MSC)-like properties, in respect of their expression of cell surface markers and multipotent differentiation potential along mesenchymal cell lineages. Such MSCs are generally considered to be responsible for tissue cell turnover and reparative responses following tissue injury. Bone marrow represents the classical niche for MSCs, although they have now been reported in many tissues and organs. Whether all those in pulp are derived from cranial neural crest cells remains to be established, since some of the cells of the central pulp do not appear to be neural crest derived.[16] Furthermore, a dual origin for MSCs from both within and out with the tooth has been identified using genetic cell lineage tracing.[17] Minimal criteria for defining multipotent mesenchymal stromal cells have been published[18] and slight variations in cell surface marker expression, and differentiation potential between cells isolated from different sources, may reflect the influences of their local tissue niches and growth conditions. The impact of these slight variations in properties between different MSC populations, on their responsiveness to various potential morphogens and multipotent lineage differentiation, remains to be fully elucidated. Interestingly, however, cell surface expression of CD271 (p75NTR) or nerve growth factor receptor (NGFR), may be associated with maintenance of the undifferentiated status of MSCs,[19] and allows selection of neural crest derived cells.[20,21]

Isolation of MSC-like stem cells has now been reported from both deciduous and permanent teeth. Dental pulp stem cells (DPSCs) were the first population of dental stem cells to be described.[22] In contrast to bone marrow stromal cells (BMSCs), a significant proportion of DPSCs express the pericyte marker 3G5,[23] and this possibly reflects their probable perivascular tissue niche. Characterization of their expression of surface markers identified the presence of STRO-1, CD29, CD44, CD73, CD90, CD105, CD146, CD166 and CD271.[24]

More recently, a population of cells termed stem cells from the root apical papilla (SCAP) has been reported.[25] Transplantation of these SCAPs, and a population of periodontal ligament stem cells (PDLSCs) in a mini-pig model, allowed generation of a root/periodontal complex capable of supporting a porcelain crown.[25] The development of therapeutic applications for SCAPs in immature teeth with incomplete root development offers significant potential. The opportunity to isolate SCAPs from extracted third molars allows a ready source of these cells. Use of autologous cells should always be a priority in clinical applications to avoid potential problems from immune rejection.

The isolation of Stem cells from Human Exfoliated Deciduous teeth (SHED) provides a further valuable potential source of autologous cells, that can be isolated non-invasively.[26] SHED are distinct from DPSCs, exhibiting higher proliferation rates and exposed to appropriate morphogenic signals, are capable of differentiating into functional odontoblast-like cells,[27] and being used to tissue engineer dental pulp.[28] A population of Dental Follicle Precursor Cells (DFPCs) has also been reported.[29] While these cells can form mineralized nodules *in vitro*, transplantation *in vivo* did not result in cementum or bone formation. These various dental stem/progenitor cell populations offer exciting opportunities for future exploitation in regenerative endodontics (Figure 29-1). Characterization of each population, and detailed comparison with MSCs from other sources,[31] will help to underpin their clinical application, although the challenges of generating sufficient numbers of pulp stem cells to GMP standards,[32] and their translation to robust clinical regenerative protocols should not be underestimated.[33]

Signaling Networks (Growth Factors, Cytokines, Morphogens) for Regenerative Endodontics

Physiological tooth development is characterized by an elaborate sequence of reciprocal epithelial-mesenchymal

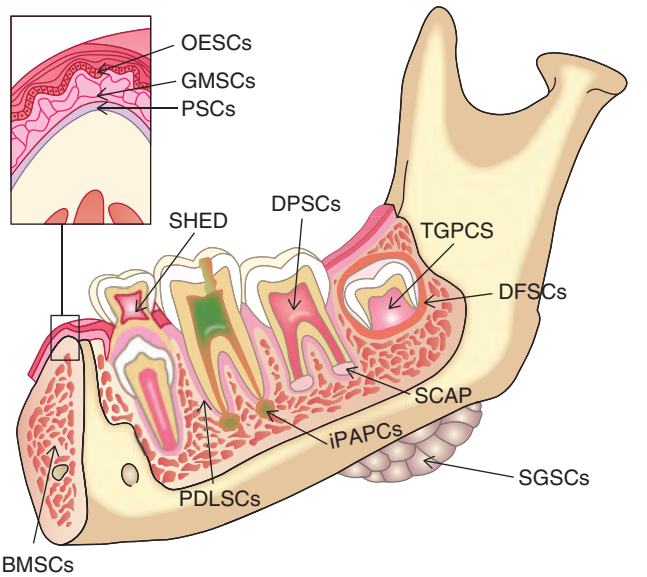


FIGURE 29-1 Schematic illustration showing potential sources of postnatal stem cells in the oral environment. Cell types include tooth germ progenitor cells (TGPCs), dental follicle stem cells (DFSCs), salivary gland stem cells (SGSCs), stem cells of the apical papilla (SCAP), dental pulp stem cells (DPSCs), inflamed periapical progenitor cells (iPAPCs), stem cells from human exfoliated deciduous teeth (SHED), periodontal ligament stem cells (PDLSCs), bone marrow stem cells (BMSCs), and, as illustrated in the inset, oral epithelial stem cells (OESCs), gingival-derived mesenchymal stem cells (GMSCs), and periosteal stem cells (PSCs). (Adapted with permission from Hargreaves et al.[30])

interactions that are temporo-spatially regulated, resulting in the very reproducible formation of an appropriately shaped tooth in the correct anatomical position from previously uncommitted cells. The regulation of these developmental events is achieved through the temporo-spatial expression of various signaling mediator molecules (transcription factors, growth factors and other proteins).[13,34]

Our understanding of the molecular signaling involved in tooth development provides a potential blueprint from which we might build therapeutic strategies for regenerative endodontics. However, the absence of both dental epithelial signaling networks and the physiological control processes in the mature tooth during repair and regeneration, requires the identification of alternative signaling sources. In particular, molecular signaling of the following cellular events is required: recruitment of stem/progenitor cells (chemotaxis/cell homing) in adequate numbers (cell division); induction of terminal differentiation of odontoblast-like cells (to replace any primary odontoblasts lost through cell necrosis); up-regulation of synthetic/secretory activity of odontoblast-like cells (and also, up-regulation of primary odontoblasts in the case of reactionary dentinogenesis) and subsequent down-regulation when complete to avoid pulp canal obliteration; stimulation of local angiogenesis at sites of injury to support dentinogenic events. The combination of all of these cellular events, represents the process of reparative dentinogenesis, that is the tertiary dentinogenic sequence of events seen after focal death of primary odontoblasts, beneath a more active carious lesion, and is responsible for the matrix secretion comprising a dentin bridge at sites of pulp exposure. The other variant of tertiary dentinogenesis is reactionary dentinogenesis that represents the focal up-regulation of primary odontoblast secretion of dentin beneath a less aggressive carious lesion, where primary odontoblasts have survived.[35]

Consideration of the pathogenic events taking place during dental caries, the principal cause for endodontic disease, provides valuable clues as to how much of the molecular signaling during natural repair and regeneration in the tooth may occur, also provides ideas for development of potential therapeutic intervention for tissue regeneration. In caries, the metabolic products of plaque bacteria, especially organic acids, will diffuse into dentin causing demineralization and degradation of the matrix. Dentin matrix is now recognized to contain a very heterogeneous mixture of non-collagenous protein (NCP) components, in addition to its predominant structural component, collagen.[10,36] Many of the NCPs will be acid-soluble leading to their release during carious demineralization and subsequent diffusion towards the pulp along the dentinal tubules. Even those NCPs, which are insoluble or incompletely released from the matrix, may still be exposed to contact and interaction with pulp cells at sites of injury in the tooth. The ability of soluble preparations of dentin NCPs to signal regenerative events in dentin-pulp[37] has allowed further investigation of the possible signaling role(s) of individual molecules in these more complex preparations.[38] As well as a diverse mixture of dentin specific and more ubiquitous matrix components, these dentin NCP preparations contain a rich cocktail of growth factors and cytokines with potent cell signaling properties. The growth factors and cytokines include several members of the TGF-ß superfamily, angiogenic, neurotrophic and other bioactive molecules.[39–43] Some of the dentin specific NCPs demonstrate previously unrecognized biological activities.[44] Others may well be found in the future.

Considerable progress has been achieved in identifying bioactive NCPs in dentin matrix preparations, but our understanding is still incomplete and other molecules with potential actions on regenerative processes may still be characterized. Importantly, the bioactive molecules sequestered within the dentin matrix can be considered to exist in a fossilized state since they are both immobilized their biological activities are protected by their interaction with the mineral phase of dentin. Thus, these bioactive molecules may be present in dentin into old age and may only be released after injury to the dentin leading to its partial or complete focal dissolution. However, opportunities exist for targeted release of some of these molecules for therapeutic purposes through the actions of irrigants, intracanal/cavity medicaments and various restorative materials.[45–50] Some similar bioactive molecules may also be present in dental pulp as a result of cellular activities.[51] However, pulp-derived bioactive molecules will likely have quite a high and more rapid turnover. Their presence in pulp may be relatively short-lived. Some growth factors have half lives of the order of minutes and thus, their continued presence during regenerative events will be dependent on continuing cellular activity responsible for their generation.

There has been considerable interest in correlating specific bioactive molecules with the various signaling steps involved in the recruitment, commitment and differentiation of odontoblast-like cells and other cellular events in dentin-pulp regeneration. It is clear, however, that networks of molecules may be involved in some of these signaling steps. Also, more than one molecule sometimes appears to be able to exert the same action. Whether the latter represents an example of biological redundancy or an as yet undiscovered nuance of signaling remains unclear. Several dentin and pulp matrix components, including growth factors, cytokines and matrix molecules, can stimulate pulp cell migration.[38,44,52,53] These molecules may be important for cell recruitment during regeneration.

Signaling odontoblast-like cell differentiation may show parallels to that occurring for primary odontoblast terminal differentiation during physiological tooth development with involvement of growth factors, especially of the TGF-ß superfamily.[13,54,55] Criteria for identification of differentiation of odontoblast-like cells as opposed to other cell phenotypes will often include expression of several genes (or their protein products) characteristic of odontoblasts and importantly, secretion of a tubular matrix. While valuable, the rigor of these criteria are such that some odontoblast-like cells may show aberrations from physiological odontoblasts as emphasized by the range of tubular structure seen in dentin

during reparative dentinogenesis. Regulation of odontoblast secretory activity is critical to maintenance of normal tooth and pulp chamber/root canal morphology. It is also fundamental to our concepts of the up-regulation of odontoblasts during reactionary tertiary dentinogenesis. TGF-ß1 has been implicated in the signaling of odontoblast secretory activity,[56–58] and the p38-MAPK pathway can mediate this signaling in tertiary dentinogenesis.[59,60] The p38 pathway may be a target for therapeutically controlling odontoblast secretory activity in pulp regenerative strategies. Signaling of the angiogenic events that accompany pulp regeneration may be achieved through the effects of growth factors and other proteins released from the dentin matrix after injury.[28,40,61]

ENVIRONMENTAL INFLUENCES (MICROBIAL AND INFLAMMATORY) ON REGENERATIVE EVENTS

With continuing carious injury to the tooth, microorganisms and their metabolic products penetrate dentin matrix and invoke cellular responses in the underlying pulp. Initially, these responses arise from diffusion of microbial products to the pulp, although in time, the microbial load will significantly increase and microorganisms may be found within the pulp itself causing further escalation of defense responses. The odontoblasts, with their peripheral localization in the pulp chamber, play important environmental sensing and defense roles in addition to their well-established matrix synthetic/secretory functions.[11,62–65] The significance of this localization is further enhanced by the extensive network of lateral processes, in addition to the main odontoblast cell process facilitating communication between the odontoblasts and dentin matrix.[66]

Microbial Sensing: Odontoblast sensing of microorganisms will invoke defense responses, including release of antimicrobial peptides and various cytokines and chemokines with autocrine and paracrine actions.[62,64–66] These defense responses, however, may be overwhelmed as disease progression increases and evidence for regeneration, seen as tertiary dentinogenesis, is limited to more slowly progressing carious lesions, but not in more aggressive, rapidly advancing lesions.[67–69] Microorganisms and their byproducts are detected by a family of pattern recognition receptor (PRR) molecules, called Toll-like receptors (TLRs),[70] that sense pathogen-associated molecular patterns (PAMPs). Of the 11 members of the TLR-family, TLRs 1 to 6 and 9 are expressed on odontoblasts and pulp fibroblasts, and can be envisaged to sense a range of oral microorganisms PAMPs.[63,71–73] Nucleotide-binding oligomerization domain (NOD) -1 and -2 proteins in the pulp also contribute to oral microorganisms sensing[74–76] and with the Nod-like receptor (NLR) pyrin domain containing 3 (NLRP3) molecule[77] can be assembled into multi-protein "inflammasome" structures, capable of detecting microbial and tissue danger signals.[78] These various microbial sensing responses will then trigger activation of several intracellular signaling pathways including nuclear factor-kappa B (NF-κB), p38 MAP-kinase and Jun N-terminal kinase (JNK).[62,63,71–73,76,79,80] The outcomes of these signaling pathways will be the release of inflammatory mediators in the extracellular milieu,[74,75] that will amplify the defense responses and contribute to the recruitment of immune cells.[81,82] The involvement of various immune cells, signaling mediators and defense responses reflects a complex interplay of events and our understanding of these interactions will continue to grow.

Modulation of the Pulpal Environment During Disease Progression

The combination of both direct microbial and indirect inflammatory/immune influences on the pulpal environment, provides significant opportunity for carious disease to impact on regenerative events. However, characterization of these influences is challenging since the intensity of microbial infection and inflammatory challenge may impact on responses, as well as the nature of the experimental model in which these influences are investigated.

At the cellular level, there are conflicting reports as to the effects of inflammation on dental pulp stem cell differentiation and regeneration.[83–85] Stem/progenitor cells have been isolated from inflamed dental pulps and their ability to participate in regenerative events *in vivo,* as examined in an animal model,[83] and a number of their properties *in vitro* appear similar to cells isolated from healthy pulp.[83,84] However, exposure of isolated dental pulp stem cells to TNF-α *in vitro* to mimic inflammation has been reported to impair their differentiation.[85] In contrast, LPS treatment *in vitro* has been suggested to promote regenerative events in pulp stem cells.[86–88] These various apparently conflicting reports may well reflect the experimental model systems in which the cells have been investigated. Isolation of these cells and their study using *in vitro* culture will place them in a growth environment that is significantly different from that *in vivo*, both in the absence of the cells' tissue extracellular matrix/niche environment and exposure of the cells to the complexity of microbial and inflammatory challenges, normally encountered in the inflamed dental pulp.

Such limitations are common to many *in vitro* cell culture studies where compromises in cellular environments have to be balanced against reproducibility, control of variables and feasibility of experimental models. Likewise, most studies of pulp regeneration *in vivo* have adopted experimental models lacking ongoing microbial/inflammatory challenge where regenerative events will likely differ from when such challenges persist. Clearly, there is a need to interpret reports of developments in pulp regeneration with care as we strive for clinical translation.

As well as effects on cells, microbes and inflammation will also act on the various molecular constituents of pulp resulting in their partial or complete degradation potentially influencing their functional activities. While such degradation might be predicted to negatively impact on function, this does not necessarily appear to be the case. Acid and

enzymatic breakdown of pulp and dentin extracellular matrix components enhanced their chemotactic activities and contributed to selective recruitment of stem/progenitor cells involved in reparative processes.[38] The effects of these partial degradation products of pulp and dentin ECM components on other regenerative events require further study to understand their relative importance to the cellular signaling milieu present in pulp after injury.

Inflammatory/Immune Response-Regeneration Interplay

Mesenchymal stem cells (MSCs) can modulate the proliferation and functions of various types of immune cells.[89] Pulp stem cells also demonstrate such immuno-modulatory properties.[90–93] Our understanding of the activation of these immuno-modulatory properties is still limited, but involvement of TLR agonists has been reported.[93] While TLRs are typically activated by PAMP sensing of microorganisms, they may also be activated by host ligands (e.g. heat shock proteins, fibronectin and fibrinogen) or synthetic agonist molecules,[94] reflecting the diverse and complex nature of environmental sensing by cells. Interestingly, it is becoming apparent that considerable interplay exists between inflammatory/immune and regenerative responses, especially during the early stages of disease progression, or later stages of its resolution when low-level cytokine stimulation may mediate events.[95] The cytokine, TNF-α, can activate p38-MAPK signaling,[96] the latter of which may be a key regulatory switch for tertiary dentinogenesis.[59,60]

Low levels of immune cell-derived cytokines and reactive oxygen species (ROS) can also promote dental stem cell differentiation and mineralization.[97,98] It appears that regenerative events are stimulated by lower levels of inflammation and impeded by higher levels of inflammation; such biphasic responses are well recognized in biology, being termed hormesis.[99] These correlate well with clinical and histological observations of disease progression in teeth.[67–69] Increasing evidence for this interplay between inflammation and regeneration emphasizes some of the challenges for the clinical translation of regenerative endodontics.[12]

REGENERATIVE STRATEGIES

The biological strategies underpinning Regenerative Endodontics are dependent upon those elements associated with normal tissue development and tissue engineering: cells, extracellular matrix/cellular scaffold and signalling molecules. Targeting of these elements has been approached in a variety of ways that can generally be categorized under the headings of cell-free and cell-based approaches.

Cell-free Approaches

Cellular synthesis and secretion of tissue components are fundamental to the formation of any tissue. However, even where localized areas of cellular necrosis exist within a tissue, its regeneration may be achieved without the need to directly transplant cells to the injury site. Natural tissue wound healing responses stimulate the recruitment of stem or progenitor cells to injury sites thereby facilitating wound healing. Cell lineage studies suggest that cell recruitment, from both tissue-resident and other sources, may occur in the pulp after injury.[17] Such recruitment of stem/progenitor cells also provides the cells for differentiation of odontoblast-like cells in reparative dentinogenesis for dentin bridge formation following direct pulp capping. Thus, cell-free regenerative approaches avoid some of the issues associated with cell-based therapies, including the generation of sufficient numbers of cells of the necessary clinical quality and sourcing cells that will not result in immune rejection. These cell-free strategies are dependent upon providing a conducive environment in which regeneration can take place and thus, as well as providing cellular signaling, they will also generally consider how the cell matrix or scaffold might be optimized. The provision of cellular signaling for regenerative strategies may involve the direct application of either tissue recombinant, synthetic or tissue-derived bioactive molecules. It may also be achieved through chemical treatment of the tissue, to release and expose endogenous fossilized stores of bioactive molecules sequestered within the dentin matrix.

The currently employed strategies in regenerative endodontics to treat immature teeth with pulp necrosis, constitute a good example of cell-free approaches. These procedures (discussed later) depend on stem cell transfer from the apical tissues into a disinfected root canal system. This transfer of cells has been commonly achieved by mechanically releasing cells from the apical tissues along with bleeding.[100] The blood containing the cells also works as a fibrin scaffold, favouring tissue formation. Another clinically used approach is the use of platelet-rich plasma or platelet-rich fibrin as scaffolds.[101,102] These two natural scaffolds contain various chemotactic factors capable of recruiting undifferentiated mesenchymal stem cells.[103]

Cell-based Approaches

Cell-based regenerative strategies will include the transplantation of cells to the site of injury in the tissue to promote regeneration. Clearly, such cells should be of the highest good manufacturing practice (GMP) quality and ideally, should be from autologous sources to minimize any risks of immune rejection. These requirements and the necessity for appropriate clinical facilities to handle these cells, provide a number of challenges, and in the current context of endodontic practice, cell-based therapies are only likely to be undertaken in a hospital setting. As well as the direct transplantation of cells, these cell-based approaches may also involve the application or local release of signalling molecules and include the use of cell matrix/scaffolds.

TRANSLATIONAL STUDIES IN REGENERATIVE ENDODONTICS

The pulp-dentin complex is a tissue with intricate morphology and composition. This highly specialized tissue

has very unique characteristics compared to other tissues in the body. These include exquisite nociceptive innervation, a rich capillary network and highly specialized cells such as odontoblasts and fibroblasts with pleiotropic functions. As stated above, all of these cell types are active participants in the process of repair and regeneration following injury. The understanding of the complex interaction among these different cells, within the microenvironment of an infected root canal, remains a major gap in knowledge.

Translational science can be defined as the multidirectional integration of basic science and patient-oriented clinical research and practices, with the long-term goal of improving clinical procedures and practice to promote public health. This crucial cross-talk between bench and clinical research requires a focused multidisciplinary effort in order to move the field of regenerative endodontics forward. Indeed, there has been a substantial increase in translational studies aimed to understand and improve the biologic processes associated with the current procedures while paving the way for future generation regenerative endodontic procedures. This fertile area of endodontic research has gained substantial momentum, and has resulted in several improvements to vital pulp and non-vital pulp therapies. These studies often used models that mimic important aspects and challenges seen in the clinical setting. These models include: in-vitro, ex-vivo, and in-vivo animal models.

In-vitro Models

Tissue engineering involves the combination of stem-cells, growth factors and scaffolds. In the context of regenerative endodontics, these three important components of tissue engineering must interact with a complex array of molecular signals such as attachment molecules and local growth factors, antigens, residual microorganism-derived molecules and inflammatory mediators. Researchers often rely on *in-vitro* experimentation to test and evaluate these complex interactions.

Various MSCs have been used in regenerative endodontic research including dental pulp stem cells (DPSCs),[22] stem cells from human exfoliating deciduous teeth (SHEDs),[26] and stem cells from the apical papilla (SCAP).[25,104] In addition, mixed populations of cells have also been used such as cultured dental pulp cells,[105] that represent a heterogeneous population of cells that include MSCs.

It is important to emphasize that the characterization of "true" MSCs is still controversial. Surface markers such as cluster differentiation molecules (CD) have been extensively used to characterize different population of MSCs.[106] However, their expression alone is not often sufficient to ascertain the cellular identity. The potential use of these cells in cell-based therapeutics demanded the establishment of minimal criteria for the identification of these cells. In 2009, Mesenchymal and Tissue Stem Cell Committee of the International Society for Cellular Therapy established that MSCs are characterized by adhering to plastic in culture, the expression of CD73, CD90 and CD105, while lacking the expression of CD45, CD34, CD14 or CD11b, CD79 alpha or CD19 and HLA-DR, and the differentiation potential into adipocytes, chondrocytes and osteocytes in culture.[18]

As discussed previously, care should be employed when directly translating information from *in vitro* studies to the clinical practice since stem cells in culture lack the environmental cues that govern their fate, such as attachment molecules and growth factors. The culture of these stem cells in different *ex-vivo* biological environments has been used by researchers to mimic physiological conditions and minimize shortcomings of *in vitro* experimentation alone.

Ex-vivo Models

The combination of stem cells with the correct composition of scaffolds, and mixture of growth factors that most commonly resemble the conditions in tooth regeneration is daunting. Instead, millions of teeth are extracted every year offering ample opportunity for whole teeth (e.g., whole tooth culture) or parts of the tooth (e.g., root segments used in orgnotype root canal models). In addition, tissues can be harvested from animals and maintained in culture allowing for experimentation in tissues that more closely resemble conditions found in the body.

The culture of a whole tooth was reported by Tecles et al.[107] and allowed for the evaluation of stem cells response in the human dental pulp to a direct pulp capping with either MTA or calcium hydroxide. Stem cells or progenitor cells proliferated and were recruited to the site of injury (capped pulp exposure) and formed a mineralized matrix over the exposed pulp, similar to the mineralized bridge formed in direct pulp capping procedures, while the tooth was maintained suspended in culture for 21 days.

This elegant model allows for the examination of cellular responses in their niches with the correct composition of scaffolds (attachment molecules) and growth factors and a plethora of cell-to-cell interactions, although such responses occur in the absence of more systemic influences that exist *in vivo*. This same model was later used to demonstrate that Biodentin, similarly to MTA, allowed for the formation of a mineralized barrier over the capped pulp, and that this process was mediated at least in part through the release of TGF-beta 1 from the adjacent pulp cells.[108]

The *ex-vivo* culture of tissues can become increasingly complex, including sectioned jaws containing teeth still in their natural niche without the added trauma of extraction.[109] This model was developed to test the regenerative potential of teeth while allowing their interaction with cells and growth factors from the periradicular tissues. It facilitates the evaluation of the impact of immune reactions and microorganisms on the repair and regeneration of the pulp and periradicular tissues.[110,111]

Organotype root canal models have also been employed. These models are composed of either tooth slices (e.g., transection of pulp chambers),[28] or prepared root canals in root segments of extracted teeth.[112] The addition of stem cells

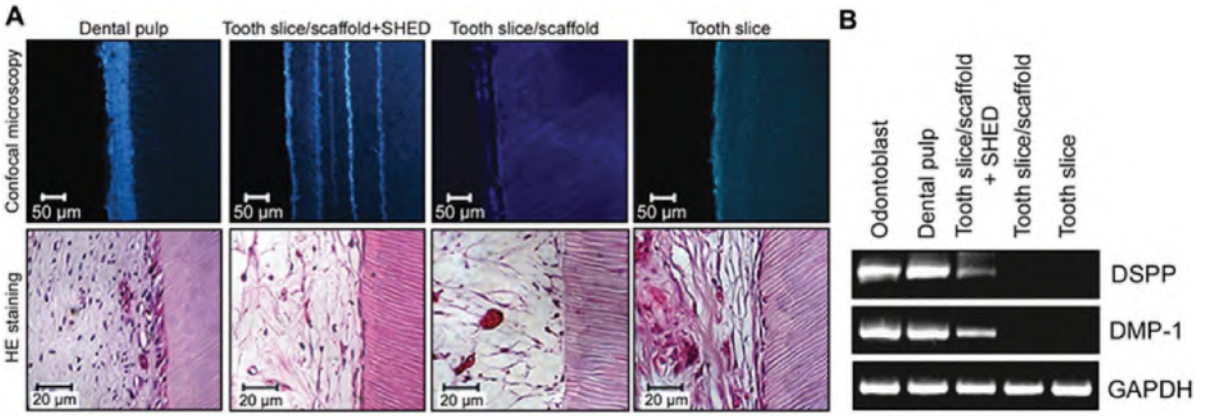


FIGURE 29-2 SHED differentiate into functional odontoblasts that generate new tubular dentin *in vivo*. ***A.*** Photomicrographs of confocal microscopy (top row) and hematoxylin and eosin staining (bottom row) performed in specimens retrieved from immunodeficient mice 32 days after implantation. ***B.*** RT-PCR analysis of DSPP and DMP-1 mRNA expression on tissues retrieved from the pulp chamber of tooth slices from the four experimental groups (i.e., tooth slice/scaffolds seeded with SHED, negative control tooth slice/scaffolds without SHED, negative control tooth slices without SHED and without scaffold, and positive control tooth slices with pulp tissue) 32 days after transplantation into immunodeficient mice. Positive controls were odontoblasts retrieved from freshly extracted human third molars. (Adapted with permission from Sakai et al.[118])

in a suitable scaffold to these "dentin culture environments" allows for the *in vitro* evaluation of their interaction, being particularly important for the investigation of the effect of conditioning dentin with different irrigants and medicaments on stem cell fate.[49,112–114] A study found that SCAP in PRP placed in root canals previously treated with either 6% NaOCl or irrigant combinations that contained 2% chlorhexidine did not survive in culture. However, they thrived if the dentin had been treated with 17% EDTA.[112] Another study, using a tooth slice model found that dentin treatment with 6% NaOCl prevented the differentiation of SHED into an odontoblastic phenotype when cultured in contact with the conditioned dentin. Conversely, dentin conditioning with 17% EDTA increased the expression of odontogenic markers.[114] These studies highlight the value of these models to test clinically relevant questions.

In vivo/Animal Models

The use of animal models of regeneration is a natural transition from the *in vitro* and *ex-vivo* evaluation of mechanisms and interactions between stem cells, growth factors, and scaffolds. Animal model experiments can involve the use of the animal's own dentition as a regenerative model,[25] or the implantation of "constructs" of human teeth (whole of fragments) seeded with stem cells in a suitable scaffold.[50,115]

The severely compromised immune deficient (SCID) mouse is a murine strain that lacks functional T cells and B cells, among other immune deficits. These animals accept allografts and xenografts with minimal to no rejection risk. Thus, they have been widely employed as hosts for the implantation of human teeth[53] and human stem cells.[116] Extracted immature third molar implanted subcutaneously into the dorsum of SCID mice demonstrated continued dental development with new dentin formation and no rejection or immunogenic reaction to the xenograft.[117] Tooth slices with SHED in a tooth slice/scaffolds were implanted subcutaneously in SCID mice injected periodically with tetracycline allowing formation of a pulp-like tissue after 32 days with cells that expressed the odontogenic markers dentin sialophosphoprotein (DSPP) and dentin matrix acidic protein-1 (DMP-1).[118] Importantly, de novo tubular dentin formation was visualized by tetracycline staining seen under confocal microscopy (Figure 29-2). Thus, the use of SCID mice as a host or "living bioreactor" allows for direct evaluation of tissue engineering approaches that are conducive for regenerative endodontics.

Another translational model involves procedures and techniques tested in animal models of regenerative endodontics. These procedural experiments usually employ larger animals, such as dogs,[119–123] minipigs[25] and ferrets that allow for better simulation of conditions and procedures more applicable to patients. Revitalization or revascularization procedures performed in dogs have demonstrated successful healing of apical periodontitis, and radiographic root development.[119,120,123] However, the tissues formed in these procedures did not resemble the native pulp tissue and the mineralized tissue resembled cementum or osteoid tissues. It is important to note that these findings closely resemble those found human teeth treated with these techniques (discussed later in this chapter). This strong correlation suggests that these models are valid and important for the investigation of treatment modifications that could become the next generation of regenerative endodontic procedures.

REGENERATIVE ENDODONTIC PROCEDURES (REPS)

Patients undergoing cranio-skeletal development with mixed dentitions are at risk of pulpal injury. Permanent teeth erupt in the oral environment with a very immature or undeveloped root structure. Upon eruption, teeth are exposed to a new

environment that contains microorganisms and is susceptible to traumatic injuries. It takes approximately up to three years for a root to be fully formed from the time of the first signs of eruption[124]. Trauma to immature teeth has been recognized as the primary etiology for pulpal necrosis in these teeth followed by dental anomalies (dens evaginatus for example).[125] Unfortunately, trauma to permanent immature teeth is a relatively common occurrence affecting approximately 30% of all children.[126] Despite an elaborate pulpal defense system that includes specialized dendritic cells,[127] odontoblasts,[65] fibroblasts,[65] neurons[128] and a vascular arteriovenus (A/V) shunt mechanism,[129] the dental pulp may succumb to the ingress of microorganisms and/or ischemia following trauma. The subsequent liquefaction necrosis of the dental pulp arrests the dental development leaving immature teeth with thin, fragile dentinal walls and open "blunderbuss" apices. For more details, see Chapter 12, "Endodontic considerations in dental trauma."

For many decades, the management of many such cases had been considered unpredictable. Tooth loss in patients still undergoing cranio-skeletal development has 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.[130,131] Since implants are contraindicated in growing young patients, tooth replacement is not possible until they mature (usually >18 years old).

Until recently, the clinical treatment of immature teeth with pulpal necrosis applied the prevailing traditional philosophy of disinfection, followed by providing a "microbial-tight" seal of the root canal system with an inert material such as gutta-percha. From a biological perspective, this approach is not without challenges since the large root canals found in immature teeth harbor high microbial loads. In addition, these microorganisms penetrate further into dentinal tubules in immature teeth than fully formed teeth, making disinfection a harder goal to be achieved.[132] Conventional root canal treatment relies on chemo-mechanical debridement for the removal of planktonic microorganisms and biofilms in the infected dentinal surface. This approach, that includes the mechanical removal of dentin, is challenging, since these canals have greater diameter than most available instruments, and its practice is ill advised in immature teeth with thin dentinal walls, that are also prone to fractures. Furthermore, adequate obturation to achieve a microbial-tight seal is challenging due to the large canal volume, inverted taper (wider apically) and lack of apical constriction.

Apexification procedures were introduced in the United States by Frank in 1966, who demonstrated that an apical calcific barrier may be formed in immature teeth following adequate debridement and intracanal placement of calcium hydroxide.[133] Several studies demonstrated successful outcomes, that used the dependent measure of resolution of the disease process, and the formation of a calcific barrier (apexification), following the use of calcium hydroxide for several months.[134–136] Furthermore, it became clear that the formation of this natural calcific barrier depended on the arrest of the infection and inflammatory process.[137] Therefore, when this approach is employed, clinicians commonly use calcium hydroxide as an intracanal dressing until symptoms are resolved, and the apical barrier is formed, a process that may take upto 18 months.[137] Calcium hydroxide has been shown to reduce tooth resistance to fractures in a time dependent-fashion,[138] with significant decrease occurring with its use for longer than 3 months.[139] Indeed, teeth treated using calcium hydroxide-induced apexification have been found to show greater rate of fractures and reduced survival.[140,141]

The introduction of mineral trioxide aggregate (MTA) in 1993 addressed several unmet needs in endodontics, allowing its use in root perforations, capping procedures and apexification procedures.[7,142] It was recognized at an early stage that MTA was not only biocompatible but also, it induced the "regeneration" of mineralized tissues upon direct contact. Indeed, MTA predictably induced the formation of a calcific barrier in dogs demonstrating that it could be used as an osteo-inductive artificial barrier or "apical plug."[143] These findings, in conjunction with many successful reports of "osseous regeneration" induced by MTA, quickly accelerated its use in apexification procedures. The possibility of sealing the apical foramen with MTA revolutionized the treatment of immature teeth with pulpal necrosis since the procedures could be performed in a single appointment, or short-term in multiple appointments with similarly high success rates, without any concern of apical extrusion of the material.[144–146] Although MTA apexification procedures allowed for resolution of symptoms, and did not further weaken teeth following the long-term use of calcium hydroxide, they did not promote continued root development and reestablishment of pulpal functions (discussed later in this chapter).

Regenerative endodontic procedures emerged in 2001 as a case report demonstrating striking evidence of root development, and regain of responses to vitality testing, following a procedure that involved the use of an intracanal antibiotic paste containing metronidazole and ciprofloxacin (later called double antibiotic paste) (Figure 29-3).[147] This case report was followed by another noteworthy report demonstrating similar results with the use of a triple antibiotic paste as an intracanal dressing (Figure 29-4).[148] Importantly, the biologic foundations and philosophy for these procedures were established many decades previously with the pioneering work of Dr. Nyggard-Ostby,[149] that aimed to investigate the role of the blot clot in healing following endodontic infections. In his seminal studies, it was observed that reestablishment of a vital tissue within the root canal spaces was possible when apical blood was allowed to ingress into a disinfected root canal. In these studies, the included teeth had mature, fully formed roots, and canals were disinfected without consideration for making the environment hospitable for the cells, including stem cells (not yet identified at that time). Nonetheless, remarkable results were found that included: 1) resolution of symptoms of inflammation related to foraminal enlargement and over-instrumentation, 2) resolution of signs and symptoms of pathosis for the necrotic cases, 3) radiographic evidence of apical closure, and 4) ingrowth of connective tissue into the canal space, and varying deposition of mineralized tissue found along the canal walls, as well as "islands" of mineralized tissue embedded within the newly

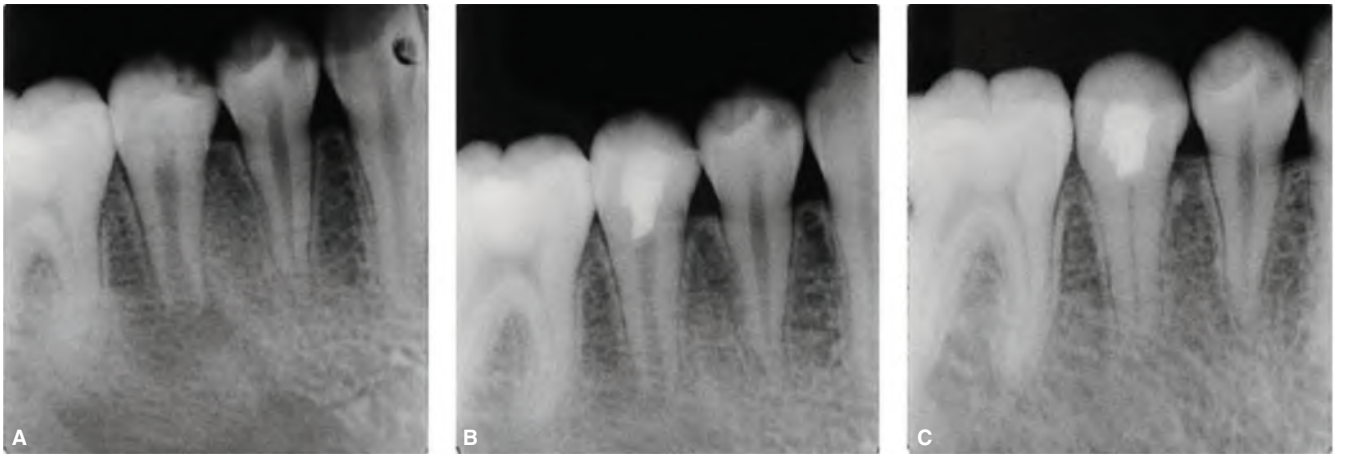


FIGURE 29-3 A case treated with a regenerative endodontic procedure using double antibiotic paste (ciprofloxacin and metronidazole) as intracanal medicament. ***A.*** The immature mandibular second premplar was diagnosed with pulpal necrosis and acute apical abscess. ***B.*** Five-month follow-up reveals complete resolution of apical radiolucency and formation of a coronal mineralized bridge and thickening of the dentinal walls. ***C.*** 30-months follow-up reveals completion of root development with apical closure, thickening and elongation of root structure. (Adapted with permission from Iwaya et al.[147])

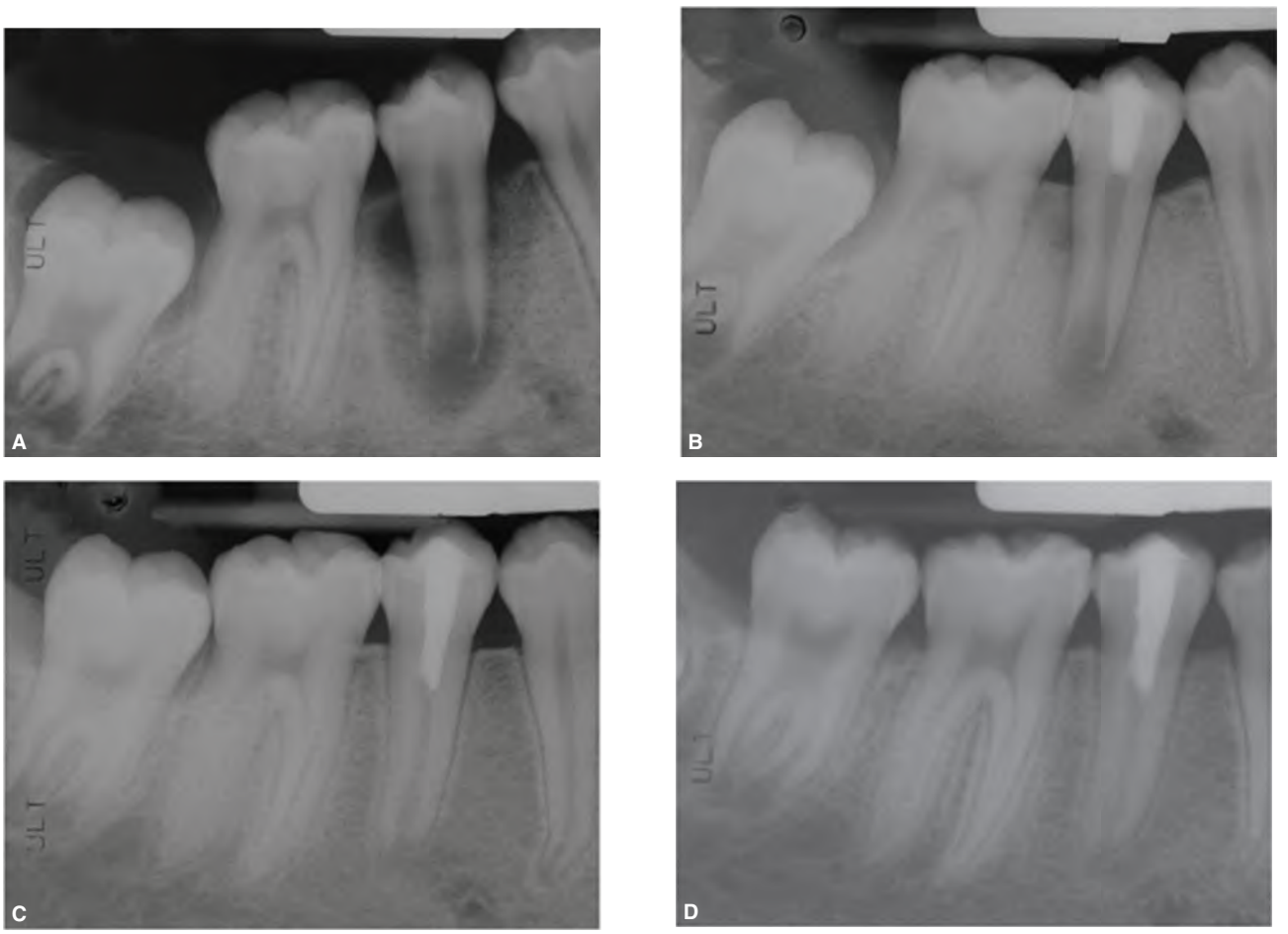


FIGURE 29-4 An 11-year-old male patient was diagnosed with pulpal necrosis and chronic apical abscess in the mandibular second premolar. The tooth was treated with a regenerative endodontic procedure (revascularization) using triple antibiotic paste (minocycline, ciprofloxacin and metronidazole) as intracanal medicament. ***A.*** Preoperative radiograph shows large periradicular radiolucency and immature root with open apex. ***B.*** Post-operative radiograph taken 26 months after treatment showing signs of healing; ***C.*** 12-month follow-up showing complete resolution of radiolucency and continued root development. ***D.*** 18-month follow-up showing further root development. The patient remained asymptomatic during the follow-up period and the tooth responded to both electric and cold sensitivity tests. (Adapted with permission from Banchs and Trope.[148])

formed tissue. Although some studies followed this thought-provoking initial report, the exploration of biologic-based endodontic therapies was not prioritized for many decades until the surge of the contemporary regenerative endodontic procedures (REPs) in the early 2000's.

COMMON FEATURES OF REGENERATIVE ENDODONTIC PROCEDURES

Since the initial case report in 2001, more than 200 cases have been reported in the scientific literature.[125,150] Despite pronounced variations in clinical protocols, some common features can be identified in these procedures, regarding case selection, disinfection strategies, evoked-bleeding and restorative approaches. Although most of these reports consist of case reports, they provide immediate documentation of procedures that may or may not have worked in patients, while providing crucial feedback to researchers for the development of future REPs. Therefore, it is important to highlight some of the features present in most REPs.

Case Selection

Trauma to the permanent developing dentition is the underlining etiology of approximately 49% of all the published cases using REPs as therapy.[125] This finding is not surprising, since it is consistent with the reported high incidence of dental trauma affecting up to 35% of children,[126] particularly between the ages of 7 and 15 years old,[151,152] when most permanent teeth are in an incomplete stage of root development. Unfortunately, approximately 50% of the traumatized teeth may be diagnosed with pulpal necrosis,[153] with greater incidence occurring following severe injuries such as intrusions and avulsions[154,155] and combination injuries.[156–158] Therefore, the high incidence of trauma-evoked pulpal necrosis reported in the patients included in case reports and retrospective and prospective studies in regenerative endodontics is likely biased by the high incidence of these injuries. Moderate to severe trauma to the developing dentition can potentially damage the Hertwig's epithelial root sheath (HERS), that is crucial to dictating root formation and maturation by directing the concerted proliferation and differentiation of MSCs.[159] However, the effects of the etiology (trauma versus dens evaginatus, for example) on the outcomes of REPs are still unknown. This is largely due to the relatively small sample sizes of the existing studies, constraining statistical analysis of outcome predictors, and importantly, contributing to the prevailing confusion on what should be the expected outcomes of these procedures (discussed later).

The second most common etiology is the presence of an existing dental anomaly that renders the developing tooth susceptible to pulpal necrosis. Dens evaginatus is considered a prominent etiologic factor for pulpal necrosis in 33% of all the reported cases, followed by dens invaginatus (3% of the cases).[125] Dens evaginatus is manifested as an additional cusp, typically projecting into the occlusal table of a mandibular premolar (more common) or the facial or lingual surfaces of maxillary anterior teeth (less common). The incidence of dens evaginatus has been reported to affect up to 6% of the population with greater incidence in certain ethnic groups.[160,161] Although the incidence of this dental anomaly is relatively rare, its presence is often associated with the fracture or wear of the enamel-dentinal tubercle leading to the direct exposure of the pulp to the oral environment, and the rapid development of pulpal necrosis in immature teeth.

Most published regenerative endodontic cases, but not all,[162] report treatment outcomes in immature teeth with an open apex.[125] These teeth have been shown to have greater chance of becoming "revascularized" upon reimplantation. Indeed, the clinical outcome of these reimplanted teeth appears to be directly related to the degree of maturity based on the stage of root development and apical closure with more desirable outcomes being seen in teeth with a radiographic apical diameter of at least 1 mm.[163] Although the process of tissue formation in regenerative endodontics greatly differs from the process of revascularization of an existing sterile pulp as seen in reimplantation, it equally relies on angiogenesis and the ingress of newly formed blood vessels through the apical opening.[164,165] It is probable that an immature tooth with a wide apical opening shows a much greater vascularity than seen in the roots of more mature teeth, and this could have significant influence on regenerative endodontic treatment outcomes.[166]

It is important to note the effects of stage of root development and apical diameter on clinical outcomes have never been established. Interestingly, a recent study found that vascularized tissue could be formed in teeth with an apical diameter of approximately 0.3 mm, that is similar to the apical diameter of most canals in fully mature roots.[167,168] In summary, most REPs have been performed in immature teeth, but the minimum clinical critical apical diameter for successful outcomes has not been established. A recent animal study and a case report challenge the preconceived notion that an apical diameter of greater than 1 mm is a requirement for desirable outcomes in REPs.[162,167]

Clinical Protocol

There are more than 200 published cases of REPs. Notably, these published cases are marked by a tremendous heterogeneity in the clinical protocols used. This is largely due to the empirical nature of the protocols adopted in the original reports that preceded the more translational studies aiming to provide a more targeted approach to REPs. The American Association of Endodontists (AAE) have established a long-standing committee that periodically evaluates both clinical and translational studies to help establish clinical considerations and guidelines for regenerative endodontics.

Despite this heterogeneity in the treatment protocols reported in published cases, some common features can be readily seen. Most cases have been performed with minimal to no instrumentation and with the use of copious irrigation with sodium hypochlorite as a means of disinfecting and debriding the root canal space. Intracanal medicaments were used in all cases with exception of one published case report.[169] On the last visit, the canal was commonly irrigated once more, dried and the intracanal bleeding evoked by instrumentation of the apical tissues.

Certain cases were performed without intracanal bleeding and with its substitution by the application of platelet rich plasma (PRP)[102] or platelet rich fibrin (PRF).[170] The use of intracanal bleeding represents an important step in these procedures since it is capable of transferring a substantial number of undifferentiated MSCs into the root canal system[100] and the blood clot can act as a scaffold. Both PRF and PRP have also been found to serve as scaffolds and provide chemotactic factors that are likely to promote MSC recruitment into the root canal system.[171]

Another important step seen in most published cases is the coronal placement of a bioactive "coronal plug" using pro-mineralizing dental materials such as MTA or Biodentine. These materials have been shown to promote the differentiation of MSCs into an odontoblast-like phenotype while allowing for greater proliferation of MSCs.[8,107,108,172] Importantly, these materials provide a "safe zone" barrier to the restorative material used to seal the pulp chamber that is typically noxious to stem cell survival and differentiation. Interestingly, there have been case reports of REPs performed without the use of these materials with acceptable clinical outcomes.[150] Therefore, the role of the bioactive coronal plug in promoting the proliferation and differentiation of MSCs through the length of the canal is not fully understood. Nonetheless, in certain cases a calcific barrier can be seen immediately underneath the material, suggesting that these materials induce the formation of at least a biological coronal seal.

Common Challenges

The clinical techniques usually employed in REPs are not without challenges. The diameter of the root canals in most immature teeth far exceeds the diameter of the larger endodontic instruments. This makes mechanical instrumentation technically challenging. In addition, the mechanical preparation of immature teeth could further weaken already fragile thin dentinal walls.[140] Due to these limitations, most cases are treated without instrumentation. However, bacterial biofilms have been found in a failed regenerative endodontic procedure.[173] Clinicians rely on the disinfection and tissue dissolution properties of sodium hypochlorite for debridement followed by placement of intracanal medicaments. Resistant bacterial biofilms represent a "reservoir" of continuing microbial insult that could jeopardize treatment outcomes. Therefore, it is prudent to lightly brush circumferentially the canal walls with endodontic instruments (e.g., Hedstrom files) to aid in the disruption of the bacterial biofilms without any aggressive removal of dentin. The use of ultrasonic activation of irrigants is warranted in these cases as long as the instrument is positioned at least 2 mm from the radiographic apex to prevent direct damage to the apical tissues. Lastly, the inclusion of a final irrigation with 17 % EDTA for approximately two minutes in the first visit, opens dentinal tubules and allows for greater antimicrobial effect of the intracanal medicament. EDTA treatment may also contribute to release and exposure of endogenous bioactive signaling molecules in the dentin that can promote regenerative events (Figure 29-5).

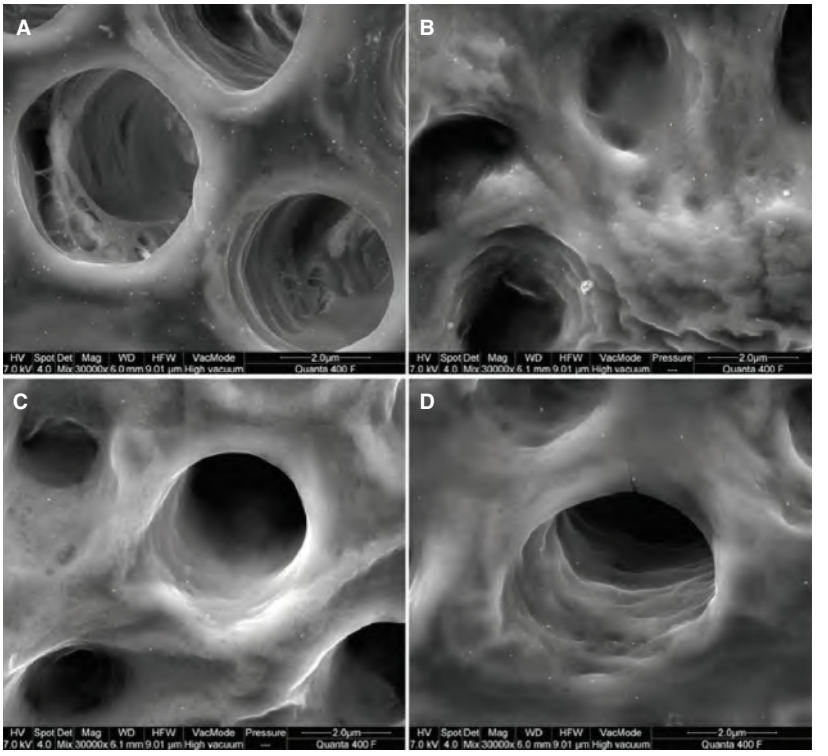


FIGURE 29-5 Gold labeling on dentin disks after EDTA conditioning (10%, pH 7, 10 minutes) of ***A.*** collagen; ***B.*** TGF-β1; ***C.*** FGF-2; and ***D.*** VEGF. *Dots* indicate gold nanoparticles after antibody binding that show growth factor exposure on the dentin surface. (Adapted with permission from Galleret al.[50])

Tooth discoloration has been identified as an adverse event associated with many REPs.[174,175] Tooth discoloration may have three origins in these procedures. The most common origin of discoloration is the intracanal use of tetracycline-like drugs, such as minocycline commonly present in the triple antibiotic paste (minocycline, ciprofloxacin and metronidazole).[176] The staining evoked by this antibiotic combination is often severe,[177] but it can be minimized by occluding the coronal dentinal tubules with a dental adhesive, or completely prevented by the use of calcium hydroxide as an intracanal medicament instead.[177–180] The second cause of coronal discoloration is the intracanal bleeding that is a part of the currently recommended protocols. Blood when present in the dentinal tubules, as often seen following trauma, is capable of causing coronal discoloration.[181] Paradoxically, blood should ideally be encouraged to flow as coronally into the pulp chamber as possible, and not below the CEJ as suggested and promoted in most published protocols. The placement of a coronal plug of a bioactive material (e.g., MTA or Biodentine) at the CEJ prevents root development in this region that is crucial for providing cervical strength to the tooth.[182,183] The application of dentin bonding agent to the dentinal tubules of the pulp chamber allows for placement of blood above the CEJ while minimizing the chances of blood-induced discoloration. The third possible cause of discoloration is the use of MTA as a "coronal plug" that often causes substantial discoloration, even when the white form of the material is used.[184–187] This undesirable results can be prevented by the use of other materials such as tricalcium silicate (e.g., Biodentine) in esthetic zones.[188,189] 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.[176,190] Therefore, the root canal system should be temporarily sealed with a sterile sponge and a temporary filling material, such as Cavit, followed by etching and bonding of the pulp chamber with a dental adhesive prior to placement of intracanal medicament to minimize the possibility of discoloration from the 3 sources (medicament, bleeding and restorative material).

Inadequate intracanal bleeding has been identified, as another challenge to REPs. The clinician should refrain from using vasoconstrictor-containing local anesthetics in the second visit to minimize challenges associated with achieving adequate bleeding into the tooth. Mepivacaine without epinephrine is a commonly used alternative to other dental local anesthetics containing vasoconstrictors. However, it is important to appreciate that unlike most local anesthetics that have robust vasodilator effect, it evokes moderate vasoconstriction by itself.[191,192] Conversely, lidocaine has been recognized as a potent vasodilator.[193] The use of lidocaine without epinephrine is not expected to provide long-lasting anesthesia due to its high rate of absorption by the systemic circulation.[194] However, its use minutes before the attempted apical bleeding may increase the chances of having adequate bleeding from the apical region. Although not ideal, clinicians may elect to place the bioactive coronal plug more apically if bleeding was found to be insufficient and not reaching the desirable level (above the CEJ) (Figure 29-6). Alternatively, PRP or PRF could be used after intracanal bleeding attempts are unsuccessful, as discussed above. Unfortunately, the use of these alternative scaffolds is not trivial since it requires drawing intravenous blood in young patients followed by laboratorial preparation of the bioactive scaffold.[101,170,195,196]

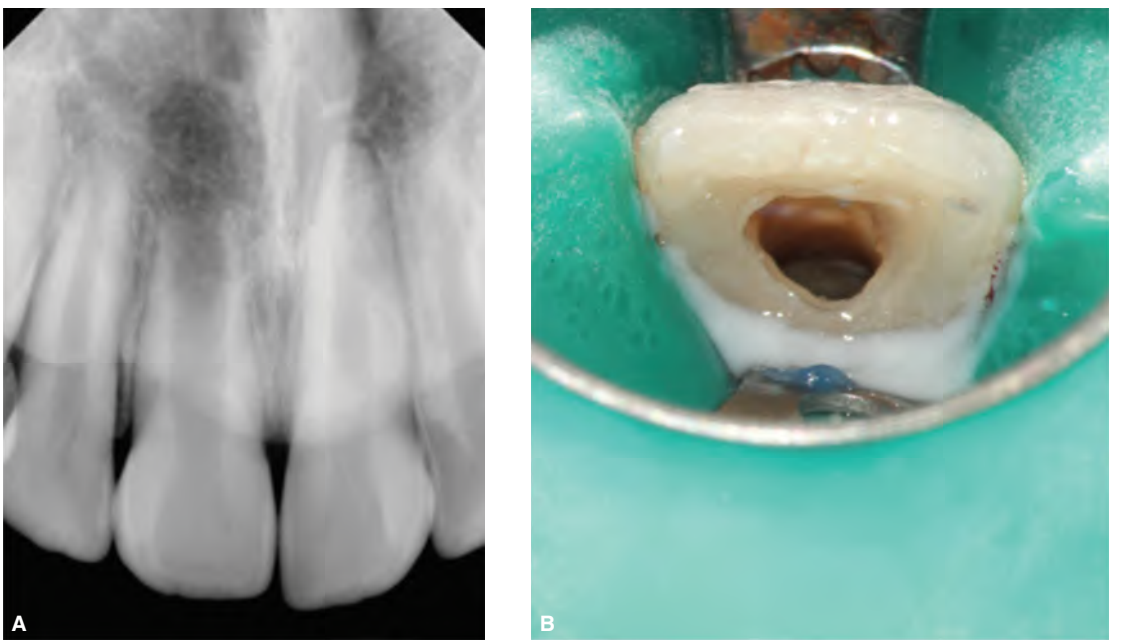
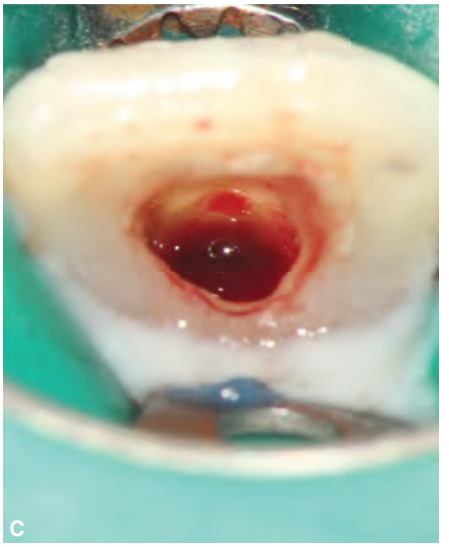
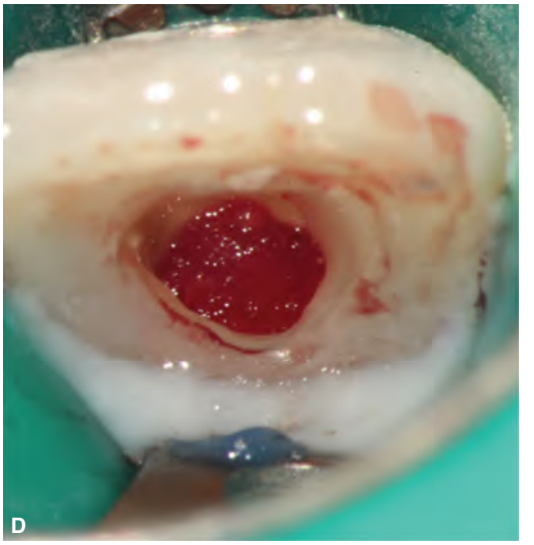
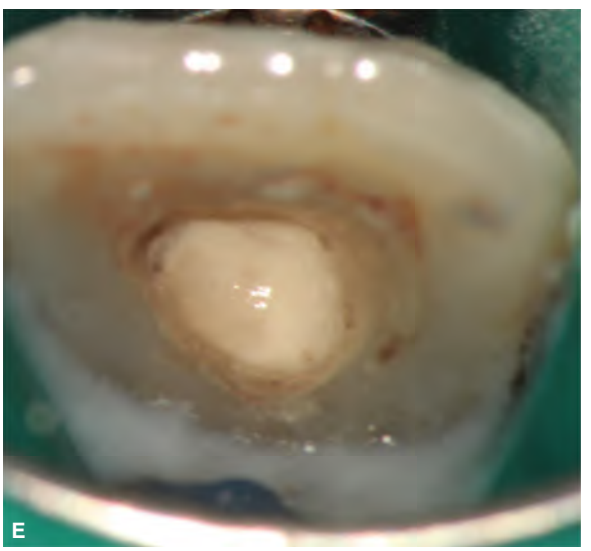
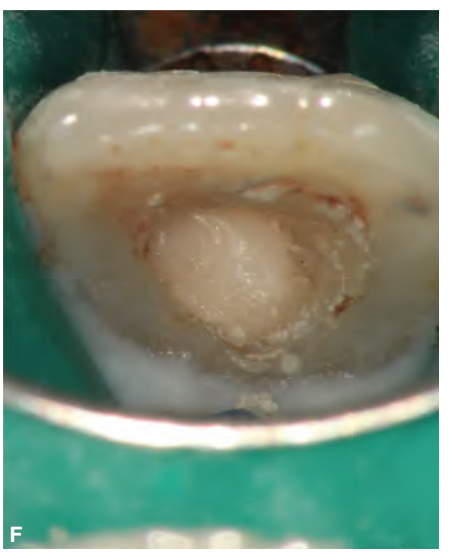


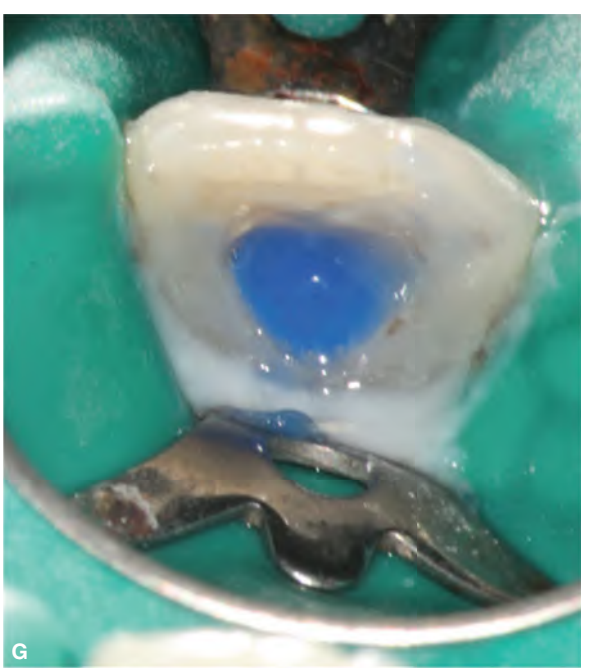
FIGURE 29-6 (*Continued*)











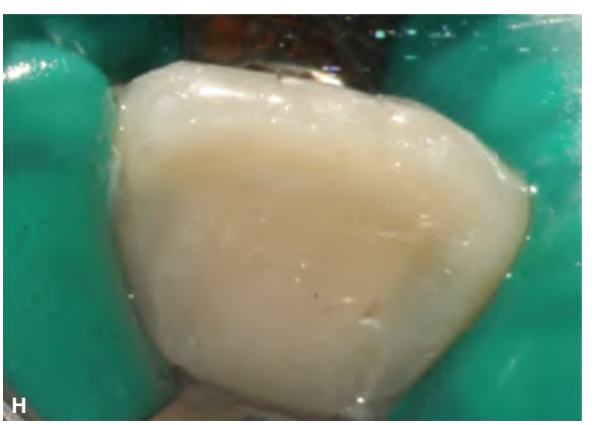


FIGURE 29-6 (*Continued*)

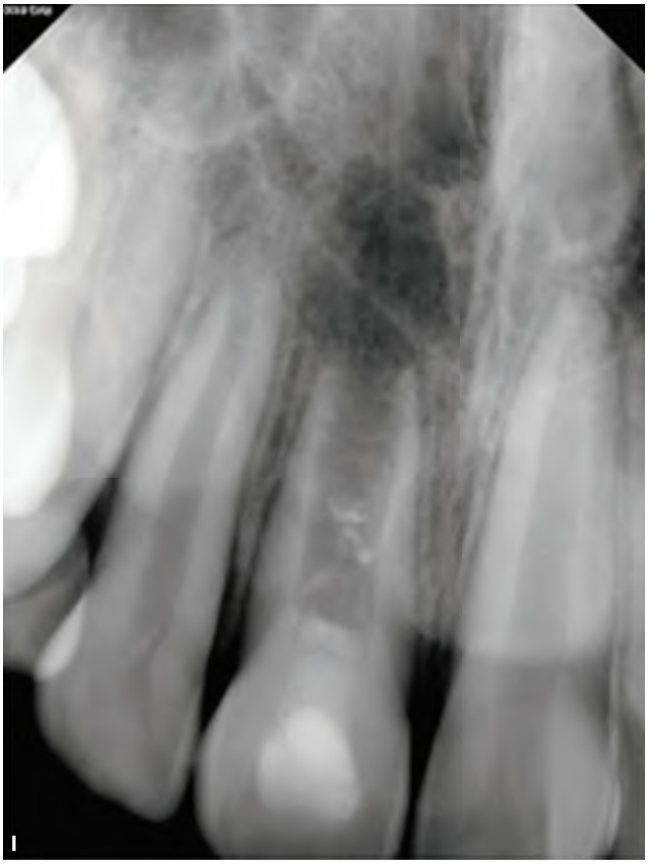


FIGURE 29-6 A 12-year-old female patient presented with the maxillary right central incisor diagnosed with pulpal necrosis and acute apical abscess due to a lateral luxation injury 2 years prior to the appointment. ***A.*** Preoperative radiograph revealed an immature root with thin dentinal walls and open apex. Rregenerative endodontic procedure was performed with placement of calcium hydoxide as intracanal medicament for one month. At the second vist, the patient was asymptomatic without swelling or sinus tracts. ***B.*** Several attempts of evoking intracanal bleeding were unsuccessful with blood only coming to the apical third of the root. ***C.*** Lidocaine 2% without epinephrine (1 mL) was administered as buccal infiltration injection and intracanal bleeding was attempted after five minutes producing profuse bleeding into the canal. ***D.*** Collaplug™ (Zimmer Dental Inc, Warsaw, IN, U.S.A) was placed over the blood clot followed by ***E.*** A 3-mm layer of Biodentin (Septodont, France) and ***F.*** 2 mm of Fuji IX (GC America, Alsip, IL, U.S.A.) ***G.*** The access was then etched with 35% phosphoric acid (Ultra-etch®; Ultradent Products Inc., U.S.A.) and ***H.*** Restored by a bonded composite restoration (Filtek Supreme Ultra; 3M ESPE, U.S.A). ***I.*** Post-operative radiograph. (Courtesy of Dr. Anibal Diogenes, San Antonio, Texas, U.S.A.)

Example of a Clinical Protocol

The field of Regenerative Endodontics has seen substantial advances in our understanding of the biological processes that provide explanation for clinical findings, such as radiographic continued root development and vitality responses.[125] Translational research has been crucial to this gain in knowledge, especially emphasizing that the disinfection process to be employed in regenerative procedures needs to be fundamentally different to that employed for conventional nonsurgical root canal treatment. Chemicals and medicaments must be chosen and have their concentrations adjusted to provide the greatest possible antimicrobial effect while creating a microenvironment conducive for stem cell survival, proliferation and differentiation. The technical recommendations below represent the current status of these procedures taking into consideration clinical and translational scientific findings. These recommendations are likely to change as the new field of regenerative endodontics rapidly evolves.

The Initial Visit

First Treatment Visit

1. Patient's informed consent, including explanation of risks and alternative treatments or no treatment.
2. After ascertaining adequate local anesthesia (2% Lidocaine with epinephrine), dental dam isolation is obtained.
3. The root canal systems are accessed and working length is determined.
4. The root canal systems are slowly irrigated first with 1.5% NaOCl (20 ml/canal, 5 min). The irrigant is then aspirated from the canal space.
5. Canals are dried with paper points and temporarily sealed with a sterile sponge and a thin layer of temporary filling material, such as Cavit or IRM at the CEJ level. The pulp chamber is etched, primed and bonded with a dental adhesive.

6. The temporary filling material and sponge are removed, and the canals irrigated with 17% EDTA (10 ml in 2 min). The canals are then dried, and calcium hydroxide, or antibiotic paste at a combined concentration of antibiotics of no greater than 1 mg/ml is delivered to canal system.
7. Access is temporarily restored.

Final (Second) Treatment Visit

(The second visit is scheduled 2 to 4 weeks after the first visit)

1. A clinical exam is first performed to ensure absence of symptoms to palpation or percussion. If such symptoms of persistent disease, or a sinus tract or swelling are noted, then the treatment provided at the first visit is repeated. At this point, the clinician may elect to apply triple antibiotic paste (at no more than 100 μg of each drug/mL).
2. After ascertaining adequate local anesthesia with 3% mepivacaine (no epinephrine), dental dam isolation is obtained.
3. The root canal systems are accessed; the intracanal medicament is removed by irrigating with 17% EDTA (20 mL/canal, 5 min).
4. The canals are dried with paper points. The clinician may infiltrate the apical region with 2% Lidocaine without vasoconstrictor to induce vasodilation and temporarily improve local anesthesia.
5. Bleeding is induced by rotating a pre-curved K-file size #25 at 2 mm past the apical foramen with the goal of having the whole canal filled with blood, possibly having it into the chamber above the level of the cemento-enamel junction.
6. Once a blood clot is formed, a premeasured piece of Collaplug™ (Zimmer Dental Inc, Warsaw, Indiana, U.S.A) is carefully placed on top of the blood clot to serve as an internal matrix for the placement of approximately 3 mm of white MTA (Dentsply, Tulsa, Oklahoma, U.S.A) or Biodentine (Septodont, France).
7. A (3–4 mm) layer of glass ionomer (e.g., Fuji IX ™, GC America, Alsip, Illinois, U.S.A.) is flowed gently over the MTA and light cured for 40 secs.
8. A bonded reinforced composite resin restoration (e.g., Z-100™, 3M, St. Paul, Minnesota, U.S.A) is placed over the glass ionomer.
9. The patient should be reviewed at three months, six months, and yearly after that for a total of four years.

REGENERATIVE ENDODONTIC OUTCOMES

The definition of clinical success has been a controversial topic in endodontics throughout the history of the specialty. Success has been typically evaluated by lack of symptoms and a strict radiographic criteria[197,198] and the absence of cultivable microorganisms.[199] Currently, the most widely accepted outcomes for conventional non-surgical root canal treatment include the absence of clinical and radiographic signs of pulpitis and apical periodontitis following treatment.[200,201]

Regenerative endodontic procedures are a significantly newer treatment modality than conventional root canals therapies, and their expected outcomes have not been clearly defined in the scientific literature. This is particularly troublesome when these procedures are directly compared to apexification procedures. These new procedures are providing unprecedented results that, in certain cases, go beyond the primary goal of healing of apical periodontitis. Common findings of continued radiographic root development, greater tooth survival and positive vitality testing prompt consideration of more ambitious clinical expectations for REP as compared to more traditional treatment approaches. In reality, a multilevel approach to evaluating outcomes is required for REPs. This approach should be focused on three levels of outcomes: patient-centered, clinician-centered and scientist-centered outcomes.

Patient-Centered Outcomes

Simply stated, the outcomes that directly matter to the wellbeing of our patients should be considered the first level of outcome, or the base of the outcomes pyramid (Figure 29-7). These outcomes are aligned with patient's expectations from any given clinical intervention. In 2010, the Patient-Centered Outcomes Research Institute (PCORI) was established in the United States of America though the Patient Protection and Affordable Care Act.[202,203] This initiative underscores the need to focus on patient outcomes when making any comparative analysis of the effectiveness of treatment that could bring a clinical meaningful benefit to patients treated by these interventions.

Traditional medical clinical research has focused on specific dependent measures, such as biological markers and other surrogate measures of treatment effectiveness. This approach of evaluating treatment interventions is very important since it focuses on a clinician-scientist established set of criteria and parameters, facilitating mechanistic

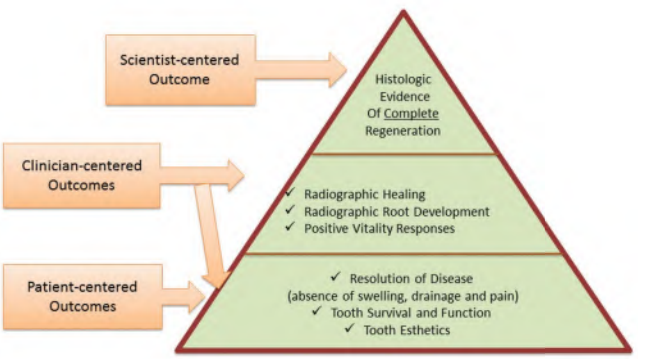


FIGURE 29-7 Regenerative endoodontic procedures must be assessed and evaluated in a systematic way, acknowledging that there are different levels of outcomes carrying different meanings for patients, clinicians, and scientists. However, potential benefit of improving patients' health and quality of life remains the goal and foundation of these procedures.

investigation. This approach, however, also tends to generalize findings, not necessarily addressing the impact on each individual patient treated. For example, c-reactive protein and vascular cell adhesion molecule-1 (VCAM-1) levels have been used as biological markers of atherosclerosis and angina pectoris.[204–206] The use of these markers as surrogate measures of treatment effectiveness allows for quantitative evaluation, but does not capture basic patient concerns, such as possible intense chest pain (angina) and possible myocardial infarction.

In endodontics, radiographic criteria were often applied as measure of clinical success.[207–209] This approach focuses on the resolution of symptoms and radiographic signs of "healing" as dependent measures. In this approach, a treatment that resolved moderate to severe debilitating pain, but resulted in a residual radiolucency, may have been considered a failure. This traditional approach largely ignores the overall benefit that the treatment brought to the patient. From the patient's perspective, the treatment resolved the symptoms and allowed normal function and survival of the tooth. Thus, a disconnect exists between the expectations of patient, clinicians and scientist. These expectations are not mutually exclusive but synergistic as they pertain to different aspects of the treatment, and their pursuit is a major driver of developments in the field. Lastly, patients should be empowered to make decisions about treatment effectiveness, reporting back to clinicians and researchers what has been beneficial or not beneficial. This approach places patients as stakeholders of their health, and of the development of new therapies.

Regenerative endodontic procedures, as a new treatment modality, must be compared to the longer established, more traditional endodontic treatment procedures for immature teeth, namely apexification procedures.[125,133] The primary concern for a practicing clinician must be to promote healing of the diseased tissues and patient well-being (patient-centered outcomes). Thus, the primary therapeutic goal must be resolution of apical periodontitis while promoting the survival and function of the tooth. Simply put, patients and guardians (in the case of young patients undergoing treatment) value the fact that the tooth is rendered pain free, and that it remains functional in the long term (survival).

Regenerative endodontic procedures have been proposed as an alternative treatment modality for teeth with immature roots and pulp necrosis.[9] Therefore, the direct comparison of REPs and apexification procedures is warranted. In 2012, a retrospective study aimed to directly compare the clinical outcomes for these two treatment modalities.[141] This is an important study because it was the first to establish standardized protocols for each treatment arm. Also, it included calcium hydroxide-mediated apexification (n = 22) and MTA-mediated apexification (n = 19) groups as variations of the apexification technique while comparing these to REPs (n = 22).[141] Resolution of the disease process (no pain, swelling or sinus tracts) was found in 100% of the REPs, 95% of the MTA apexification and 77% of calcium hydroxide apexification cases. On the other hand, another retrospective study that did not include standardization of treatment protocols found REPs to promote healing in 79% of patients treated, whereas apexification procedures promoted healing in 100% of the patients; this difference was found not to be significant.[210] In a prospective study, both MTA apexification and REPs were found to promote healing in 100% of all patients.[211] Further evidence of successful outcomes can be found in case series and retrospective patient cohort studies. Despite significant variations in etiology, inclusion and exclusion criteria, these studies demonstrate that in average both procedures successfully resolve the signs and symptoms of disease in approximately 90% of patients.

Survival of the tooth is another important patient-centered outcome. An ideal treatment, from the patient's perspective, is one that prolongs the functional life of an asymptomatic tooth. The survival of an immature tooth is of crucial importance to the patient since the tooth required an intervention at an early developmental stage, requiring the tooth to remain functional throughout the patient's life. In a retrospective study including more than 1.4 million patients, conventional root canal therapy was found to promote the eight-year survival in 97% of the cases.[212] Unfortunately, far less is known regarding the long term survival of immature teeth treated with either apexification or REPs. A retrospective study compared the survival of teeth treated with either REPs or apexification procedures with calcium hydroxide or placement of an MTA apical plug.[141] The survival of teeth treated with REPs (100% of teeth survived) during the follow-up period of the study (18 months) was significantly greater than that of teeth treated with calcium hydroxide (77%), but not different from teeth treated with an MTA apical plug (95%). These findings are in agreement with previous reports that prolonged use of calcium hydroxide can significantly weaken teeth, increasing their susceptibility to fracture.[138,140] Regenerative endodontic procedures have been shown to arrest the disease process and allow for retention of teeth with otherwise very poor prognosis (Figure 29-8) in patients still undergoing craniofacial development. Factors affecting the long-term survival of these teeth require further investigation with studies employing appropriate sample sizes and follow-up.

Clinician-Centered Outcomes

In addition to assessing patient's satisfaction with the treatment, clinicians further assess treatment outcome by evaluating whether the treatment provided the added benefit of continued radiographic root development and responses to vitality testing. There are numerous published cases that demonstrate continued root development post-treatment.[125] These reports provide subjective evidence that REPs promote root development. However, a retrospective study was the first to directly compare continued root development between apexification procedures (n = 40) and REPs (n = 48).[213] This

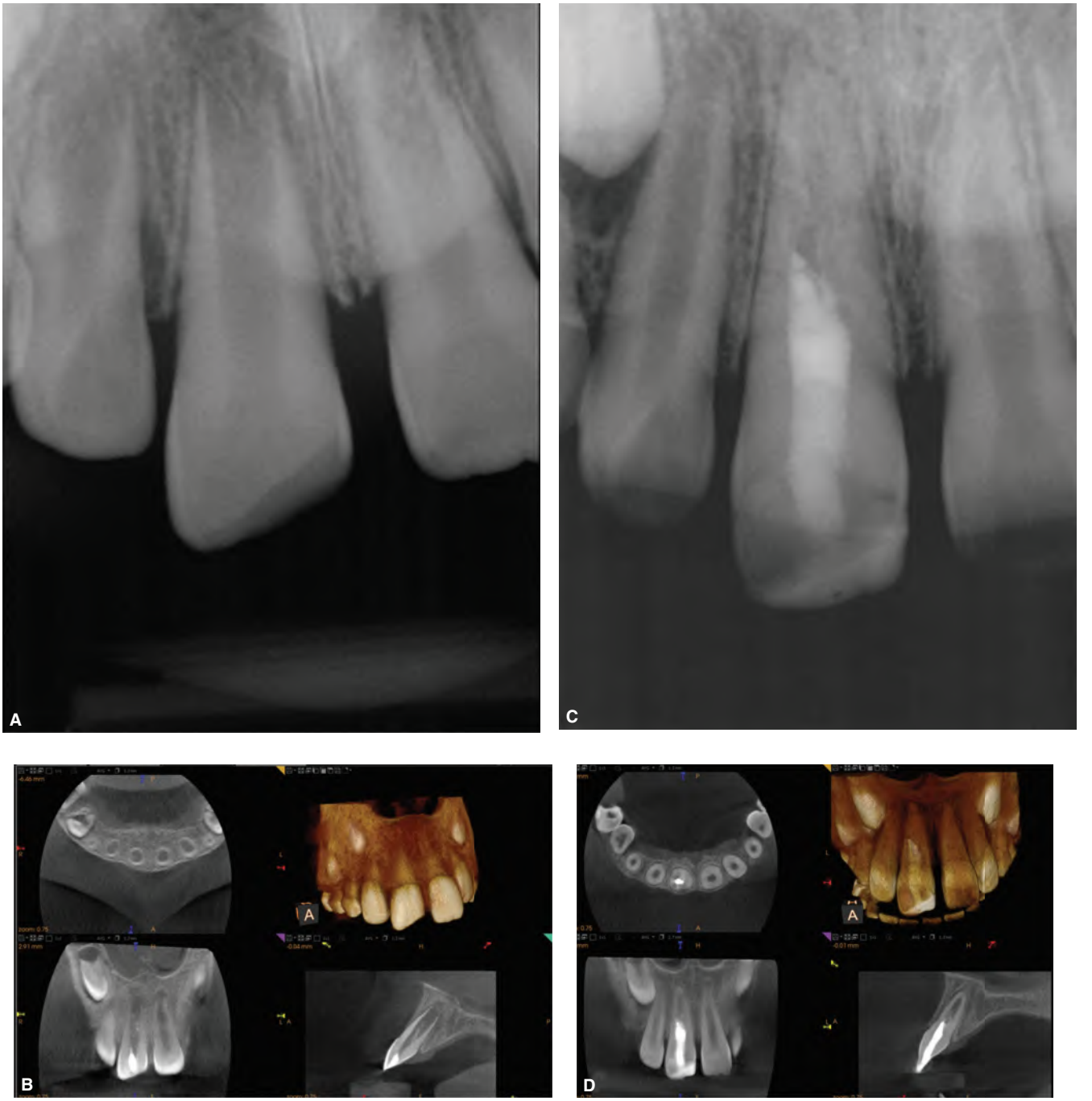


FIGURE 29-8 A 9-year-old boy suffered lateral luxation of the maxillary lateral and central incisors and an incomplete crown fracture of the right central incisor. ***A.*** The patient developed an acute apical abscess associated the right central incisor diagnosed with pulpal necrosis. The immature central incisor presented with fragile dentinal walls and open "blunderbuss" apex in the pre-operative radiograph. ***B.*** A regenerative endodontic procedure(revascularization/revitalization) was initiated with the use of double antibiotic paste as intracanal medicament in conjunction with an incision and drainage of the intraoral swelling resulting copious purulent exudate removal. At the final visit, patient was asymptomatic and intraoral swelling had resolved a limited-volume cone beam computer tomography (LV-CBCT) was taken providing a three-dimensional view of the immature root. The intracanal medicament was removed and the canal irrigated with 17% EDTA, dried and followed by intracanal bleeding evoked by over-instrumentation of apical tissues. An MTA coronal barrier was placed below the CEJ, followed by a glass ionomer barrier and composite restoration. ***C.*** At the one-year follow-up visit, the tooth responded to EPT and significant radiographic root development was evident on the periapical radiograp and on ***D.*** limited-volume cone beam computer tomography (LV-CBCT). (Courtesy of Dr. Julie Berkhoff, Colorado Springs, Colorado, U.S.A.)

study used a mathematical image-correction approach that permitted the comparison of non-standardized radiographs, revealing that regenerative procedures performed with either calcium hydroxide or triple antibiotic paste as intracanal medicaments produced significantly greater increases in root wall thickness and length. Importantly, this study established a methodology, later revised,[214] allowing unbiased and objective quantification of changes in root growth. These findings have been confirmed by Jeeruphan et al.[141] using a similar quantitative technique, reporting that teeth treated with revascularization showed a significantly greater percentage increase in root length (14.9%) compared with teeth treated by either MTA apexification (6.1%) or calcium hydroxide apexification (0.4%). 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 (1.52%).[141]

Further evidence of continued root development can be found in a prospective randomized clinical trial that compared two different protocols of REPs to MTA apexification procedures.[211] Regenerative procedures promoted an average increase of 12% in root thickness and a 50% decrease in apical diameter (apical closure), whereas apexification procedures demonstrated no change during the 18-month observation period.[211] Interestingly, a recent retrospective study reported that changes in radiographic root development were highly variable and unpredictable, being not statistically different from those seen in MTA-apexification cases.[210] This discrepancy is likely due to the greater number of included cases with trauma as the etiology for pulpal necrosis, and the relatively small sample size. Collectively, studies using quantitative analysis have provided evidence that REPs promote increases in radiographic root development in most cases, but not all. The factors that influence this lack of response in certain cases are largely unknown, but could include the persistence of bacterial biofilms or antigens, the nature of the etiology, delay in treatment, and the impact of disinfection agents on the creation of a microenvironment conducive to regeneration.

Reestablishment of normal nociception must also be one of the goals of REPs. The role of innervation in the defense and homeostasis of the dental pulp has been highlighted in many elegant studies.[215–218] Primary afferents in the dental pulp are equipped to sense microbial antigens, participating in the inflammatory process evoked by invading microorganisms.[219,220] They modulate the immune reaction by the release of neuropeptides with vasoactive properties that increase immune cell recruitment and vascularity, localizing areas of insult to microabcesses within the dental pulp.[221] Also, trigeminal neurons have been reported to increase odontoblastic differentiation and dentinogenesis.[222] Furthermore, there has been recent evidence that glial cells normally present surrounding pulpal primary afferent fibers, participate in the process of odontogenesis by stimulating differentiation of cells of the pulp dentin complex, including odontoblasts.[223] Therefore, pulpal innervation is a crucial component of the pulp-dentin complex with many functions in addition to nociception. Reestablishment of "normal" nociception is one of the important criteria for the functional regeneration of the pulp-dentin complex.

Positive pulpal responses are observed and reported in approximately 50% of all published REP case reports.[125] The presence of functional nociceptors following REPS suggests that free nerve endings of primary afferents found in the periradicular area grow and sprout into the newly formed tissue, a process called axonal targeting. The nociceptive pulpal innervation depends largely on the targeting of axons from sensory neurons located in the trigeminal ganglia into newly developed tissues during organogenesis.[224,225] Specific chemical signals are required to mediate the directed growth of axons into a newly formed tissue. Several elegant studies have directly implicated nerve growth factor (NGF), glial-derived neurotrophic factor and brain-derived neurotrophic factor (BDNF) in the process of pulpal innervation.[226–231] A recent study demonstrated that stem cells of the apical papilla (SCAP) release neurotrophins that promote axonal growth and axonal targeting.[116] This process was completely blocked by a monoclonal antibody against BDNF revealing that this neurotrophic factor is crucial to the recruitment of adjacent free nerve endings.[116] Interestingly, complete pulpal regeneration, including innervation, was achieved in dogs with the use of autologous transplantation of DPSCs.[122] These cells were found to express high levels of BDNF compared to adipose and bone marrow MSCs.[232] Collectively, these findings support the hypothesis that MSCs transferred into the root canal space during REPs are likely important in the recruitment of apical primary afferent fibers through a specific mechanism of post-natal axonal targeting.

The presence of innervation alone does not explain the observed responses to either electric or cold tests. Likewise, the hydrodynamic theory does not explain responses to cold stimuli since it is not expected to be operative in regenerative cases that lack innervated coronal dentin (pulp chamber roof). Instead, the innervated tissue is likely to be in intimate contact with the coronal plug (MTA or other materials) layered with other restorative materials (e.g., glass ionomer and composite restoration). The explanation for this clinical phenomenon probably lies in the expression of cold-sensing channels in trigeminal ganglial neurons, such as the Transient Receptor Potential Ankyrin type 1 (TRPA1) and Transient Receptor Potential cation channel, subfamily M member 8 (TRPM8).[233–236] These cation-permeable channels are expressed in the free nerve endings of trigeminal neurons, including those present within the dental pulp, and undergo a conformational change at temperatures below 17°C and 26°C, respectively, allowing the influx of cations and membrane depolarization.[237] Their function as cold sensors may underlie the mechanisms by which pulp-like tissue can detect cold stimuli. On the other hand, the electric pulp test bypasses activation of specific channels in the free nerve endings and nonselectively activates voltage gated sodium channels expressed throughout the length of the peripheral fibers.[238,239]

In summary, clinicians often rely on the pre-established criteria of radiographic continued root development and pulpal testing responses to further evaluate the "success" of a regenerative procedure. The meaning of these criteria

for the patient in not clear since most patients and guardians simply expect the tooth to be pain free and functional. Nonetheless, it adds valuable information that clinicians can use to determine whether further interventions are necessary. Importantly, the clinician-centered criteria, that are often used to evaluates REPs, are not directly applicable to apexification procedures since root development and vitality responses are not expected following these apexification procedures.

Scientist-Centered Outcomes

Histological evaluation of teeth, which 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.[240–242] This is the best example of a scientist-based outcome since it has strong scientific merit, but it does not necessarily interfere with consideration of rate of healing and continued root development seen in cases treated with REPs. Scientist-based outcomes can be some of the strongest drivers of advances in the field as researchers focus on the complex interplay between stem cells, scaffolds, growth factors and inflammation in order to achieve all desired outcomes, including complete histologic regeneration, or *de novo* formation of the pulp-dentin complex.

A 9-year-old male patient with an immature mandibular right first molar diagnosed with pulpal necrosis was treated with a "revitalization" procedure that included disinfection, use of triple antibiotic paste as intracanal medicament, intracanal bleeding in the distal canal and placement of PRP in the mesial canals.[242] The patient was asymptomatic for two years with both clinical and radiographic evidence of success. Unfortunately, the tooth was later diagnosed with an oblique fracture, perhaps due to the lack of adequate cuspal coverage. Histological examination of the extracted tooth revealed that the canals were filled with a vital connective tissue containing several foci of ectopic mineralization and the canal walls were covered by a mineralized tissue that resembled cellular and acellular cementum (Figure 29-9 and 29-10).

This case report highlights that the clinical success, seen until the time of fracture, was not significantly influenced by the nature of the tissue formed. Also, it demonstrated that regeneration, in its strict sense (formation of new dentin and odontoblast-like cells), was not accomplished. Instead, the histological outcome of this case suggests that a "guided

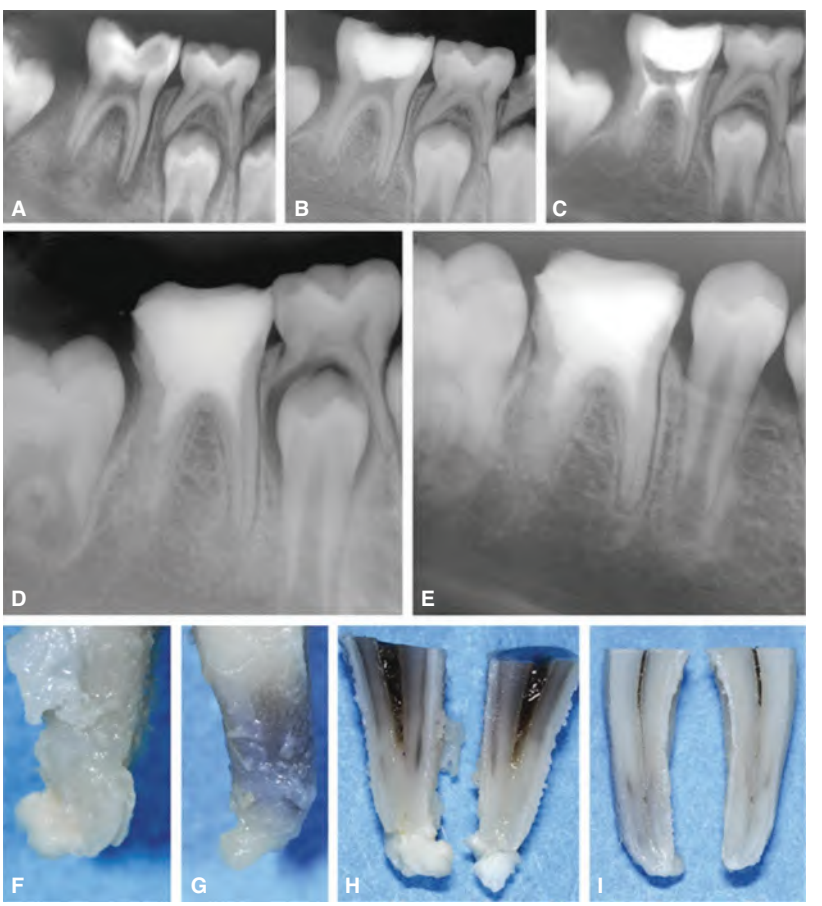


FIGURE 29-9 Mandibular firt molar of a 9-year-old boy treated with a revascularization/revitalization procedure. ***A.*** Preoperative radiograph. ***B.*** Radiograph of the tooth taken five months after triple antibiotic treatment. ***C.*** Postoperative radiograph after revascularization/revitalization. ***D.*** 14-month follow-up radiograph. ***E.*** Radiograph taken after 25 months at the time of root fracture. ***F,G.*** Photographs of the distal and mesial roots after tooth extraction. ***H,I.*** Distal and mesial roots separated longitudinally into two portions. (Adapted with permission from Martin et al.[242])

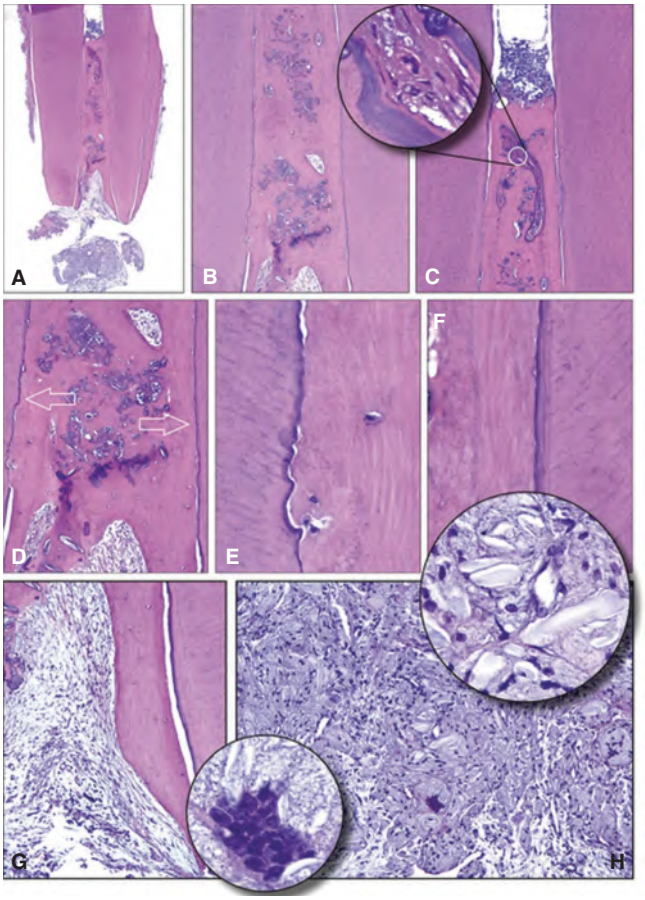


FIGURE 29-10 Histological examination of the distal root of a tooth treated with revascularization/revitalization procedures (Figure 29-9). ***A.*** The section passing approximately at the center of the root canal (hematoxylin-eosin; original magnification ×16). ***B.*** A detailed view of the apical canal in *A* (original magnification ×50). ***C.*** A detailed view of the upper portion of the canal in ***A.*** Coronally to the mineralized tissue, amorphous debris is present (×50; inset, ×400). ***D.*** Magnification of the apical portion of the canal in *B* (×100). ***E.*** A high-power view of the area of the root canal wall indicated by the *left arrow* in *D*. Few cells housed in lacunae can be seen in the newly formed mineralized tissue, resembling cementocytes or osteocytes (×400). ***F.*** A high-power view of the area of the root canal wall indicated by the *right arrow* in *D*. No cells can be seen in this area (×400). ***G.*** A detailed view from the apical foramen in *A* (×100). ***H.*** A detailed view from the center of the periapical soft tissue (×100; upper inset, ×400; lower inset, ×1000). (Adapted with permission from Martin et al.[242])

tissue repair" took place. Therefore, in this case, both patient and clinician-centered outcomes were met, but not the scientist-centered outcome of histologic regeneration.

The significance of the nature of the tissue formed for the clinical success of these cases remains largely unknown. Regenerative procedures are successfully performed on vital pulps, namely direct pulp capping procedures. Advances in biomaterials (e.g., MTA and Biodentine) that induce greater proliferation and differentiation of progenitor and stem cells have greatly improved the clinical outcomes of the procedures. In a large randomized clinical trial involving 35 dental practices, patients were randomized into direct pulp capping procedures with either calcium hydroxide (n = 181) or MTA (n = 195).[243] This study revealed that the use of MTA resulted in significantly greater clinical success (81.3%) than calcium hydroxide (69.5%). It is now established that newer generation biomaterials such as MTA or Biodentine, induce both direct and indirect effects on stem and progenitor cells of the dental pulp driving their recruitment and differentiation into odontoblast-like cells.[8,108,172] However, histological examination of the mineralized bridge suggests that the tissue formed does not resemble tubular dentin.

Tertiary dentin formed in response to an insult by surviving odontoblasts (reactionary dentinogenesis) is typically tubular, similar to primary and secondary dentins. Conversely, tertiary dentin formed by recruited "odontoblast-like" cells typically shows a range of tubularity from a regular tubular structure through to an atubular morphology, and when formed rapidly can present cellular inclusions (entrapment) of the mineralizing cells, being named osteodentin.[244] It is important to note that these cells do not have the morphological appearance of odontoblasts, but have been shown to express several of the recognized odontoblastic markers (such as DSP, DMP-1 and NeuN).[245,246] Thus, the desired formation of a biological seal (calcific barrier or mineralized bridge) in direct pulp capping procedures may not be considered true regeneration but "guided tissue repair." Nonetheless, the formation of this tissue allows for the maintenance of pulp vitality and normal physiology with high success rates.[247]

In summary, there is increasing evidence that the currently employed regenerative endodontic procedures (e.g., revascularization and revitalization procedures) are not allowing the formation of intracanal tissues that completely recapitulate the dental pulp. Histological studies have demonstrated the presence of connective tissues that are vascularized and innervated, that show some of the features of a pulp-like tissue. However, the presence of ectopic calcifications within the canal lumen and mineralized tissues resembling cementum along the dentinal walls suggest that clinicians and scientists are not yet able to achieve spatial, temporal, and lineage-specific control of stem cell differentiation. The ideal formation of dentin lined with odontoblast-like cells still remains elusive clinically. Nonetheless, these shortcomings largely ignore the benefits that these procedures bring to patients. Instead, the pursuit of true histological regeneration of physiological-like tissues as the pinnacle of sophistication for these biologically-based procedures is promoting substantial growth in our knowledge of the processes involved in repair and regeneration. Importantly, these advances are already being translated into clinical practice generating variations on the originally proposed clinical protocols and new treatment modalities, such as cell homing strategies and cell-based therapies.

FUTURE DIRECTIONS

It has become clear that greater effort should be made to preserve pulp vitality. Ideally, clinicians should be able to determine how much, if any, of the inflamed pulp to remove, before placing a bioactive coronal barrier and final restoration. However, clinicians still use subjective sensibility tests, and direct visualization of bleeding pulp stumps to establish pulp status, and to make decisions on whether to perform a pulpotomy (pulp capping) or pulpectomy (root canal therapy). Furthermore, these procedures have been offered mainly to young patients with a developing dentition. The impressive reparative and regenerative potential of dental pulp tissue makes these procedures a possibility, not only in deciduous or permanent immature teeth, but also in permanent fully mature teeth.[248]

A randomized multicenter clinical trial evaluated clinical outcomes of 407 patients with teeth diagnosed with irreversible pulpitis.[249] Approximately half of the patients received non-surgical root canal therapy, while the remaining patients were treated with a vital pulp therapy, consisting of pulp chamber pulpotomy and direct pulp capping with a calcium-enriched mixture (CEM). In this trial, patients experienced more post-operative discomfort following non-surgical root canal therapy and showed similar equivalent high clinical and radiographic success rates at the six months follow-up period.[249] In another randomized clinical trial, patients with molars diagnosed with symptomatic irreversible pulpitis (n = 413), were randomized into pulpotomy treatment arms, receiving either MTA or calcium-enriched mixture as a direct pulp capping material. At one-year follow-up, success rates were 95% for the MTA group and 92% for the calcium-enriched mixture.[250] These results are encouraging since these procedures promote the maintenance of a viable, functional pulp dentin complex while resolving the patients' chief complaint. Ideally, biomarkers should be identified as a chairside diagnostic indicator, of whether the remaining pulp is in an "irreversible" state or still harbors its inherent regenerative potential. The development of such technologies would provide powerful guidance, allowing clinicians to predictably preserve uninfected healthy dental pulp, even in cases presenting with spontaneous pain and lingering responses to cold stimuli.

In the treatment of immature teeth with pulpal necrosis using revascularization or revitalization procedures, a substantial number of undifferentiated stem cells are delivered into disinfected root canals.[100] Unfortunately, these cells are transferred to an environment that lacks adequate blood supply. It has been shown that they have the capacity to thrive in hypoxic environments, where they release various growth factors and neurotrophins that are crucial to the recruitment of blood vessels[251] and neurons.[252] However, the prolonged exposure of these cells to an environment of low oxygen tension, limited nutrients and gaseous exchange, can lead to cell death and impairment of differentiation for the surviving cells.[253,254] This disconnect between cellular demands and vascular supply could be avoided if cells were recruited progressively into the root canal space as blood vessels are formed.

Mesenchymal stem cells are highly mobile and have been shown to migrate towards concentration gradients of many known chemotactic factors, a process called stem cell homing.[38,44,52,255] The periapical tissues of immature teeth have rich sources of stem cells present in the apical lesions (i.e., inflamed periapical progenitor cells), apical papilla (SCAP), bone (bone marrow stem cells) and periodontal ligament. Therefore, these cells could be recruited into the root canal system if appropriate chemical signals and a suitable scaffold were provided. This strategy has been successfully demonstrated in many elegant studies.[38,256–260] A novel, cell homing system for endodontics, might be composed of an injectable scaffold with embedded chemotactic and angiogenic factors, released in a time-delayed fashion allowing for the progressive recruitment of stem cells concomitantly with supportive blood vessels. Alternatively, endogenous chemotactic and angiogenic factors sequestered within the dentin matrix might be progressively released from the walls of the pulp chamber by a slow-action demineralizing agent, possibly contained within a hydrogel.

CONCLUDING REMARKS

The field of regenerative endodontics has witnessed tremendous advances in the past decade, with knowledge gained on stem cell biology and the effects of endogenous and exogenous growth factors and morphogens, on the reparative and regenerative potential of the dental pulp. Also, the relatively young field of regenerative endodontics brings together basic scientists and clinicians, who in a multidisciplinary approach are making significant strides in endodontic tissue engineering.

Currently employed regenerative procedures are valuable adjuvants to the treatment of immature teeth with pulp necrosis and to retention of a functional pulp in cases of injured but vital teeth. Despite the clinical success of these procedures, they appear to promote a "guided tissue repair" process rather than a true regeneration of physiological-like tissues. Indeed, in order for the full regenerative potential to be met, significant more research and development needs to take place. A number of barriers to progress exist, including the absence of diagnostic markers correlating histological with clinical outcomes following treatment to facilitate case selection, insufficient knowledge of the molecular signaling interactions within pulp, in the context of the inflammatory environment and the consequences for regeneration, together with the need for better understanding and exploitation of the interplay between regeneration and low level cytokine stimulation during early stages of disease, and later stages of its resolution. Attention to all of these areas offers significant opportunities for progress in the evolution and clinical translation of regenerative endodontics. Future procedures may well incorporate the use of injectable scaffolds containing chemical cues to promote the orchestrated proliferation and differentiation of recruited stem cells to repopulate the root canal system, reestablishing a functional regenerated pulp-dentin complex. Endodontics is in an exciting phase from which many new developments, in regenerative therapies, will emerge to advance the specialty and patient-based treatment outcomes.

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Chapter 30

Treatment of Endodontic Abscesses/Cellulitis, Cysts, and Flare-ups

J. Craig Baumgartner, Paul A. Rosenberg

Endodontic infections are caused by communities of microbes that invade tissue and produce an inflammatory disease. Molecular studies have revealed a diversity of microbiota involved in endodontic infections.[1–3] Microbes that have been detected include bacteria, spirochetes, fungi, and viruses.[4–7] These communities of microbes differ from individual to individual. Many of the organisms have not been cultivated and have not been tested for susceptibility to the antimicrobials available for treatment. Understanding the nature and diversity of endodontic infections is required to provide the best clinical treatment and adjunctive pharmacotherapeutics for a successful outcome.[8,9]

Periradicular abscesses and cellulitis occur when microbes invade periradicular tissues. The severity of the periradicular infections is related to the virulence of the organisms and host resistance. An abscess is a localized collection of pus within a tissue or confined space.[10] Pus is an accumulation of purulent exudate and consists of microbes, microbial by-products, neutrophils, cells undergoing autolysis, and cellular contents. Cellulitis is a symptomatic edematous inflammatory process that spreads diffusely through connective tissue and fascial planes; frequently associated with an infection by invasive microorganisms and subsequent breakdown of connective tissue.[10] Needle aspirates from inflamed tissue or incision for drainage often reveal pockets of purulence associated with cellulitis.[11] Clinically a cellulitis and an abscess should be considered a continuum of the infectious-inflammatory process. Once bacteria have invaded periradicular tissues an otherwise healthy patient will eventually exhibit clinical signs and symptoms. Locally the patient may experience swelling and mild to severe pain. As the infection and inflammatory response spreads to surrounding tissues the patient may experience systemic signs and symptoms such as chills, fever, lymphadenopathy, nausea, and headache. The tooth that is the reservoir of the endodontic infection will usually be sensitive to biting, percussion (touch), and eventually palpation. The tooth may or may not show radiographic evidence of periradicular disease. The pulp cavity of an infected tooth is the source of the infection spreading to periradicular tissues and for secondary (metastatic) infection that spread to fascial spaces of the head and neck. Appropriate treatment involves decreasing the bioburden by removing the reservoir of infection either by debridement of the infected root canal system or removal of the tooth along with drainage from swollen tissues. Adjunctive pharmacotherapeutics may also be indicated for cellulitis, progressive swellings, and where there are systemic signs and symptoms of infection.

The spread of infection into fascial spaces may be life threatening. There are potential spaces between anatomic structures of the head and neck, which may be involved in infection and inflammation. The fascial spaces of the head and neck have been placed into several anatomic groups.[12] An anatomic space that may be involved in mandibular infections is the ***mandibular buccal vestibule***. The swelling occurs between the cortical plate and the buccinator muscle in the posterior of the mandible and the mentalis muscle in the anterior. Mandibular anterior teeth may produce swelling of the ***mental or submental spaces*** (Figure 30-1). The ***mental space*** is located between the mentalis muscle and the platysma muscle. The ***submental space*** is between the mylohyoid muscle and the platysma muscle. Mandibular anterior teeth may also lead to swelling in the ***sublingual space*** located between the floor of the mouth and the inferiorly located mylohyoid muscle. The ***submandibular space*** (Figure 30-2) is between the mylohyoid and the platysma muscles. The source of infection in this space is usually a posterior tooth. ***Ludwig's angina*** is an infection that includes the submental, sublingual, and submandibular spaces. Cellulitis may extend to the pharyngeal and cervical spaces and produce airway obstruction.

Anatomic spaces involved in lateral face swelling include the ***maxillary buccal vestibule***. It is located between the cortical plate of the maxilla and the superior attachment of the buccinator muscle and associated with infections of maxillary posterior teeth. The ***buccal space*** (Figure 30-3) is between the lateral surface of the buccinator muscle and the overlying skin of the cheek. The source of the infection may be either a maxillary or mandibular posterior tooth. The ***submasseteric space*** lies between the lateral surface of the ramus of the mandible and the masseter muscle. The source of the infection is usually the mandibular third molar. The ***deep temporal space*** is between the lateral surface of the skull and the medial surface of the temporal muscle while the ***superficial temporal space*** lies between the temporal muscle and the overlying fascia.

Anatomic spaces involved in the pharyngeal and cervical areas include the ***pterygomandibular space*** (Figure 30-3). That space is usually associated with infection of the mandibular second or third molars. The pterygomandibular space is bounded by the lateral surface of the medial pterygoid muscle and the medial surface of the mandible. The bilateral ***parapharyngeal space*** is between the medial surface of the medial pterygoid muscle and the superior constrictor muscle.

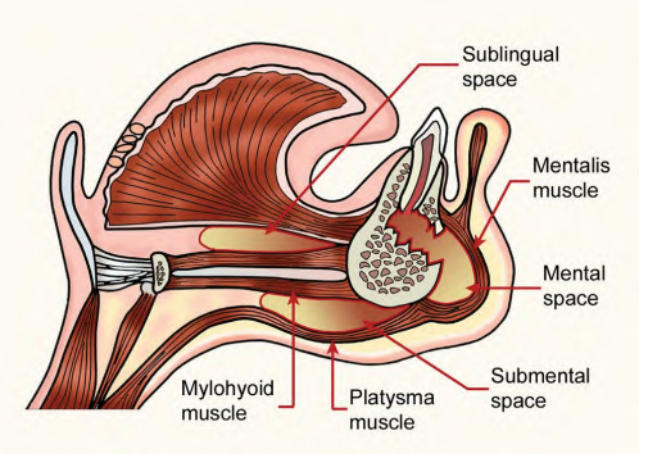


FIGURE 30-1 Mental, submental, and sublingual spaces. Courtesy of William J. Girsch, Salem, OR, U.S.A.

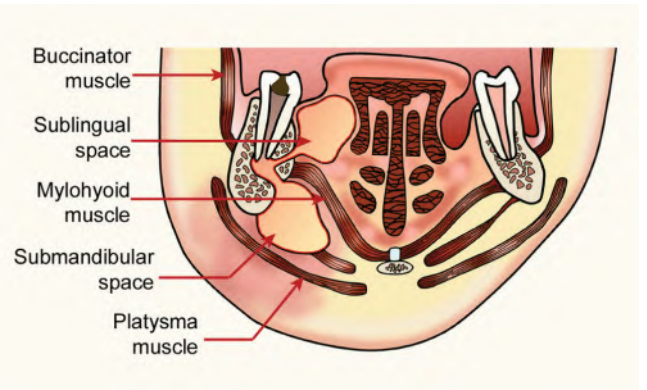


FIGURE 30-2 Submandibular and sublingual spaces. (Courtesy of William J. Girsch, Salem, OR.)

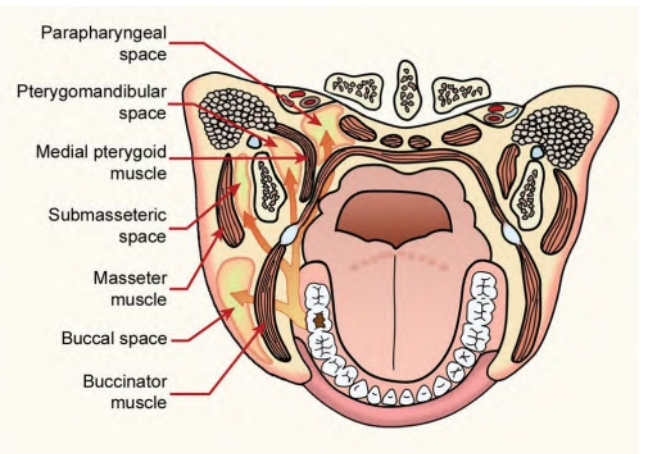


FIGURE 30-3 Buccal, submasseteric, pterygomandibular, and parapharyngeal spaces. (Courtesy of William J. Girsch, Salem, OR.)

The superior border is the base of the skull and the inferior border is the hyoid bone. The ***carotid space*** contains the carotid artery, internal jugular vein, and the vagus nerve. The ***retropharyngeal space*** (Figure 30-4) is posterior to the superior constrictor muscle and extends to the mediastinum. The ***pretracheal space*** surrounds the trachea and extends from the thyroid cartilage to the level of the aortic arch. The ***retrovisceral space*** extends from the base of the skull into the posterior mediastinum. The ***danger space*** (Figure 30-4) also extends from the base of the skull into the posterior mediastinum. The ***prevertebral space*** surrounds the vertebral column.

The anatomic spaces involved in midface swellings are the palate, the base of the upper lip, the infraorbital (canine) space, and the periorbital spaces. The source of infection for the ***palate*** is the maxillary teeth. Swelling of the ***base of the nose*** is typically caused by a maxillary central incisor with its root apex above the attachment of the orbicularis oris muscle. The ***infraorbital space*** (Figure 30-5) is between the levator anguli oris muscle and the levator labii superioris muscle. The source of the infection is usually the maxillary cuspid or first premolar. The ***periorbital space*** lies deep to the orbicularis

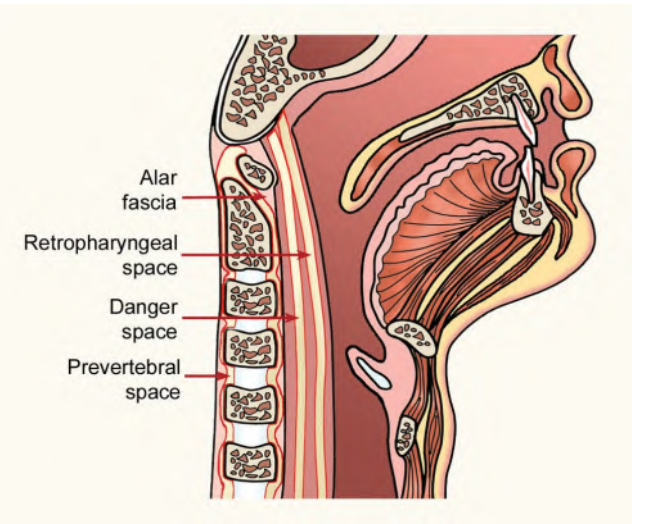


FIGURE 30-4 Pretracheal, prevertebral and danger spaces. (Courtesy of William J. Girsch, Salem, OR.)

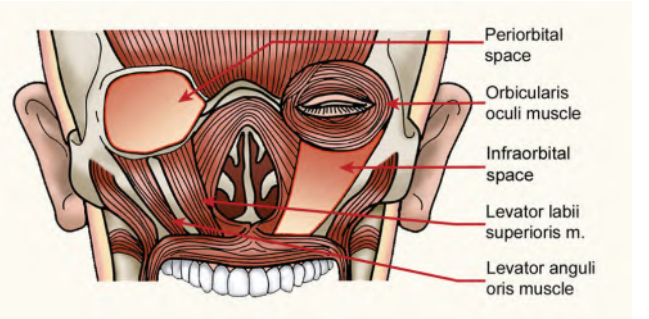


FIGURE 30-5 Periorbital and canine spaces. (Courtesy of William J. Girsch, Salem, OR.)

oculi muscle and becomes involved as a result of spread of infection from the buccal or infraorbital space. Infections of the midface are of special concern because of the possibility that they may result in cavernous sinus thrombosis. If the inflammation and resulting edema cause blood to back up into the cavernous sinus, the infected thrombi may escape into circulation and produce a life threatening event.

TREATMENT OF ENDODONTIC ABSCESSES/CELLULITIS

Prompt diagnosis and removal of the reservoir of infection are important for the successful treatment of endodontic infections. Drainage of infectious material and inflammatory mediators associated with endodontic infections may be established through the tooth, soft tissues, or alveolus. Surgical methods for drainage include incision for drainage, needle aspiration, and trephination. In otherwise healthy patients, chemomechanical debridement of the root canal system and drainage of an associated swelling will lead to rapid improvement of the patient's signs and symptoms. Tooth extraction is the alternative treatment to remove the reservoir of infection. After debridement of the root canal system, incision for drainage is indicated for periradicular swellings. Drainage from the access opening to the pulp cavity may not significantly reduce pain, percussion pain, swelling, or number of analgesics taken (Figure 30-6).[13]

Incision for Drainage

The importance of removing the source of the infection and drainage must be emphasized. Successful resolution of an infection requires removal of the source of infection and drainage of accumulated exudates. Drainage from a swelling caused by an abscess/cellulitis decreases the number of microbes, microbial by-products, and inflammatory mediators associated with the swelling. The removal of pockets of purulence and edematous fluid also improves circulation and allows delivery of prescribed antibiotics in a minimum inhibitory concentration. An incision for drainage or needle aspiration of a swelling associated with cellulitis will often reveal accumulations of purulence consistent with an abscess. An incision provides a pathway for drainage to prevent the further spread of the abscess/cellulitis. Nerve block anesthesia should be used and if necessary supplemented with local infiltration.

To achieve drainage from a swelling, a stab incision through the periosteum is made at the most dependant site of the swelling. A periosteal elevator or hemostat is then used for blunt dissection to allow drainage of any accumulated exudate and inflammatory mediators. Except for very small-localized swellings a drain should be sutured into place (Figure 30-7). Drains may be a piece of dental dam, a Penrose drain, or a capillary drain (Figure 30-8). The latter is ribbed and may stay under the tissues better than the

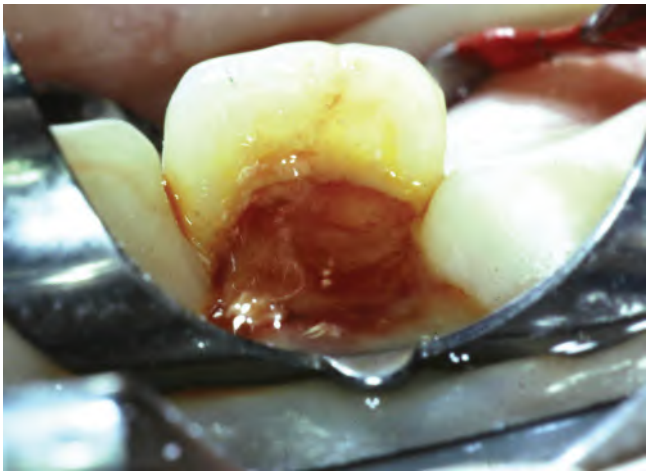


FIGURE 30-6 Purulent drainage upon access to the pulp cavity. (Courtesy of J. Craig Baumgartner, Kailua, HI, U.S.A.)

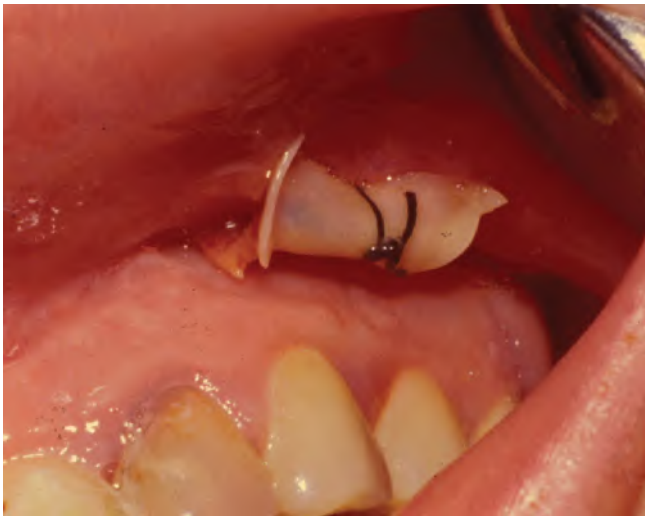


FIGURE 30-7 Drain sutured in place. (Courtesy of J. Craig Baumgartner, Kailua, HI, U.S.A.)

FIGURE 30-8 Top drain is a Capillary drain, middle—Penrose drain, bottom—dental dam drain. (Courtesy of J. Craig Baumgartner, Kailua, HI, U.S.A.)

non-ribbed drains. Non-latex drains should be used for those allergic or sensitive to latex. A sutured drain keeps the incision open for continued drainage and encourages compliance by the patient to return for follow-up evaluations. The use of warm intraoral rinses may help promote drainage. The drain can usually be removed in 1 or 2 days when there is improvement of the patient's clinical signs and symptoms. Consultation and referral is indicated for severe infections or persistent infections.

Needle Aspiration

Needle aspiration may be described as the use of suction to remove fluids from a cavity or space. Needle aspiration is a surgical procedure that may provide information as to the presence and volume of exudate, cystic fluid, or blood. Aspirated samples may be used for microbial isolation and identification using either culturing or molecular methods (Figure 30-9).[3,14–16] In addition, the sample may be used for immunohistochemical analysis (Figure 30-9). In 1995, Simon et al.[17] described the clinical use of needle aspiration for intraoral swelling as an alternative technique to incision for drainage. Following regional and local anesthesia, the technique involves the use of a syringe with an 18 gauge needle to aspirate the contents of a swelling. Clinical advantages of needle aspiration over incision for drainage include reduced scarring, evaluation of volume and character of the aspirate, use of the aspirate for culture and sensitivity testing, and the lack of postoperative drain removal.

Antibiotics In Endodontics

The vast majority of infections of endodontic origin can be effectively managed without the use of antibiotics. Systemically administered antibiotics are not a substitute for proper endodontic treatment. Chemomechanical debridement of the infected root canal system with drainage through the root canal and by incision and drainage of swollen tissues will decrease the bioburden so that a normal healthy patient can begin the healing process. Antibiotics are not recommended for healthy patients with a symptomatic pulpitis, symptomatic apical periodontitis, a draining sinus tract, a localized swelling of endodontic origin, or following endodontic surgery (Table 30-1).[18–23] An antibiotic regimen should be prescribed in conjunction with proper endodontic therapy when there are systemic signs and symptoms of infection or a progressive/persistent spread of infection. The presence of a fever (>100°F), malaise, cellulitis, unexplained trismus, and progressive swelling are all signs and symptoms of systemic involvement and the spread of infection (Table 30-2).

Under these circumstances, an antibiotic is indicated in addition to debridement of the root canal harboring the infecting microbes and drainage of any accumulated purulence. Patients with serious endodontic infections should be closely followed on a daily basis. The patient's condition will usually rapidly improve once the source of the infection is removed. Because of the lack of circulation, systemically administered antibiotics are not effective against a reservoir of microorganisms within an infected root canal system. Likewise, a minimum inhibitory concentration of an antibiotic may not reach an anatomic space filled with purulence and edematous fluid because of poor circulation and the diffusion gradient that it must traverse. Pus consists mainly of neutrophils, cellular debris, bacteria, bacterial by-products, enzymes, and edematous fluid. An incision for drainage will allow drainage of the purulent material and improve circulation to the area. Drainage provides a pathway for removal of inflammatory mediators and helps prevent further spread of cellulitis in most cases.

Empirical selection of an antibiotic (antimicrobial agent) should be based on knowledge of which bacteria are most commonly associated with endodontic infections and their antibiotic susceptibility.[8,15,23–30] Many of the microbes in endodontic infections have yet to be cultivated for antimicrobial sensitivity. The clinician must be thoroughly familiar

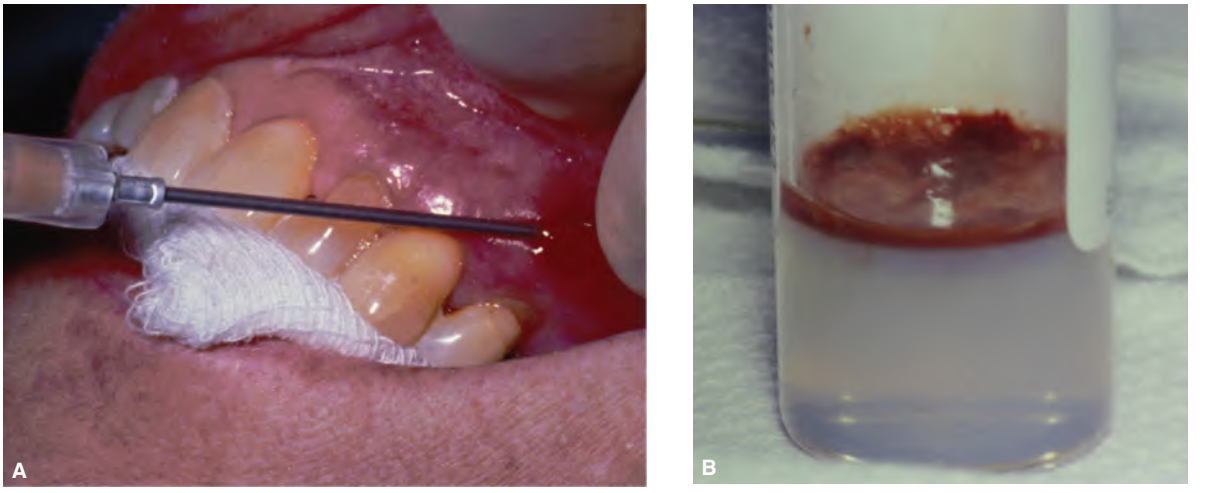


FIGURE 30-9 Obtaining aspirate from abscess/cellulitis. ***A.*** Needle aspiration. ***B.*** The aspirated sample is injected through the stopper into an anaerobic transport vial. (Courtesy of J. Craig Baumgartner, Kailua, HI, U.S.A.)

TABLE 30-1 Conditions Not Requiring Adjunctive Antibiotics

Pain without signs and symptoms of infection
Symptomatic irreversible pulpitis
Symptomatic apical periodontitis
Teeth with necrotic pulps and a radiolucency
Teeth with a sinus tract (chronic apical abscess)
Localized fluctuant swellings

TABLE 30-2 Indications for Adjunctive Antibiotics (Antimicrobial Therapy)

Systemic Involvement
Fever > 100°F
Malaise
Lymphadenopathy
Trismus
Progressive Infections
Increased swelling
Cellulitis
Osteomyelitis
Persistent Infections

with the antibiotic and inform the patient of the benefits, possible side effects, and possible sequelae of the patient's failure to take the proper dosage. A loading dose is important to provide an initial adequate therapeutic level of antibiotic. The antibiotic should generally be continued for 2 to 3 days following resolution of the major clinical signs and symptoms of the infection. Following removal of the source of the infection and adjunctive antibiotic therapy, significant improvement in the patient's status should be seen in 24 to 48 hours. A prescription written for a 7-day regimen of antibiotic therapy is usually adequate.

Penicillins are the antibiotics of choice for patients not allergic to them. However, up to 10% of the population may be allergic, so a careful history of drug hypersensitivity is important. Penicillin VK is effective against both facultative and anaerobic microorganisms commonly isolated and cultivated form endodontic infection. [8,15,25,27,28] A loading dose of 1,000 mg of penicillin VK should be orally administered, followed by 500 mg every 4 to 6 hours.

Amoxicillin is an antibiotic of choice for treating infections of endodontic origin.[8,15,23,25–28] Amoxicillin has an expanded spectrum of activity that may include bacteria involved in endodontic infections yet not cultivated in addition to bacteria not routinely associated with infections of endodontic origin. The clinician must decide if the expanded spectrum over penicillin VK is indicated. Amoxicillin is rapidly absorbed and provides sustained serum levels. Amoxicillin is recommended for medically compromised patients that require antibiotic prophylaxis. The usual oral dosage for amoxicillin to treat endodontic infections is 1,000 mg loading dose followed by 500 mg every 8 hours. An alternate dosage for amoxicillin is 875 mg every 12 hours. As stated above, a prescription written for a 7-day regimen of antibiotic therapy is usually adequate for endodontic infections once the reservoir of infection is removed by endodontic treatment or root extraction.

Clavulanate is a competitive inhibitor of beta-lactamase. Antibiotic susceptibility tests have shown excellent results against bacteria isolated form endodontic infections when clavulanate is used in combination with amoxicillin (Augmentin™).[8,15,23,25–28] This combination should be especially considered for patients that are immunocompromised. The usual oral dosage for amoxicillin with clavulanate is 1,000 mg loading dose followed by 500 mg every 8 hours. An alternate dosage is 875 mg every 12 hours.

Clindamycin is highly effective against both facultative and strict anaerobic bacteria associated with endodontic infections. It is well distributed throughout the body, especially to bone, where its concentration approaches that of plasma. Both penicillin and clindamycin have been shown to produce good results in treating odontogenic infections.[8,15,23,25,27] Clindamycin is rapidly absorbed even in the presence of food in the stomach. The oral adult dosage for serious endodontic infections is a 600 mg loading dose followed by 300 mg every 6 hours.

Erythromycin has traditionally been the alternative choice for patients allergic to penicillin, but it is not effective against anaerobes associated with endodontic infections. Clarithromycin and azithromycin are macrolides like erythromycin, with some advantages over the latter. They have a spectrum of antimicrobial activity that includes facultative bacteria and some anaerobic bacteria associated with infections of endodontic origin.[8,15] They also have less gastrointestinal upset than erythromycin. In a recent population-based Danish study, there was an increased risk of cardiac death with clarithromycin compared with those who were receiving penicillin V for similar infections.[31] The oral dosage for clarithromycin is a 500 mg loading dose followed by 250 mg every 12 hours. The oral dosage for azithromycin is a 500 mg loading dose followed by 250 mg once a day.

Metronidazole is a nitroimidazole that is active against parasites and anaerobic bacteria. However, it is ineffective against facultative bacteria.[8,15,32] It is a valuable antimicrobial agent in combination with penicillin when penicillin alone has been ineffective.[32] The usual oral dosage for metronidazole is a 1,000 mg loading dose followed by 500 mg every 6 hours. When patients fail to respond to treatment, consultation with a specialist is recommended.

Cephalosporins are usually not indicated for the treatment of endodontic infections. First generation cephalosporins do not have activity against the anaerobes usually involved in endodontic infections. Second generation cephalosporins have some efficacy for anaerobes; however, there is a possibility of cross-allergenicity of cephalosporins with penicillin.

Doxycycline occasionally may be indicated when the above antibiotics are contraindicated. However, many strains of bacteria have become resistant to tetracyclines.

Ciprofloxacin is a quinilone antibiotic that is not effective against the anaerobic bacteria usually isolated from endodontic infections. With a persistent infection it may be indicated to take culture and perform sensitivity tests to demonstrate the presence of susceptible organisms.

Prophylactic Antibiotics for Medically Compromised Patients

Prophylactic antibiotic coverage may be indicated for medically compromised patients requiring endodontic treatment. The American Heart Association (AHA) and the American Academy of Orthopaedic Surgeons have made guidelines for prophylactic antibiotic coverage.[33,34] The guidelines are meant to aid practitioners but are not intended as the standard of care or as a substitute for clinical judgment. The incidence of endocarditis following most procedures on patients with underlying cardiac disease is low. A reasonable approach for prescribing prophylactic antibiotics considers the degree to which the underlying disease creates a risk for endocarditis, the apparent risk for producing a bacteremia, adverse reactions to the prophylactic antibiotic, and the cost-benefit aspect of the regimen. Antibiotic prophylaxis is employed to prevent surgical infections or their postoperative sequelae, to prevent metastatic bacteremia, and to prevent accusation that "all was not done for the patient."[35] It is suspected that antibiotic prophylaxis is often prescribed to prevent malpractice claims.[35]

How antibiotics quickly kill bacteria in the blood is difficult to answer when many antibiotics are only effective with actively dividing bacteria. It is speculated that antibiotics may reduce metastatic infections by preventing adhesion of bacteria to tissues or inhibiting growth after attachment.[36] The principles of antibiotic prophylaxis state that the antibiotic must be in the system prior to an invasive procedure. If a patient has not taken the prescribed antibiotic, they should be rescheduled or wait an hour after administration of the antibiotic for treatment. However, there is data to support the use of an antibiotic up to 2 hours after the bacteremia.[35]

The incidence of bacteremia has been shown to be low during root canal therapy. A transient bacteremia can result from the extrusion of the microorganisms infecting the root canal beyond the apex of the tooth.[37–41] In addition, care must be taken when positioning dental dam clamps and accomplishing other dental procedures that may produce bleeding with an accompanying bacteremia. Medically compromised dental patients who are at risk of infection should receive a regimen of antibiotics that either follows the recommendations of the AHA or an alternate regimen determined in consultation with the patients' physicians.[35] It is believed that amoxicillin, ampicillin, and penicillin V are equally effective against alpha-hemolytic streptococci; however, amoxicillin is recommended because it is better absorbed from the gastrointestinal tract and provides higher and more sustained serum levels.[35] For cardiac conditions associated with endocarditis, prophylaxis is recommended for both non-surgical and surgical endodontics.[35] Antibiotic prophylaxis is recommended for cardiac conditions associated with endocarditis at a high or moderate risk category. Dental procedures for which antibiotic prophylaxis is recommended include endodontic instrumentation or surgery beyond the apex, but not intracanal endodontic treatment, post placement and buildup.[35] From a practical standpoint, it is difficult to determine with certainty that endodontic instruments did not pass beyond the apical foramen. Also included for prophylaxis antibiotics is intraligamentary (PDL) local anesthetics but not non-intraligamentary local anesthetic injections.[35]

A joint committee of the American Dental Association and American Academy of Orthopaedic Surgeons published their first advisory statement on antibiotic prophylaxis for patients with prosthetic joints.[33,34] Dental procedures of concern and the antibiotic regimens are the same as for endocarditis. Patients of potential increased risk of having a hematogenous total joint infection include all patients during the first 2 years following joint replacement, immunocompromised/immunosuppressed patients, and patients with comorbidities. For more details, see Chapter 31.

Collection Of A Microbial Sample

Adjunctive antibiotic therapy for endodontic infections is most often prescribed empirically based on our knowledge of the bacteria most often associated with endodontic infections. At times, culturing may provide valuable information to better select the appropriate antibiotic regimen. For example, an immunocompromised/immunosuppressed patient (not immunocompetent) or patients at high risk of developing an infection (e.g., history of infective endocarditis) following a bacteremia require close monitoring. These patients may have an infection caused by bacteria usually not associated with the oral cavity. Other examples include a seemingly healthy patient who has persistent or progressive symptoms following surgical or nonsurgical endodontic treatment. An aseptic microbial sample from a root canal is accomplished by first isolating the tooth with a dental dam and disinfecting the tooth surface and dental dam with sodium hypochlorite or other disinfectant. Sterile burs and instruments must be used to gain access to the root canal system. Intra-canal irrigation should not be used until after the microbial sample has been taken. If there is drainage from the canal, it may be sampled with a sterile paper point or aspirated into a syringe with a sterile 18–25-gauge needle depending on the viscosity of the exudates. The aspirate should be either taken immediately to a microbiology laboratory in the syringe or injected into pre-reduced transport media. To sample a dry root canal, a sterile syringe should be used to place some pre-reduced transport medium into the canal. A sterile endodontic

instrument is then used to scrape the walls of the canal to suspend microorganisms in the medium.

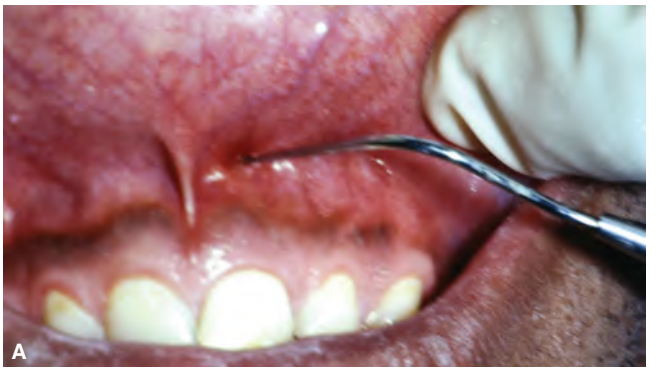
To prevent contamination by "normal oral flora," a microbial sample from a soft tissue swelling should be obtained using needle aspiration before making an incision for drainage (Figure 30-8). Once profound anesthesia is achieved, the surface of the mucosa should be dried and disinfected with an iodophor swab (The Purdue Frederick Company, Norwalk, Conn.) or a similar product. A sterile 16 to 20 gauge needle is then used to aspirate the exudate. The aspirate should be handled as described above (Figure 30-9). After collecting the specimen on a sterile swab, it should be quickly placed in pre-reduced medium for transport to the laboratory.

Good communication with the laboratory personnel is important. The sample should be Gram-stained to demonstrate which types of microorganisms predominate. The culture results should show the prominent isolated microorganisms and not just be identified as "normal oral flora." Antibiotics can usually be chosen to treat endodontic infections based on the identification of the prominent microorganisms in the culture. With persistent infections, susceptibility testing can be undertaken to establish which antibiotics are the most effective against resistant microbial isolates. At present, it may take 1 to 2 weeks to identify anaerobes using conventional methods. Some laboratories may have molecular methods available to rapidly detect and identify known opportunistic bacteria.

Cortical Trephination

Cortical trephination is defined as the surgical perforation of the alveolar cortical plate or apical foramen to release accumulated tissue exudates.[10] Cortical plate trephination is indicated for patients with severe pain of endodontic origin without intraoral or extraoral swelling and when drainage cannot be accomplished through the root canal (e.g., posts, filling material, ledging). Cortical trephination involves exposing the cortical bone, making an opening in the bone, and making a pathway through the cancellous bone to the root-end.[42–47] If the cortical plate is thin, a dental instrument such as a gutta-percha spreader may be used to penetrate the mucosa and cortical plate without an incision (Figure 30-10). Several studies have demonstrated that a patient with severe periapical pain without swelling will have significant relief following trephination.[43,44,48–50] Henry and Fraser[44] recommended trephination for the management of severe periapical pain of endodontic origin when drainage through the tooth is inadequate, impractical, or impossible. Their technique involved a submarginal horizontal full thickness flap to access the alveolar bone, the use of a surgical high speed round bur to access the involved root apex and abscessed area, and the placement of a sutured drain.

Other studies have not shown cortical trephination to be predictable in relieving periradicular pain.[45–47] One study found that the routine use of trephination for the reduction of pain or swelling in symptomatic necrotic teeth with periradicular radiolucencies was not predictable.[47] A systematic review of the literature concerning the emergency management of acute apical periodontitis in the permanent dentition also concluded that routine cortical trephination did not show significant benefit.[50] While there is no higher level of evidence justifying the routine use of surgical trephination, there are instances in which it is a reasonable treatment alternative. Patients with severe periradicular pain of endodontic origin without swelling may benefit from the procedure.



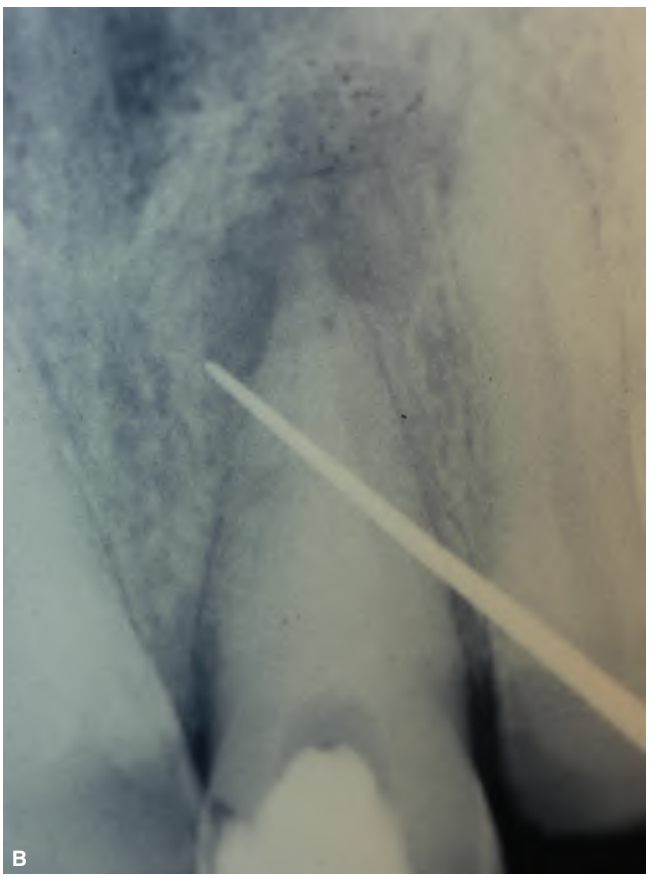


FIGURE 30-10 Trephination procedure. ***A.*** A #3 spreader is used to penetrate the mucosa and cortical plate. ***B.*** Radiograph showing tip of #3 spreader near root-end. (Courtesy of J. Craig Baumgartner, Kailua, HI, U.S.A.)

Decomprssion: Aspiration and Irrigation

The terms decompression and marsupialization are often used interchangeably. Decompression is the surgical exposure of a cyst wall and insertion of a tube or other type of drain to decompress the lesion during healing.[10] It is not uncommon for chronic periradicular pathosis to remain clinically asymptomatic and develop a bony defect of significant size. If left undiagnosed and untreated, periradicular pathosis may develop into self perpetuating entities that erode osseous supporting structures and encroach on multiple adjacent teeth, sinus cavities, neurovascular bundles, and even the nasal cavity. Bony lesions radiographically exceeding 200 square mm have a higher statistical chance of being cystic.[51] There are radicular cysts that may have matured to the extent that they are truly independent and nonsurgical endodontic treatment may no longer be enough to cause bony healing.[52,53] When nonsurgical endodontic does not resolve apical pathosis, surgical intervention is an alternative treatment recommendation.

Surgical treatment including the enucleation of extensive bony lesions may involve unintentional interruption of periradicular vascular and neural structures, development of soft tissue defects, and damage to adjacent anatomic structures. Decompression is a more conservative treatment option which allows the progressive reduction in lesion size and may eliminate the necessity of surgical enucleation. Decompression is intended to disrupt the integrity of the lesion wall, reduce the internal osmotic pressure, and permit osseous regeneration (Figures 30-11 and 30-12).

In 1982, Suzuki[54] suggested treating jaw cysts using an irrigational technique. In that study, the contents of 36 cysts were irrigated weekly for months and even years. This irrigation method involves the use of Ringer's solution, glucose, and antibiotics. The fluids aspirated from the cysts were quantitatively analyzed for electrolytes, inorganic substances, proteins, and lipids.[55] Irrigation of the lesions eventually resulted in the reduction in the volume and size of the cysts. The irrigation method is effective for the treatment of cysts in jaws.[54] Large cysts have been decompressed using acrylic stints, obturators, and tubing that extend into the lesion.[56–60] Acrylic stints or tubing was often left in for months with irrigation of the lesion. Neaverth and Berg described several cases of large lesion decompression that lasted from several weeks to more than a year.[59] The method used radioopaque tubing in conjunction with water irrigation by the patient. The tube was removed once there is evidence of elimination of the cystic lesion.

A surgical technique was described in case series format by Wong.[61] After flap reflection, a surgical fenestration was used to obtain some tissue for biopsy but the majority of bony defect was left intact. Copious drainage was accomplished and the defect irrigated with saline prior to suturing. This surgical treatment was effective in producing healing while avoiding potential complications. In 1997, Rees[62] reviewed and highlighted the treatment of large maxillary cysts by root canal treatment and subsequent decompression. The described technique used a drain made from surgical suction tubing. This seems to be the consensus treatment sequence currently in the dental literature. Figure 30-12 shows the radiographic and clinical appearance of decompression tube that was left in position for 1 week. At 1 week after placing the tube, the root canal was obturated. A 6-month follow-up radiograph shows bone filling in the apical lesion.

A 20-patient cohort study of decompression results by Enislidis and colleagues[63] is perhaps the best evidence of the technique's effectiveness. The authors describe the advantages as ease of treatment, confirmed diagnosis with biopsy, low morbidity, and low incidence of complications. Loushine[64] reported the removal of the decompression tube after only 2 days with follow-up examinations showing progressive osseous repair at 3, 6, and 12 months.[64] The use of decompression to treat odontogenic keratocyst (OKC) has also been reported by August et al.[65] A pediatric nasal airway was modified and placed in 14 OKCs for an average of 8.4 months. They were irrigated twice a day with chlorhexidine. At the time of cystectomy, 9 of 14 no longer showed histological features of OKCs. The epithelium had dedifferentiated and loss cytokeratin10 production in 64% of the patients.

Mejia et al.[66] reported in a case series format the use of a vacuum system within the root canal system. The technique produces a vacuum effect capable of removing copious amounts of exudate and inflammatory fluids. Perhaps the removal of the rather high osmolarity fluid and disruption of the bony defect lining is the impetus for subsequent healing.

The combination of aspiration and irrigation as an alternative to surgical endodontic treatment was reported by Hoen et al.[67] This case series demonstrated success using a single visit aspiration and saline irrigation of non-healing bony lesions associated with previously endodontically treated teeth. Following profound anesthesia, mucosa disinfection and aspiration of the cyst contents was accomplished using a 16 or 18 gauge needle attached to a syringe. Several milliliters of viscous aspirate were routinely obtained. The aspirates were submitted for aerobic and anaerobic culturing, Gram-staining, and immunoglobulin quantification. The level of IgG was significantly elevated in each specimen. Above normal levels of IgG have been shown to be consistent with cyst fluid.[55] It has also been shown that there is a high level of albumin and globulin in cysts compared to granulomas.[68] No bacteria were seen or cultured from any of the aspirates. At the 1 year follow-up appointments, the patients were asymptomatic and significant bony healing was seen on radiographs. It is important to develop a clear differential diagnosis and to have timely reevaluations of the patient's signs and symptoms to determine if further treatment is needed.[67]

An additional use of aspiration is to obtain a biopsy sample. August et al.[65] concluded that the use of needle aspiration for biopsy is a useful technique to distinguish between malignant and benign intraosseous jaw lesions. The described technique involved the use of a 10 ml syringe containing 1 or

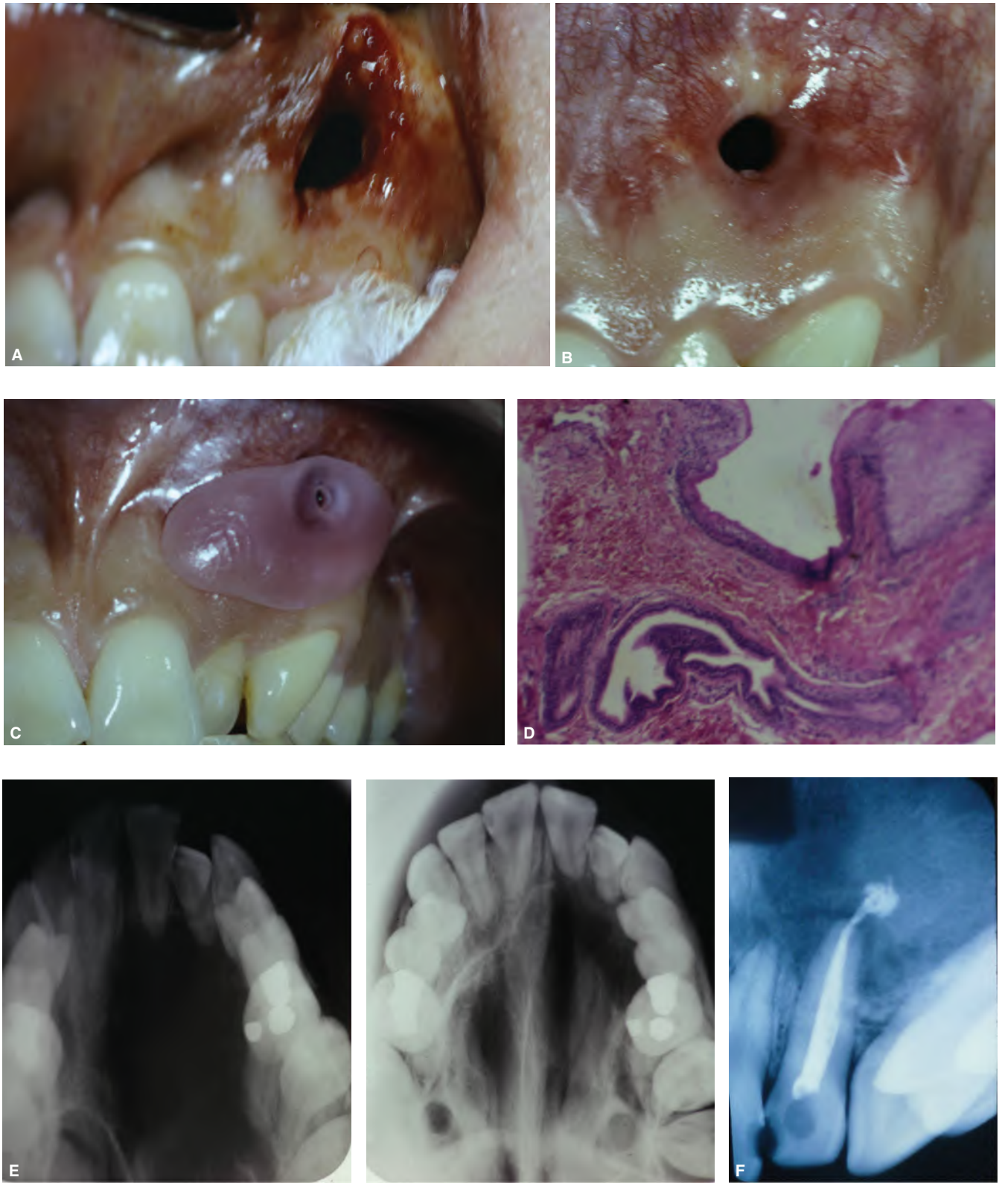


FIGURE 30-11 Decompression procedure. ***A.*** Surgical window into cyst. ***B.*** Healed surgical window. ***C.*** Acrylic stint in place. ***D.*** Biopsy from window consistent with radicular cyst. ***E.*** Palatal radiographs showing loss of bone on left and bone fill after 3 months of decompression (right). ***F.*** Maxillary left lateral incisor obturated after 3 months of decompression. (Courtesy of J. Craig Baumgartner, Kailua, HI, U.S.A.)

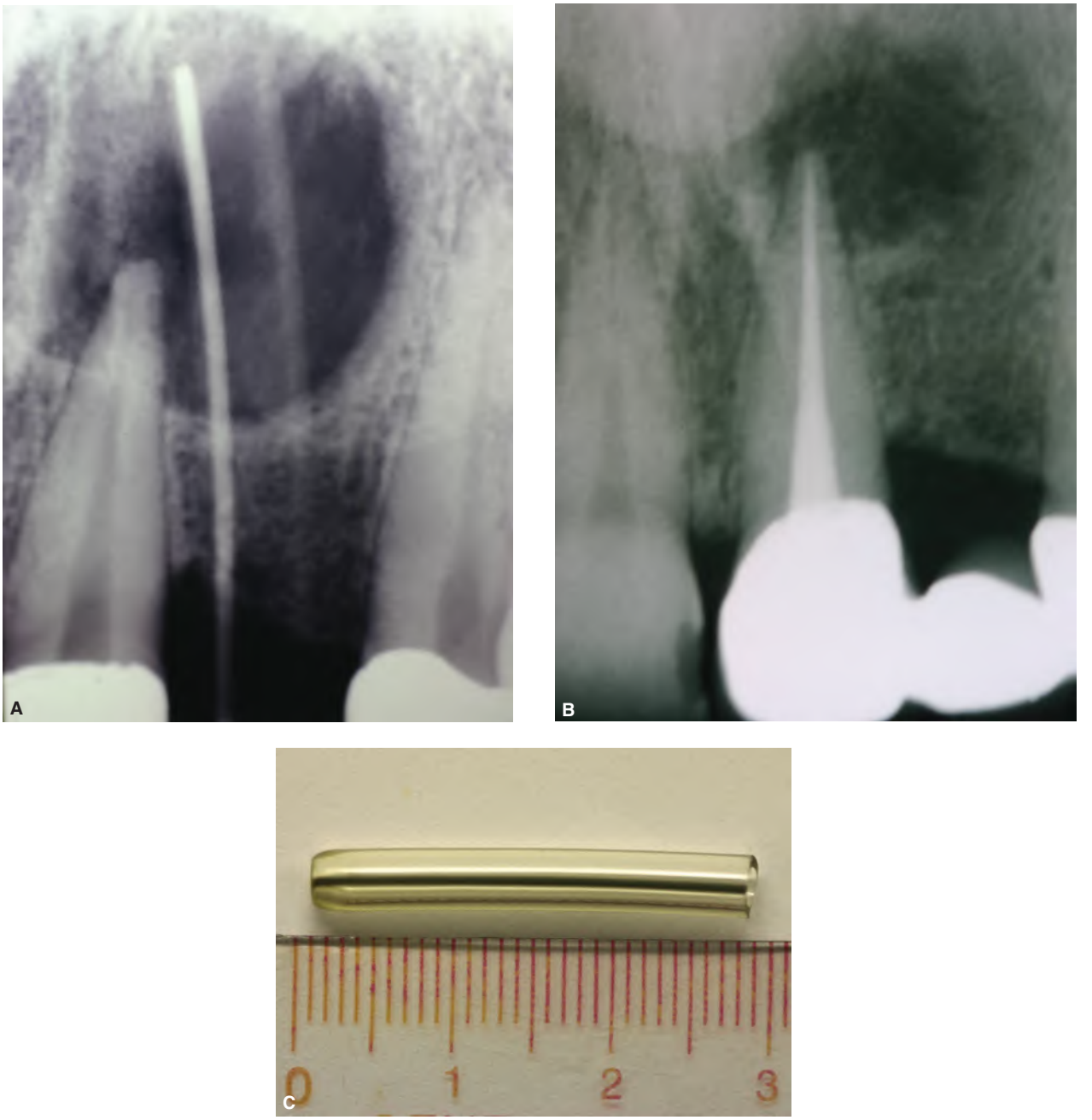


FIGURE 30-12 Cyst decompression. ***A.*** A 20 mm piece of nasogastric tubing placed in cyst for one week. ***B.*** Six month follow-up of non-surgical root canal filling. ***C.*** Tubing used next to ruler. (Courtesy of J. Craig Baumgartner, Kailua, HI, U.S.A.)

2 mls of air attached to a 23 or 25 gauge needles. Once within the lesion, suction was applied and several quick passes were performed to obtain cellular material. The specimens were then placed on glass slides for smear preparation. The authors further suggested that aspiration may be the diagnostic tool of choice in a hospital setting due to its simplicity, suitability as an outpatient procedure, rapidity of interpretation, and minimal morbidity.[65] The accuracy of fine-needle aspiration biopsy of head and neck tumors has been reviewed in 218 patients.[69] The technique was determined to be a useful modality for the diagnosis of head and neck tumors. The use of such a technique requires coordination with a pathologist familiar with needle biopsy specimens.

ENDODONTIC FLARE-UPS

A flare-up is an acute exacerbation of an asymptomatic pulp/or periapical pathosis after the initiation or continuation of root canal treatment.[10] Treating similar teeth in patients with comparable medical and dental histories, and the same endodontic technique, is no assurance of a common outcome. While one patient may remain asymptomatic, another

may have a flare-up. The contrasting clinical outcomes may seem to occur in a random manner or lead to erroneous conclusions about the cause–effect relationship of endodontic procedures to the flare-up. While some flare-ups have an iatrogenic component, others do not. The development of moderate to severe inter-appointment pain, with or without swelling, is an infrequent but challenging problem. Severe pain and swelling associated with flare-ups represent the clinical manifestation of complex pathologic changes occurring at a cellular level. There is increasing evidence pointing to multiple complex factors involved in producing a flare-up. These factors include mechanical, microbial, chemical, immunological, gender, and psychological components. The regulation of periapical inflammation is highly complex and represents another factor in patients' response to endodontic procedures.[70–75]

Incidence of Flare-ups

Meta-analysis is a statistical procedure that integrates the results of several independent studies considered to be combinable. Pooled data from multiple studies increases the sample size and power, thus providing a more precise estimate of a treatment's effect. The decision about whether or not results of individual studies are similar enough to be included in a meta-analysis is a critical issue.[76]

A meta-analysis of the results of previous studies concerning the incidence of endodontic flare-ups reviewed all relevant articles published in dental journals in English from 1966 to May 2007. Only six studies met all inclusion criteria. Prospective case series and clinical trials were included in the meta-analysis.

The average percentage of incidence of flare-ups for 982 patients was found to be 8.4%.[74] Differences in experimental design do not allow for a direct comparison of studies; however, the presence of pre-operative pain or mechanical allodynia (defined as a reduced mechanical pain threshold, or percussion sensitivity) was a positive predictor of post-operative pain in more than 15 studies involving more than 6,600 patients.[77]

Predisposing Conditions

When symptomatic pretreatment patients have been included in the study cohort, predisposing conditions include periapical abscess, acute apical periodontitis, pre-operative pain, and swelling.[74,78] Studies have found that the lowest incidence of flare-ups occurred in patients without periapical pathosis and when a sinus tract is present.[78] It is reasonable to hypothesize that a sinus tract allows drainage and prevents an increase in periapical tissue pressure.

A recent prospective clinical study evaluated 500 one-visit root canal treatments referred to an endodontist.[79] A predictive model of pain following treatment was developed. The study determined the probability of the incidence, intensity, duration and triggering of post-endodontic pain. Factors considered included the patient's age, gender, medical status, location of the tooth, number of canals, pulp vitality, preoperative pain, periapical radiolucencies, previous emergency access, and presence of occlusal contacts with opposing teeth.

The predictive models showed that the incidence of post-endodontic pain was significantly lower when the treated tooth was not a molar (P = 0.003), demonstrated periapical radiolucencies (P = 0.003), had no history of previous pain (P = 0.006), or emergency endodontic treatment (P = 0.045), and had no occlusal contact. The probability of experiencing moderate or severe pain was higher with increasing age (P = 0.09) and in mandibular teeth (P = 0.045). The probability of pain lasting more than 2 days was increased with age (P = 0.1) and decreased in males (P = 0.007) and when a radiolucent lesion was seen on radiographs (P = 0.1). Results indicated that the most important factor in predicting the incidence of post-endodontic pain is the absence of occlusal contacts. It was predicted that the patient with the highest probability of developing post-endodontic pain had previously experienced pain in a molar with prior endodontic treatment, no apical radiolucency and occlusal contacts. In contrast, a hypothetical patient with no previous pain in an incisor, a cuspid or a bicuspid with an apical radiolucency and free from occlusion will have a 0.07 probability of developing post-endodontic pain. Those odds would be a very good bet.[79]

The etiology of an exacerbation should be seen as multifactorial, and dependent on interactions between the host's immunological response, infection, and physical damage.

Studies evaluated factors related to post-operative endodontic pain and flare-up. The presence of pre-operative pain or mechanical allodynia (reduced mechanical pain threshold, or percussion sensitivity) was a significant predictor of post-operative pain in more than 15 studies with a large number of patients.[80] The etiology of a flare-up is multifactorial, and dependent on interactions between the host's immunological response, infection, and physical damage. The major causative factor has been described as microbial in origin.[76]

There are additional factors that may also predispose a patient to pain. They include genetics, gender, and anxiety. These non-dental factors are being evaluated, and at this time it seems likely that in the future their significance will be more fully understood. Although no single factor completely predicts the occurrence and severity of post-operative pain, an astute clinician should recognize that the presence of pre-operative pain or mechanical allodynia (sensitivity to percussion) is a warning sign that post-operative pain is likely. That warning sign is an indication that pain-preventive steps should be taken. They include pain-preventive post-operative analgesics and occlusal reduction when there is evidence of mechanical allodynia.

Occlusal Adjustment

A number of clinical studies have evaluated the pain-preventive value of occlusal adjustment. It is difficult to compare their results of the trials due to different methodologies, inclusion and exclusion requirements, and specific purposes of the study. A clinical study investigated the answers to

the following questions: "Are there specific clinical conditions that may indicate a need for occlusal reduction? Can a reliable clinical profile be developed of patients most likely to benefit from occlusal reduction?"[81] The research hypothesized that there may be specific pre-operative conditions that are statistically significant indicators for occlusal reduction following instrumentation. This approach varied from previous research that placed all endodontic cases together in a single group without accounting for the importance of clinical variables. Among the conditions evaluated was the presence or absence of pulp vitality, pre-operative pain, and percussion sensitivity, presence of a periapical radiolucency, a stoma, swelling, and a history of bruxism. The purpose of the study was to evaluate specific clinical factors as indicators for occlusal reduction following endodontic instrumentation. A statistically valid profile of patients most likely to benefit from occlusal reduction was developed. In that study of 117 patients, approximately twice as many patients (80%) with a diagnosis of irreversible pulpitis, who underwent occlusal reduction, reported no post-treatment pain when compared to control subjects with no occlusal reduction.[81]

Research Findings

Occlusal reduction was found to result in the prevention of post operative pain when any or all of the following indicators were present:

- Sensitivity to percussion
- Vital Tooth
- History of pain
- Absence of a periapical radiographic lesion

Even when all of those pain predictors were present, occlusal reduction resulted in the complete absence of post-operative pain. That remarkable result seems to be due to relieving occlusal stress from the periodontal ligament (Figure 30-13).[81]

Other studies concerning occlusal reduction have reached different conclusions.[82–84] Significant differences in exclusion and inclusion criteria and methodologies may account for the varied findings. For example, one study examined only teeth with "mild" sensitivity to occlusion, and excluded those with more severe sensitivity.[84]

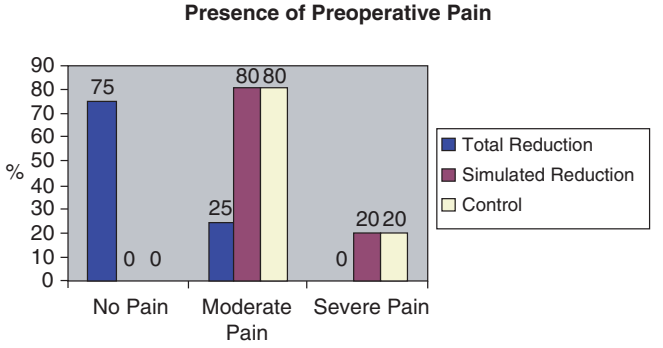


FIGURE 30-13 Effect of occlusal reduction on pain. (Courtesy of Paul A. Rosenberg, NY, U.S.A.)

Genetics

The role of genetics is an entirely new and exciting variable for dentists to consider. Genetics may play a role in predisposing some patients to a variety of complications including pain, poor healing, and abscess formation. Considering the role of genetics in endodontics is complex and at an early stage of development. Increasingly, endodontic literature provides examples of how genetics may influence endodontic symptoms and outcomes. Genetic variants may play an important role in the healing of apical lesions, abscess formation, and pain. Findings suggest that specific markers associated with the pro-inflammatory regulator Il-1B, a key regulator of host response, may contribute to increased susceptibility to periapical pathosis.[85] It has also been suggested that genetic factors are associated with a susceptibility to develop symptomatic dental abscesses.[86,87] These preliminary findings point to a complex mix of factors associated with patient's pain and treatment outcomes. It is possible that as more information is collected we will be better able to identify those patients predisposed to pain and have a diminished capacity for healing.[88]

Pain Responses: Male And Female

Although a person's biological sex exerts a major influence on their gender identity, "sex" and "gender" are not interchangeable terms. The term "sex" refers to biologically based differences, while the term "gender" refers to socially based phenomena. If research subjects are to be categorized by anatomical features (chromosomes, reproductive organs), it is appropriate to describe the study as one of "sex differences." In contrast, if additional measures of masculinity/femininity or gender identity are used to describe subjects, then the term "gender differences" is appropriate.[89]

During the last 10 to 15 years, there has been a growing body of evidence indicating that there are substantial sex differences in clinical and experimental pain responses for women and men. It seems that women are at a substantially greater risk for many clinical pain conditions. An extensive review reported that a survey of the currently available epidemiological and laboratory data indicates that there is overwhelming evidence for clinical and experimental sex differences in pain. Numerous reasons for these findings have been given, including hormonal and genetically driven sex differences in brain neurochemistry. Furthermore, some highly prevalent chronic pain syndromes that are found in both sexes (including chronic fatigue syndrome, fibromyalgia, interstitial cystitis and temporo mandibular disorder) occur overwhelmingly more often (in more than 80% of cases in which treatment is sought) in women.[89]

Vital and Nonvital Pulps

A clinician may categorize flare-ups as occurring in vital or nonvital cases. The vital case can be defined as a case in which the pulp is still capable of conducting an impulse to higher centers. It does not imply that the pulp is healthy. In fact, although a tooth may test vital it may contain areas of acute

inflammation, chronic inflammation, and necrotic tissue in a dynamic mix. However, as long as sensibility tests (thermal or electricity) provoke a response, the tooth should be considered vital. The challenge for the clinician, while treating a vital case, is to completely extirpate well-innervated tissue without shredding it or instrumenting through the apical foramina, causing additional inflammation to periapical tissues.

The nonvital pulp poses a different problem. If we consider the vital case to be primarily a challenge of managing inflamed tissue, the nonvital case represents a challenge of managing infected tissue. Thus it poses a microbial challenge. The dentist must avoid pushing microbes and necrotic tissue debris through and beyond the apical foramen into the periapical tissues. The resulting inflammatory/immune response is the basic cause of the swelling and pain associated with nonvital case flare-ups. A chronic nonvital case may flare-up even with meticulous care by the clinician. Instrumentation of a canal or irrigation may inadvertently cause a severe reaction in the periapical tissues due to extrusion of bacteria and tissue debris. As stated earlier, this is not a simple case of extrusion of debris being the only factor involved. There is increasing evidence that genetic and other biological factors may play a role in predisposing some patients to exacerbations.

Retreatment Cases

Retreatment cases, in most studies, have had a significantly higher incidence of flare-ups than conventional cases (Figure 30-14).[74,75] One study found an extremely high incidence of flare-ups (13.6%) in retreatment teeth with apical periodontitis.[78] It can be hypothesized that retreatment cases are often technically difficult to treat, and there is a tendency to push remnants of gutta-percha, solvents, and other debris into the periapical tissues. Microbes may also be pushed apically during the retreatment process. Extrusion of infected debris or solvents into the periapical tissues during preparation of the canals is allegedly one of the principal causes of postoperative pain.[90–92] Retreatment cases are usually associated with a persistent or secondary root canal infection by therapy-resistant microorganisms that may be more difficult to eradicate when compared to primary infections.[72,92,93] In contrast, others have found no statistical significance in the relation of retreatment to flare-ups.[75]

Working Length

The apical portion of the root canal system has been considered the most critical anatomic area with regard to the need for cleaning, disinfection, and sealing.[72,94] Over extended instrumentation should be avoided as it can result in postoperative pain.[75] Nonvital teeth associated with a periapical lesion, as well as root-filled teeth with recalcitrant lesions, represent a different biological challenge.[75] In those cases, microorganisms may be at or near the apical foramen and accessory foramina that are in close contact with the periapical tissues.[72,95–97] Correct working length in infected teeth is essential.[72,98] Inaccurate working length or inadvertent over- or under-instrumentation can result in negative outcomes for the patient. Over-instrumentation may force infected debris into the periapical tissues eliciting a severe inflammatory response and pain. Under-instrumentation will leave microorganisms in close proximity to the apical foramina where they or their virulence factors can gain access to the periapical tissues.[72,99] Incomplete instrumentation can disrupt the balance within the microbial flora and allow previously inhibited species to overgrow.[100] If those strains of bacteria are virulent and/or reach sufficient numbers, damage to the periapical tissues may be intensified and result in an exacerbation of the lesion. Furthermore, environmental changes, induced by incomplete debridement, have the potential to activate virulence genes.[73] A change in host resistance or microbial virulence may allow a previously

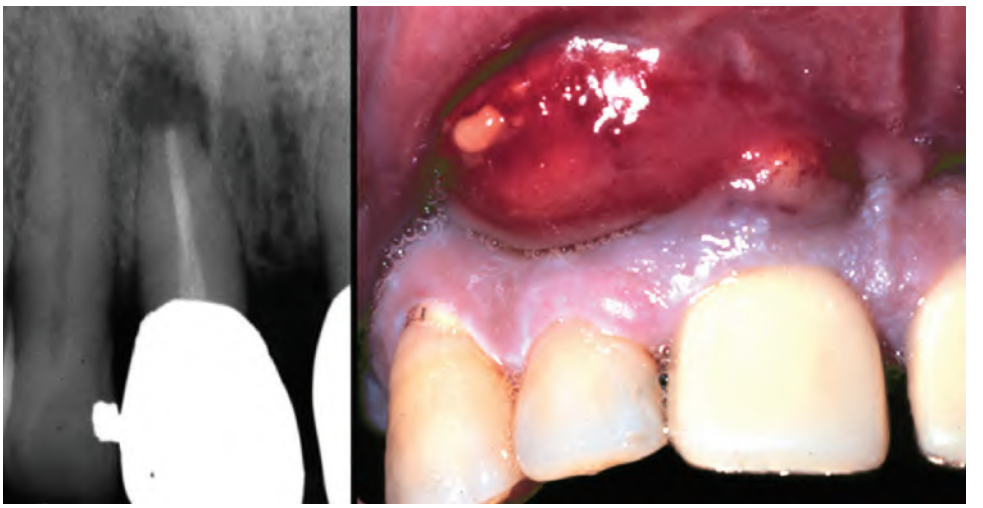


FIGURE 30-14 Swelling associated with flare-up following revision of previous endodontic treatment. (Courtesy of Paul A. Rosenberg, NY, U.S.A.)

asymptomatic situation to become symptomatic.[73] Clinical studies, however, have not linked incomplete canal preparation to flare-ups.[75,101,102]

Obturation

Overfilling can cause postoperative pain particularly when a substantial amount of filling material extrudes through the apical foramen. Gross overfilling involves the introduction of excess sealer (and its cytotoxic components) into the periapical tissues causing tissue damage and inflammation.[72]

A study found that overfilling was associated with significantly increased rate of pain and percussion sensitivity in 1-week follow-up examinations as compared with teeth not overfilled.[103] Scheduling of the obturation visit in relation to instrumentation may be another important factor. Obturation in the presence of acute apical periodontitis can be considered to be a predictor of postoperative pain. In order to avoid increased postoperative pain, patients who present for obturation but have significant acute apical periodontitis should have the procedure postponed until the tooth is more comfortable. Relief of pain can be achieved by treatment directed at reducing tissue levels of factors that stimulate peripheral terminals of nociceptors or by reducing mechanical stimulation of sensitized nociceptors (e.g., occlusal adjustment). Thus by deferring obturation of a tooth with pericementitis, further stimulation of sensitized nociceptors is avoided.[104]

Strategies to Prevent Flare-Ups

Variability in patients' pain sensitivity, perception, and tolerance may explain why they may react differently to endodontic treatment. Additional factors that may dispose patients to pain include genetics, level of anxiety, and their sex. Because there are a numerous factors associated with endodontic flare-ups an effective pain-preventive strategy must be multifaceted.[105–108]

Anxiety Reduction

High levels of stress, anxiety, or pessimism predict poor outcomes in measures that range from speed of wound healing to duration of hospital stay. Over 200 studies indicate that preemptive behavioral intervention, to decrease anxiety before and after surgery, reduces postoperative pain intensity and intake of analgesics improves treatment compliance, cardiovascular and respiratory indices, and accelerates recovery.[109] In a landmark study,[110] it was found that preoperative discussion of likely postsurgical treatments and associated discomfort halved the requirement for postoperative morphine and reduced time to discharge. Patients in that study also received instructions in a relaxation technique. Providing information about the procedure is an important step in preparing patients for endodontic treatment. Information about profound dental anesthesia and preventive pain strategies is an important anxiety reduction technique. Perhaps most importantly, the dentist should assure the patient that pain prevention is a primary concern. It was determined that patients given a running commentary concerning procedures and associated sensations rated themselves as less anxious and experiencing less pain than a normal control group.[111] Information about sensations experienced during treatment as well as a description of procedures appears to have a significant impact in reducing patient anxiety.[111]

Pharmacologic Strategies for Flare-Ups

Antibiotics

Antibiotics are frequently recommended to endodontic patients without a rational biologic basis.[112,113] An evidence-based review determined that the use of systemic antibiotics for the prevention of post-treatment endodontic pain should be discouraged.[114] As previously stated, antibiotic treatment is generally not recommended for healthy patients with localized endodontic infections. Systemic antibiotics should be considered if there is a spreading infection that indicates failure of local host responses to control bacterial irritants or the patient has a medical condition that compromises defense mechanisms and could expose the patient to higher systemic risks.[114]

NSAIDs and Acetaminophen

Nonsteroidal anti-inflammatory drugs (NSAIDs) have been shown to be effective for managing pulpal and periapical pain.[104,115] However, due to the renal effects of NSAIDs as well as interactions with many anti-hypertensive drugs, acetaminophen should be considered for post-treatment pain in patients with known sensitivity to NSAIDs or aspirin. Acetaminophen should also be considered for those with the following disorders: ulcers, ulcerative colitis, asthma, or hypertension. Pretreatment with NSAIDs or acetaminophen has also been shown to be effective for reducing postoperative pain.[73,116] Pretreatment with NSAIDs for irreversible pulpitis should have the effect of reducing pulpal levels of the inflammatory mediator prostaglandin E2 (PGE2).[73,117]

Recently, the combination of ibuprofen/acetaminophen (paracetamol) has been advocated for use in an effort to avoid unpleasant side effects associated with opioids. There is good evidence that supports the use of this drug combination for the treatment of moderate to severe pain. A powerful clinical study used a double blind design that was placebo controlled randomized, single dose, and utilized three sites.[118] The study was designed to compare the efficacy and tolerability of different analgesic combinations, including a novel-single tablet combination of ibuprofen/paracetamol (acetaminophen). It investigated moderate to severe post-operative dental pain following extraction of at least three impacted molars, two of which were mandibular molars.

The study compared the efficacy of the following analgesics:

- Placebo
- Acetaminophen 500 mg/codeine 15 mg
- Ibuprofen 200 mg/codeine 12.8 mg
- Ibuprofen 200 mg/acetaminophen 500 mg
- Ibuprofen 400 mg/acetaminophen 500 mg

The results ranked the five treatments from best to worst:

- Two tablets of ibuprofen 200 mg/acetaminophen 500 mg
- One tablet of ibuprofen 200 mg/acetaminophen 500 mg
- Two tablets of ibuprofen 200 mg/codeine 12.8 mg
- Two tablets of acetaminophen 500 mg/codeine 15 mg
- Placebo

This study found that one or two tablets of the single-tablet combination of ibuprofen 200 mg/acetaminophen 500 mg was statistically significantly more effective than two tablets of acetaminophen/codeine or one tablet of the ibuprofen/acetaminophen combination. The peak pain relief was found to be higher and more sustained in patients taking two tablets of the single-tablet combination of ibuprofen/acetaminophen (*p* values compared with all other treatments). The proportion of subjects reporting adverse effects was significantly less with either one or two tablets of the single tablet combination of ibuprofen/acetaminophen compared with the codeine analgesic combinations. Codeine is known to be associated with several side effects, including nausea and vomiting.[118]

Combining ibuprofen with acetaminophen (APAP) provides dentists with an additional therapeutic strategy for managing moderate-acute postoperative dental pain. This combination has been reported to provide greater analgesia without significantly increasing the adverse effects that often are associated with opioid-containing analgesic combinations. The strategy of combining two analgesic agents having distinct mechanisms or sites of action, such as combining a peripherally acting analgesic with a centrally acting analgesic, has been advocated for many years. Analgesic formulations containing an opioid and a peripherally acting analgesic consistently provide greater pain relief than do the component agents when administered alone.

It is advisable to have endodontic patients take their analgesics "by the clock" rather than on an "as needed basis.[116] Patients should take an NSAID or acetaminophen just prior to, or immediately after, treatment. If they wait to take medication until after the onset of pain, there is usually a delay of up to 1 hour before they experience pain relief. It has been suggested that instructing patients to take their analgesics by the clock for the first few days provides a more consistent blood level of the drug and may contribute to more consistent pain relief.[117] The combination of ibuprofen and acetaminophen taken together has been shown to produce additive analgesia when treating dental pain.[104,116,119–121] Opioids may be added when indicated.

Long-acting Local Anesthetics

Long-acting local anesthetics (e.g., bupivicaine) can provide an increased period of post-treatment analgesia beyond the usual duration of anesthesia.[122,123] By blocking the activation of unmyelinated C-fiber nociceptors, the anesthetic decreases the potential for central sensitization.[104] Long-acting local anesthetics can provide a period of analgesia for up to 8 to 10 hours following block injections and may reduce pain even 48 hours later.[104,123] Use of long-acting local anesthetics is a valuable biologically based strategy that provides analgesia during the immediate postoperative period.[104] Endodontic treatment by itself can be expected to provide significant pain relief.

Treatment of Endodontic Flare-Ups

Selecting the appropriate treatment after an endodontic flare-up is dependent upon understanding its biological cause. For example, the clinician must determine if a flare-up is primarily iatrogenic in nature, as in the case of inaccurate canal measurement control, gross overfilling or microbiologically based, as in a necrotic case.

Diagnosis and Definitive Treatment

Treatment is always directed at the cause of the problem. If a nonvital tooth has not been fully instrumented, the canal must be instrumented and irrigated in order to eliminate the microbiological cause of the problem. Pain relief in the over-instrumented case is often dependent on an analgesic strategy. The under-instrumented case may require further instrumentation to the correct measurement, as well as the use of analgesics. Supplementary antibiotics are usually not necessary unless there is a significant swelling that appears to be spreading. History of the onset of pain is important in determining if the pain is spontaneous or provoked by a specific stimulus. For example, if a tooth had a history of acute apical periodontitis and its occlusion had not been reduced, that could be identified as a probable cause of postoperative pain.[81] In contrast, a complaint of swelling, pressure, and throbbing in the interproximal area might suggest a periodontal component of the problem that should be explored. If inaccurate measurement control was used or proper measurement not maintained, the clinician must determine if the canal was under- or over-instrumented. Working length should be reconfirmed, patency to the apical foramen obtained, and thorough debridement with copious irrigation completed. Remaining tissue, microorganisms and their products, and extrusion beyond the apex are major factors responsible for post-treatment symptoms.[124] Abscesse/cellulitis associated with a flare-up should be treated as previously described.

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Chapter 31

Management Considerations for the Medically Complex Endodontic Patient

Bradford R. Johnson, Joel B. Epstein

One of the challenges facing dental specialists today is the assessment and management of patients with increasingly complex medical conditions. Not only has the average life expectancy increased dramatically over the past 50 years, but our elderly patients are much more likely to be at least partially dentulous and may have a complex medical history with multiple medical problems and use of numerous medications.[1] Approximately 25% of patients ages 65 to 74 and 35% of patients age 75 and older have a medical condition that would place them in ASA* category III or IV.[2] An aging population with both the desire and resources to preserve their natural dentition will drive the demand for root canal therapy for patients with complex medical conditions. Even in a typical population of younger and presumably healthier patients, approximately 50% of patients referred to a dental specialty practice may be expected to report at least one medical finding on their health history questionnaire.[1,3] Since medical complexity is often an indication for referral to a dental specialist,[4,5] endodontists should be prepared to accurately evaluate medically complex patients and identify situations that require a modification of normal treatment procedures and identify oral and systemic conditions requiring diagnosis and management.

This chapter provides an overview of well-known medical conditions requiring modification of treatment protocol ensuring safe endodontic treatment in an ambulatory dental setting. It is not intended as a substitute for case specific clinical judgment or consultation with medical experts. For purposes of this chapter, a medically complex patient will be defined as any patient requiring modification of the usual treatment procedure.

MEDICAL HISTORY AND PATIENT INTERVIEW

"Never Treat a Stranger" (Attributed to Sir William Osler)

The value of a thorough medical history and patient interview cannot be overemphasized. Recognition of a medical condition that requires treatment modification *prior* to treatment can avert significant treatment complications. Approximately 25% to 30% of patients seeking treatment in a dental office can be expected to report at least one medical condition that has potential relevance to dental treatment, although not all of these conditions will require treatment modification.[6,7] An adverse outcome, due to failure to recognize a known risk factor and modify treatment accordingly, is a major predictor of a successful liability claim.[8] Cardiovascular disease, drug allergies, diabetes, and concerns about the safety of vasoconstrictor use are some of the most common medical reasons for referral to a specialist.[1,9,10] This is consistent with an analysis of the most common medical emergencies in the dental office: angina, hypoglycemia, adverse reaction to local anesthetics, and seizures.[11]

A standard health history questionnaire should cover all common medical conditions (both treated and untreated), surgeries, hospitalizations, medications, and allergies. Although many standard forms are readily available and are usually convenient and easy to complete, most of these forms do not clearly lead to a specific determination of risk for dental treatment. More useful medical risk assessment models are under development and will be briefly discussed later in this chapter. The written health history questionnaire should always be supplemented with a patient interview to help decrease false positive and false negative findings and to further explore positive findings.[12] The clinician should review any positive findings with the patient and determine the patient's level of compliance with medical treatment recommendations. For example, a compliant patient, with well-managed hypertension, presents a relatively low risk, compared to a patient who refuses to take prescribed medications, or does so erratically. Unfortunately, for a variety of reasons, the reliability of self reported information in the health history may be less than ideal. Patients may simply forget to report important medical information, but it has also been shown that some patients will intentionally omit relevant information due to concerns over privacy or failure to understand how the information could be relevant to dental practice.[13,14]

Medications and Allergies

The list of medications and allergies should be consistent with disclosed medical conditions, and can alert the clinician to unlisted medical conditions as well as potential drug interactions. Relevant medical conditions and severity of systemic disease can often be determined by a careful analysis of the patient's list of medications.[15]

Herbs, dietary supplements, vitamins, and other over-the-counter (OTC) medications can contribute to complications in the dental setting although patients often fail to report

use of these substances in the initial evaluation.[16] In a recent survey of surgical patients, approximately one-third reported use of a non-prescription medication that could potentially inhibit coagulation or interact with anesthetics.[17] In particular, *Ginkgo biloba*, ginger, garlic, ginseng, feverfew, and vitamin E all inhibit platelet aggregation and can increase the risk of bleeding.[18] Ingredients in some OTC weight loss products can potentiate the effect of epinephrine and increase cardiac stress. The most obvious example of this phenomenon, ephedra, has been removed from the U.S. market by FDA order.

Previous Dental Treatment

A standard screening question for all patients should enquire about any problems with previous dental treatment. This line of questioning serves several important functions. First, it allows the patient to discuss any previous negative dental experiences as well as express possible anxiety related to the proposed treatment. A report of difficulty in achieving profound local anesthesia is a common finding, especially for root canal therapy. This provides an opportunity to demonstrate concern for one's patient and discuss how one plans to avoid a repeat of the previous experience. Second, potential adverse reactions to dental materials or drugs may emerge in response to this question. Finally, since this question is designed at least in part to help develop rapport with your patient, it can serve as a good lead into other important but potentially more sensitive questions (e.g., use of oral contraceptives, history of HIV).

Physical Exam—Vital Signs

In addition to the mandatory health history questionnaire and patient interview, vital signs (blood pressure, heart rate, respiratory rate, temperature, height, and weight) should be recorded prior to dental treatment whenever possible. In particular, blood pressure, heart rate, and respiratory rate provide essential risk assessment baseline information for all patients. Temperature may be routinely recorded but is specifically indicated in the presence of infection or signs of generalized malaise or toxicity. Height and weight can usually be obtained from the patient or guardian, and this information is particularly important in determining appropriate drug dosages in pediatric and geriatric patients and assessing unexplained changes in weight.

Although routine medical lab screening tests are not currently standard practice in the majority of dental offices, a recent multi-center study conducted in an academic dental school environment found that 83% of study participants had one or more abnormal test results and did not report a relevant explanatory disease in their medical history.[19] This finding suggests that many patients may be unaware of existing medical condition(s).

Relative Stress of the Planned Procedure and Behavioral Considerations

A patient's ability to tolerate the stress of dental treatment depends on the procedure planned, time required to complete the procedure, physical health status, and psychological factors. Anticipation of a stressful dental procedure can increase the heart rate and endogenous secretion of epinephrine in otherwise healthy adults.[20,21] In fact, even anticipation of a routine dental checkup can result in increased blood pressure in some patients.[22] Most changes in heart rate and blood pressure are within the normal physiologic range although more significant changes have been observed before administration of local anesthetic, during subgingival scaling and during extractions.[23] Since there are no specific guidelines for assigning stress levels to various dental procedures, clinical judgment is essential in determining whether or not stress reducing treatment modifications should be employed. Dental specialists, by virtue of additional training and experience, should be expected to provide treatment in a shorter time period and should be better prepared to manage complications than a general practitioner.

Endodontic treatment in general is often considered a high stress dental visit, especially among patients with no prior endodontic treatment experience, or patients who have had a previous negative experience with endodontic treatment. For many, the perception of the treatment often differs from the reality, that most current endodontic treatment is minimally invasive, relatively comfortable, efficient, and very well tolerated by the majority of patients when skillfully performed. In a study that measured salivary cortisol levels in patients undergoing a variety of dental procedures, root canal treatment was no different than a routine dental exam, prophylaxis, or restorative treatment and only tooth extraction resulted in a significant increase in salivary cortisol.[24] Surgical endodontic procedures, the presence of acute pain, self reported dental anxiety or difficulty with previous treatment, and lengthy procedures would all be expected to increase the level of stress.[23,25,26] If any of these conditions are present in addition to significant systemic disease, treatment modification including a stress reduction protocol should be considered.

Physical Health Status

The American Society of Anesthesiologists' (ASA) Health Classification System is the most widely used system for assessing physical health status, and helps determine the potential need for medical consultation and treatment modifications prior to dental and/or medical procedures (Table 31-1).

In general, ASA I status represents a healthy patient who does not require any treatment modification. An ASA II patient presents with well-controlled systemic disease and usually will not require significant treatment modification although stress reduction may be indicated. Patients in the ASA III category or above will almost always require medical consultation and possible treatment modifications. On the other hand, the ASA classification system has several significant limitations and should be used only as a general guide for determining pre- and post-operative risk. Even experienced anesthesiologists exhibit differences of opinion

TABLE 31-1 American Society of Anesthesiologists (ASA) Health Classification System and Treatment Modifications*

ASA physical classification	Description	Therapy modifications (McCarthy and Malamed, 1979)
ASA 1	Normal healthy patient	None (stress reduction as indicated)
ASA 2	Patient with mild systemic disease	Possible stress reduction and other modifications as needed
ASA 3	Patient with severe systemic disease that limits activity, but is not incapacitating	Possible strict modifications, stress reduction, and medical consultation are priorities
ASA 4	Patient with incapacitating systemic disease that is a constant threat to life	Minimal emergency care in office (may consist of pharmacologic management only); hospitalize for stressful elective treatment; medical consultation urged
ASA 5	Moribund patient who is not expected to survive the operation	Treatment in the hospital is limited to life support only, for example, airway and hemorrhage management
ASA 6	Declared brain-dead patient whose organs are being removed for donor purposes	Not applicable

*Adapted from Tables 1 and 2, Goodchild J, Glick M [29]

in the classification of cases.[27,28] In addition, when used alone, the ASA system is not a good predictor of operative risk.[29] Various authors have proposed a medical risk assessment process that focuses more clearly on medical conditions specifically relevant to dental practice.[2,6,29,30] Clinicians may want to consider health history questionnaires and verbal questions that focus primarily on medical conditions that would, when present, elevate the patient to ASA II status or above. That is, any positive response to one of the health history questions would automatically classify the patient, as at least ASA II and further exploration would be necessary to determine the extent of systemic disease. de Jong et al.[12,30,31] have developed and validated a patient questionnaire consisting of approximately 30 questions for use in assessing risk for dental treatment.

Medical Consultations

Approximately one-third of patients referred for a medical consultation result in recommendations for some modification of treatment procedures.[9] A request for consultation may occur by phone or letter and each approach has certain advantages and disadvantages. A phone conversation is immediate and may allow for the discovery of additional useful information. However, the lack of a written letter from the physician increases the risk for potential misunderstanding and provides a lower level of documentation from a medico-legal standpoint. The substance of all medical consultation conversations should be documented in the patient's record and, whenever possible, a letter detailing the physician's recommendations should be requested. A letter provides more formal documentation of the communication between health care providers. An additional consideration is current security standards for protecting certain health information that is transferred in electronic form (e.g., email) established by the HIPPA Security Rule. Encryption of email is now required if patient health information is exchanged via this format.

When requesting a medical consultation, the clinician should be specific and concise. The medical condition in question should be identified and the nature of dental procedure planned should be described (e.g., brief description of the procedure, type of anesthetic planned, potential for bleeding, expected length of procedure). The clinician should specifically ask the physician if the patient's medical condition requires any modification in the usual treatment protocol and, if so, what modifications are recommended. Since the ultimate responsibility for providing safe treatment rests with the dentist, any concerns about recommended treatment modifications should be resolved with the physician prior to treatment. Consultation may also be obtained from dental specialists with expertise and experience in a specific oral or medical condition, such as immunosuppressed or in cancer patients. In such a case, documentation as above is suggested.

ASSESSING THE NEED FOR TREATMENT MODIFICATIONS

Multi-Dimension Risk Assessment Model (MD-RAM)

Systemic disease can result in the loss of reserve capacity to handle stress.[15] A patient's ability to handle stress decreases in direct relation to the extent of systemic disease. The primary components of risk assessment for dental treatment are: physical health status, emotional or psychological status, and type of dental procedure planned.[32] Lapointe et al.[15] proposed a two-dimensional risk assessment model that correlated severity of disease and procedural stress. However, this model presented only two specific medical conditions as examples (ischemic heart disease and chronic obstructive pulmonary disease) and did not explicitly consider patient anxiety as a variable. Dental anxiety can increase sympathetic activity and can for some patients potentially precipitate a medical emergency.[33]

We propose a new model, the Multi-Dimension Risk Assessment Model (Figure 31-1 and Table 31-2) that incorporates the three primary aspects of risk assessment into a unified approach for evaluating perioperative risk related to treatment for the ambulatory dental patient. In this model, the dental patient is assigned a score ranging from 1 to 4 in each of three domains: physical health status (using the ASA classification system); procedural stress (type of procedure, length of time to complete the procedure, and clinician expertise) (Table 31-3); and psychological status (self-reported dental anxiety) (Table 31-4).

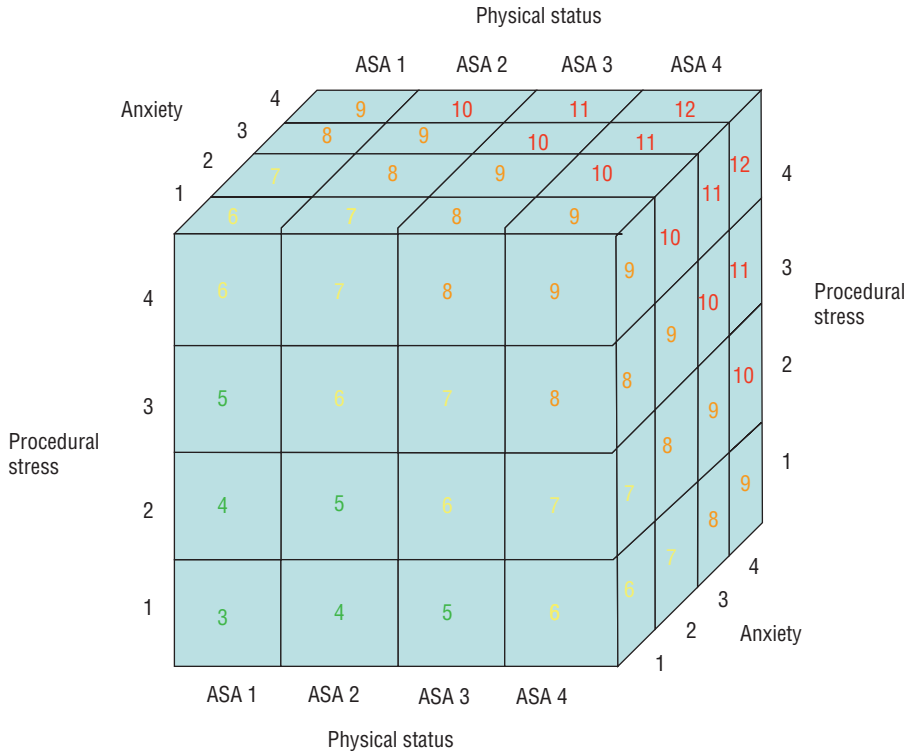

FIGURE 31-1 Multi-Dimension Risk Assessment Model (MD-RAM) (January 2015).

TABLE 31-2 Interpretation of Multi-Dimension Risk Assessment Model (MD-RAM) Scores

MID-RAM score	Interpretation
3-5	No treatment modification usually indicated
6-7	Possible medical consultation and treatment modification
8-9	Medical consultation often indicated and probable treatment modification
10-12	Medical consultation strongly advised; may require treatment in a hospital or specially equipped outpatient facility

TABLE 31-3 Estimates of Procedural Stress

Procedural stress scale	Examples*
1	Denture adjustment; noninvasive oral exam; radiographs
2	Procedures requiring local anesthesia; prophylaxis with subgingival scaling; simple restorative procedures; uncomplicated nonsurgical root canal treatment
3	Patients with acute pain and/or significant infection; extractions; surgical root canal; periodontal surgery
4	Bony impactions; trauma surgery

*Clinician should consider possible upgrading if procedure is lengthy and move either up or down depending on clinician's experience. (Adapted in part from Lapoint et al. [15])

TABLE 31-4 Behavioral Scale: Patient's Self-Report of Dental Anxiety

Dental anxiety scale	Verbal descriptor
1	No anxiety
2	Mild anxiety
3	Moderate anxiety
4	Severe anxiety

As with any model, the output is only as good as the information input. This model may provide a general guide for patient assessment and is not intended to be all-inclusive or substitute for case-specific clinical judgment. The primary purpose of this model is to assist clinicians in determining if treatment modification, often a stress reduction protocol, may be indicated prior to dental treatment. Disease-specific recommendations will be discussed later in this chapter.

Cardiovascular Disease

Heart disease is the leading cause of death in the United States[34] and can present challenges for safe patient management in the dental office. A possible association between apical periodontitis and coronary artery disease has been suggested, although it is very important to note that a cause and effect relationship has not been established.[35,36] An association between periodontal disease and atherosclerotic vascular disease has also been discussed but, again, causation has not been established.[37]

An interesting positive relationship between the use of systemic statins (one of the most commonly prescribed classes of drugs) and pulp chamber calcification was recently described.[38]

Hypertension

Hypertension is one of the most common medical conditions likely to be encountered in a dental office. Hypertensive patients are commonly defined as those receiving treatment for hypertension or those with a mean systolic blood pressure (SBP) of 140 mm Hg or greater and/or a mean diastolic pressure (DBP) of 90 mm Hg or greater. By this definition, approximately 31% of the adult population of the United States is hypertensive with about half of this group untreated and only one quarter receiving successful treatment for hypertension.[39] Since hypertension is typically asymptomatic, approximately one-third of patients with high blood pressure are unaware of their condition.[40] Even patients with normal blood pressure at ages 55 to 65 years have an almost 90% risk of developing hypertension by ages 80 to 85 years.[41] Patients with untreated or inadequately treated hypertension are at significantly increased risk for acute complications such as myocardial infarction and stroke and chronic complications of hypertension.

The clinical significance to the dental practitioner is clear since at least 15% of all adult dental patients have either untreated or inadequately treated hypertension; initial blood pressure measurement is an essential screening tool prior to dental treatment. In addition, it is appropriate to measure blood pressure at least annually during recall visits for all patients and at every visit for patients when an invasive dental procedure is planned.

Guidelines for the management of blood pressure were recently revised in the eighth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 8, 2013)[42] (Table 31-5). From a practical standpoint, patients with well-controlled hypertension or SBP below 160 and DBP below 100 should tolerate all routine dental procedures,[43] although referral for evaluation by the patient's physician is appropriate due to the well-documented benefits of maintaining blood pressure in the normal range. Patients with SBP between 160 and 180 or DBP between 100 and 110 should also be able to tolerate most dental procedures without significantly increased risk of perioperative cardiovascular complications, however complexity and stress of the planned treatment should be carefully considered with attention given to stress reduction strategies prior to treatment.[44] Although clear guidelines for establishing a cut-off point for dental treatment (emergency or routine) are lacking, it is generally accepted that patients with SBP greater than 180 and/or DBP greater than 110 should be referred for medical consultation and treatment prior to dental treatment and only emergency management of pain or acute infection should be considered.[44] Patients with SBP above 210 and/or DBP above 120 should be referred for emergent medical evaluation.

TABLE 31-5 Current 2014 Guidelines for Management of Hypertension Eighth Joint National Committee Report

Age	Blood pressure goal
≥ 60 years old	SBP < 150 mm Hg DBP < 90 mm Hg
< 60 years old (or any age with DM or CKD)	SBP < 140 mm Hg DBP < 90 mm Hg

(Adapted from James PA, et al. [42])

Vasoconstrictor Use in Patients with Cardiovascular Disease

Vasoconstrictors are used routinely in dental therapy including endodontics, as a component of local anesthetics and often as a hemostatic agent during periapical surgery. Local anesthetic with a vasoconstrictor, most commonly lidocaine with 1:100,000 epinephrine, is the usual anesthetic of choice for root canal therapy although many non-surgical procedures can be performed using local anesthetic without vasoconstrictor.[45] When a vasoconstrictor is indicated, epinephrine is preferred over levonordefrin due to a decreased potential for alpha-1 receptor stimulation.[46] Surgical procedures typically require greater quantities of local anesthetic with a vasoconstrictor than nonsurgical root canal treatment. In particular, patients with advanced cardiovascular disease, geriatric patients, and patients taking certain medications (e.g., MAO inhibitors and non-selective beta blockers) may have a reduced tolerance for vasoconstrictor containing local anesthetics. Since local anesthetic with a vasoconstrictor is very helpful in obtaining adequate hemostasis and visibility during periapical surgery, it may be difficult to perform the procedure using anesthetics without vasoconstrictors.[47]

Use of local anesthetics with vasoconstrictors in patients with cardiovascular disease has been somewhat controversial and was addressed by the previous JNC 7 report, but not discussed in the current JNC 8 guidelines. While the general goal should be to minimize the use of vasoconstrictors in patients with cardiovascular disease, the benefit of greater depth and duration of anesthesia when local anesthetics with a vasoconstrictor are used is a significant argument in favor of their use. Adequate pain control is an essential component of endodontic therapy since pain related stress could stimulate the release of significant quantities of endogenous catecholamines.

Different kinds of stress can increase the release of endogenous epinephrine by as much as 20 to 40 times over baseline values.[48] Pooled results from six studies of patients undergoing extraction or other minor oral surgery procedure demonstrated an average increase in SBP and DBP of 11.7 mm Hg and 3.3 mm Hg respectively as well as an increase in heart rate of 4.7 beats per minute. The use of local anesthetic containing epinephrine for these procedures resulted in an additional relatively minor increase in SBP of 4 mm Hg and six beats per minute.[49] Careful aspiration and use of adequate gauge needle to facilitate aspiration during injection is required to minimize the chance

of intravascular injection. Most authors feel that 0.036 mg of epinephrine (approximately two cartridges of local anesthetic with 1:100,000 epinephrine) should be safe for patients with cardiovascular disease except those with severe disease or other specific risk factors, and those with SBP requiring urgent medical attention.[44,49,50] 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.

An important exception to this general rule regarding vasoconstrictors is choice of local anesthetic for intraosseous (IO) injections in patients with cardiovascular disease. IO injections are most commonly used as a supplemental anesthetic technique for teeth that are otherwise difficult to anesthetize. A transient increase in heart rate can be expected in about two-thirds of patients receiving an IO injection using lidocaine with 1:100,000 epinephrine although heart rate returns to near baseline within 4 minutes after injection.[51] No increase in heart rate was found in patients receiving an IO injection with a 3% mepivacaine solution. Although in this study no significant change in blood pressure was found in either group, the authors recommend 3% mepivacaine without vasoconstrictor for IO injection in patients with any medical condition that could reduce their tolerance for epinephrine.

Although most experts agree that use of gingival retraction cord containing epinephrine should be avoided in patients with significant cardiovascular disease,[44,49] some uncertainty exists over the use of racemic epinephrine impregnated pellets as recently advocated for improved hemostasis during periapical surgery.[47,52] In two clinical studies, no significant changes were observed in blood pressure or heart rate when epinephrine pellets (either cotton or collagen) were placed in the bony crypt to improve hemostasis during periapical surgery.[53,54] It is unlikely that a patient healthy enough to tolerate two or three cartridges of local anesthetic with 1:100,000 epinephrine would experience any untoward effects from the proper use of epinephrine impregnated pellets during periapical surgery. Regardless, other alternative topical hemostatic agents are available and should be considered for patients with significant cardiovascular disease.

Ischemic Heart Disease

When coronary atherosclerotic heart disease becomes sufficiently advanced to produce symptoms, it is referred to as ischemic heart disease. Ischemic heart disease is relatively common in the general population, especially with increasing age, and typically presents as angina or heart failure.[55] In most cases, diminished blood perfusion of the myocardium due to coronary artery disease (atherosclerosis) is the underlying cause with hypertension as a common contributing factor in heart failure. Congestive heart failure and enlargement of the heart result from weakening of the damaged heart muscle.

Chest pain, secondary to ischemic heart disease, develops when the oxygen demand of the myocardium exceeds the oxygen supply. Transient pain, referred to as angina pectoris, is often described as an aching, squeezing sensation or tightness in the middle of the chest. Angina is often precipitated by physical activity or stress and may radiate to the arm or jaw and may present as facial or dental pain. Fear and anxiety associated with dental treatment may be a precipitating factor for angina in some patients.[55] Of particular significance to dentists is the finding that orofacial pain may be the sole symptom of cardiac ischemia in approximately 6% of patients presenting with myocardial infarction. The ratio of bilateral to unilateral orofacial pain in this group of patients was 6:1.[56,57]

Sublingual or other forms of nitrates are the standard treatment for angina and should result in rapid reversal of symptoms. Patients should always be instructed to bring their anti-anginal medicine with them for dental appointments. If symptoms are not relieved with oral nitrates and suspension of stress inducing activity, then myocardial infarction (MI) should be suspected and immediate emergency treatment should be initiated. Since angina is usually a transient event, patients with progressive pain or pain at rest are considered to have unstable angina and present a significant perioperative risk for MI.[58] These patients are usually categorized as ASA IV. Chest pain that is manageable with rest or medication and relatively unchanged in duration, frequency, or severity over time is termed stable angina and represents a better prognosis and a somewhat lower risk level than unstable angina and typically classified as ASA II or ASA III.

Compared to other surgical procedures, most dental and oral surgery procedures are considered relatively low risk.[59,60] However, a history of significant cardiac disease can be a major predictor of perioperative risk, even for procedures with relatively low procedural stress. Recent MI (less than 1 month), unstable angina, past MI with significant residual damage, decompensated congestive heart failure, significant arrhythmias, and severe valvular disease are all considered major predictors of increased preoperative cardiovascular risk and these patients would usually be classified as ASA IV. Patients with stable angina, past history of MI (greater than 1 month) with minimal residual myocardial damage, compensated congestive heart failure, or diabetes mellitus should be considered intermediate risk and would usually be classified ASA II or ASA III.[59] The presence of multiple risk factors creates an additive increase in overall perioperative risk.[61] Even patients with a history of recent MI or unstable angina should be able to tolerate routine dental procedures with local anesthesia although medical consultation is required and conscious sedation with monitoring is often recommended.[60,62]

Treatment modification considerations for patients with ischemic heart disease should include: morning appointments, short appointments, oral pre-medication with an anxiolytic drug and/or nitrous oxide/oxygen sedation, limited use of vasoconstrictors (as previously discussed),

adequate pain management (during and after the dental appointment), treatment in a semi-sitting position and possible cardiac monitoring.[63] Sedation with a short acting oral benzodiazepine (e.g., triazolam or ativan) and/or nitrous oxide can reduce the stress of a dental procedure and increase the effectiveness of local anesthesia.[33] Clinicians must be aware of the medical need and legal requirements that include special training, permits, and monitoring when anxiolysis crosses over into conscious sedation. If conscious sedation is required, it is best performed by a trained provider of anesthesia/sedation and with another operator providing the dental care.

Heart Murmurs and Valvular Disease

Patients with valvular disease present two primary considerations for dental treatment: potential risk for infective endocarditis and risk of excessive bleeding in patients on anticoagulant therapy.[55] Management considerations for patients on anticoagulant therapy are discussed later in this chapter. Most heart valve abnormalities affect either the aortic or mitral valve and may cause turbulent blood flow or partial obstruction of blood flow (stenosis) or valve incompetence (regurgitation). Heart murmurs are common and may be benign or signify major underlying disease such as degenerative valve disorders (e.g., aortic stenosis), rheumatic heart disease, congenital valve lesions, prosthetic valves, atrial fibrillation, or congestive heart failure.[64] Other conditions that place patients at increased risk for infective endocarditis are systemic lupus erythematosus (SLE) and certain medications used for weight reduction (dexfenfluramine and fenfluramine-phentermine).[64,65] Dental management requires evaluation of the type of heart condition, risk of valve infection, and the risk of bacteremia from the planned dental procedure.

Current guidelines for prevention of infective endocarditis were published in 2007 and represent a significant change from previous American Heart Association guidelines.[66] 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 potential benefits.[66]

Figures 31-2 and 31-3 list the specific heart valve abnormalities and dental procedures that are considered highest risk for infective endocarditis. In general, procedures associated with non-surgical root canal treatment such as local anesthetic injection, placement of the dental dam, and instrumentation when contained within the canal system do not

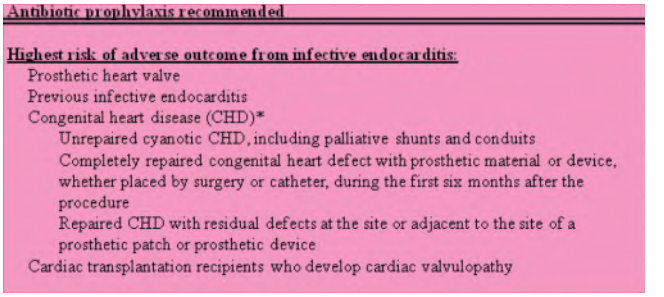

FIGURE 31-2 Recommendations for antibiotic prophylaxis based on risk stratification for infective endocarditis. (Adapted from: Wilson W, et al.[66])

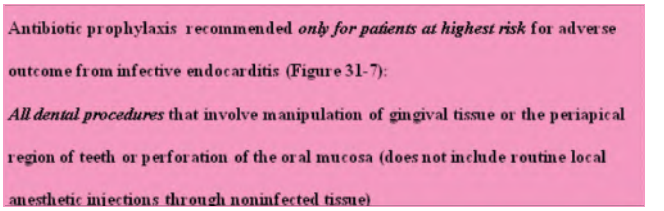

FIGURE 31-3 Risk of Dental Procedures. (Adapted from: Wilson W, et al.[66])

place the patient at significant risk for infective endocarditis.[67] The incidence and magnitude of bacteremia when canal instrumentation does not extend into the periapical tissues is very low, and almost all bacteria are eliminated from the blood within 10 minutes.[68,69] Canal instrumentation beyond the apex, intraligamentary and intraosseous injections, and periapical surgery can all be expected to result in a higher risk for transient bacteremia. In these situations, antibiotic pre-medication is recommended for patients in the highest risk disease categories. Infective endocarditis is only rarely directly linked to dental procedures and the efficacy of the recommended antibiotic regimen is questionable.[66,70–74] The current regimen and drugs of choice for antibiotic prophylaxis are presented in Table 31-6. These guidelines should be considered the current best evidence to guide clinical practice decisions; however, it is interesting to note that a recent study in England demonstrated an increase in cases of infective endocarditis following the widespread adoption of the new standards in 2008.[75] The increase in cases was found in both individuals at high risk and lower risk of infective endocarditis. Although the increase was statistically significant, the absolute number of cases was still very low and the authors caution that a causal relationship has not been established.

According to Lessard et al.,[64] some patients with significant heart murmurs may have dyspnea, fatigue, and difficulty breathing when reclined in the dental chair. Therefore the dental procedure may need to be performed with the chair in a more upright position.[64] These authors also suggest that use of a dental dam may be contraindicated for some of these patients due to restriction of air flow; however, this may be overcome with careful application of the dental dam, allowing the patient continuous

TABLE 31-6 Antibiotic Prophylaxis for Dental Procedures—All Regimens Are a Single Dose Given 30 to 60 Minutes Before Procedure

Standard oral regimen	**Adults: 20 g amoxicillin** **Children: 50 mg/kg**
Alternative oral regimen for patients allergic to penicillin or patients who are currently taking a penicillin class antibiotic	Adults: 2.0 g cephalexin or other 1st or 2nd generation cephalosporin in equivalent dosage* OR 600 mg clindamycin OR 500 mg azithromycin or clarithromycin Children: 50 mg/kg cephalexin or other 1st or 2nd generation cephalosporin in equivalent dosage* OR 20 mg/kg clindamycin OR 15 mg/kg azithromycin or clarithromycin
Patients unable to take oral medications	Adults: 2.0 g IM or IV ampicillin OR 1.0 g IM or IV cefazolin or ceftriaxone* Children: 50 mg/kg IM or IV ampicillin OR 50 mg/kg IM or IV cefazolin or ceftriaxone*
Alternative IM/IV regimen for patients allergic to penicillin and unable to take oral medications.	Adults: 1.0 g IM or IV cefazolin or ceftriaxone* OR Children: 50 mg/kg IM or IV cefazolin or ceftriaxone* OR 20 mg/kg IM or IV clindamycin within 30 minutes before the procedure.

*Cephalosporins should be used with caution in patients reporting an allergy to penicillin since approximately 5%–15% of patients who are allergic to penicillin will demonstrate cross reactivity with cephalosporins, especially first and second generation cephalosporins. (Adapted from: Wilson W, et al. [66])

air flow. Failure to use the dental dam is considered below the standard of care for root canal therapy and extraction may be the only option for these patients if a dental dam cannot be used.

Anticoagulant Therapy and Bleeding Disorders

Management of patients on anticoagulant therapy depends on the type of anticoagulant, reason for anticoagulant therapy, and type of planned procedure. Warfarin (Coumadin-DuPont Pharmaceuticals, Wilmington, DE, U.S.A.) anticoagulants are commonly prescribed for the treatment or prevention of thromboembolic events. This category of anticoagulant works by blocking 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 INR is usually between 2.0 and 3.5, depending on the underlying medical indication for anticoagulant therapy.

Limited oral surgery procedures, defined as simple forceps extraction of one to three teeth, may be safely performed on patients with INR values within the normal therapeutic range.[76–78] Non-surgical root canal treatment does not usually require modification of anticoagulant therapy although it is important to ascertain that the patient's INR is within the therapeutic range, especially if a nerve block injection is required. Periapical surgery 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 on anticoagulant therapy. Consultation with the patient's physician is required to assist in developing an appropriate treatment plan. Some patients may be able to tolerate discontinuation of warfarin therapy 2 days prior to a planned surgical procedure to allow for the INR to "drift" downward. In a prospective cohort study, Russo et al.[79] report that suspension of warfarin 2 days prior to a surgical procedure was associated with no bleeding problems and no thromboembolic events. They found that the average time spent at an INR less than 2.0 (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 and in these cases discontinuation of anticoagulant therapy would not be recommended.

In general, patients on warfarin anticoagulant therapy should present minimal risk for significant bleeding during or after oral surgery and local measures to control bleeding should be adequate.[80,81] Jeske and Suchko,[82] in a review prepared for the American Dental Association Council on Scientific Affairs and Division of Science, recommend against routine discontinuation of anticoagulant therapy prior to 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 is strongly recommended. 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. One possible alternative is the use of low-molecular-weight heparins (LMWHs), which allows for patient self-administration and may present a viable alternative for some patients who need to remain at a high level of anticoagulation but wish to reduce the cost and time associated with traditional heparin conversion therapy.

A new category of novel oral anticoagulant (NOAC) medications is gaining acceptance as an alternative to warfarin for most anticoagulant indications except prosthetic heart valves.[83] Examples of drugs in this category include: apixaban (Eliquis™), dabigratan (Pradaxa™), edoxaban (Lixiana™), and rivaroxaban (Xarelto™). Compared to warfarin, NOACs have fewer interactions with foods and other drugs, and there is no need for routine blood chemistry monitoring. An example of this drug category, rivaroxaban prolongs PT by inhibiting factor X to Xa conversion and has a half-life of 8 to 12 hours. Unfortunately, the PT test is not a reliable measure of the level of anticoagulation and risk for bleeding during surgical procedures. Although it appears likely that patients who require minor dental procedures such as extraction or root canal treatment will not require discontinuation of NOAC therapy, evidence to support this decision is currently not available. Therefore, medical consultation is advised and the patient's physician may recommend discontinuing the NOAC drug for 1 to 2 days prior to a surgical procedure.[84]

Low dose aspirin therapy is known to increase bleeding time by irreversibly inhibiting platelet aggregation. No treatment modifications should be necessary for non-surgical root canal procedures. However, surgical procedures require evaluation of the reason for and necessity of aspirin therapy. It has been common practice to advise patients to discontinue aspirin therapy for 7 to 10 days prior to an oral surgical procedure.[85] At low dose therapeutic levels (<100 mg/day), aspirin may increase bleeding time and potentially complicate surgical procedures. However, Ardekian et al.[85] concluded that low dose aspirin therapy (<100 mg/day) should ***not*** be discontinued prior to oral surgery procedures and that bleeding could be controlled by local measures. Higher dose therapy may present a greater risk for bleeding either during or after surgery. Even though a patient on aspirin therapy may not be at high risk for significant intra or postoperative bleeding, a concern for periapical surgery is the visibility problems created by oozing blood. Consultation with the patient's physician or experienced dental specialist is advised to determine the medical reason for aspirin therapy and weigh the risks and benefits of discontinuing aspirin prior to the proposed surgery. It should be possible to perform periapical surgery without discontinuing aspirin therapy if necessary, but visibility during the procedure may be compromised and the prognosis may decrease accordingly.[47]

Nonsteroidal anti-inflammatory drugs (NSAIDs) also have an antiplatelet effect but, unlike aspirin, the effect is reversible when discontinued and platelet activity should be expected to return to normal within approximately three half-lives of the drug. Other commonly prescribed antiplatelet drugs include dipyridamole, ticlopidine, clopidogrel, abciximab, integrelin, tyrafiban, lamifiban. Heavy alcohol consumption, liver disease, and certain medications can increase the risk of perioperative bleeding in patients taking antiplatelet medications. Medical consultation is advised prior to surgical procedures and lab tests to determine platelet count and function (PFA-100 and Ivy BT) may be indicated. Some herbs and dietary supplements may also affect bleeding risk.

COX-2 selective inhibitors are known to increase the risk of venous thromboembolism and major coronary events.[86,87] Even ibuprofen, which is primarily a COX-1 inhibitor, probably the most commonly prescribed NSAID for dental pain, appears to increase the risk of major coronary events, but not major vascular events.[87]

Patients with inherited or acquired bleeding disorders are also at risk for excessive bleeding during and after periapical surgery procedures and may be at risk from local anesthetic injections particularly when using nerve block injections. Impaired liver function secondary to past or current alcohol or drug abuse may also predispose a patient to excessive bleeding during surgery. A medical consultation, usually with a hematologist, is required prior to dental treatment for patients with serious bleeding disorders such as thrombocytopenia, hemophilia, and von Willebrand's disease. Replacement of deficient coagulation factors or platelet transfusion may be required prior to dental treatment,[88] particularly if an inferior alveolar nerve block is required or surgical treatment is planned. If non-surgical root canal treatment can be performed with only infiltration local anesthesia, replacement may not be necessary.[89] However, this decision must be reached in consultation with the patient's hematologist.

Arrhythmias and Cardiac Pacemakers

Cardiac arrhythmias are a heterogeneous group of conditions defined as any disturbance in the normal rate or rhythm of the heartbeat. Arrhythmias are the result of abnormal impulse generation, impulse conduction, or both and can range from harmless to life threatening.[55] The overall prevalence of cardiac arrhythmias in the general dental patient population is about 17 % and more than 4% of those represented

serious, potentially life threatening arrhythmias.[90] Although arrhythmias are not uncommon in normal, healthy adults, the possibility of underlying cardiovascular disease, systemic disease, or medication-induced arrhythmia should be carefully evaluated prior to dental treatment. Anxiety associated with dental treatment may induce arrhythmias in susceptible patients. In addition, patients with cardiovascular disease are more prone to arrhythmias during oral surgery procedures with local anesthesia.[91] Patients taking digoxin for atrial fibrillation or congestive heart failure are especially at risk for arrhythmias during oral surgery procedures.[92]

Medications are usually the first line of treatment for cardiac arrhythmias although many of these have a narrow therapeutic safety range (e.g., digoxin) and must be carefully monitored. Surgery, cardioversion, and pacemakers are also used to treat arrhythmias. It is common for atrial fibrillation patients to be treated with an anticoagulant (typically warfarin or a NOAC drug- management discussed previously in this chapter). A history of cardiac arrhythmia is often disclosed in the medical history. In addition to the medical history, patients with an irregular pulse, unusually rapid or slow pulse, reports of syncope, palpitations, dizziness, angina, or dyspnea, should be referred to a physician for evaluation prior to dental treatment. Once the nature of the arrhythmia and stability of the condition has been determined, most dental treatment can be safely performed using the same stress reduction treatment modifications listed in the discussion of ischemic heart disease. As always, the clinician and staff should be prepared to manage a medical emergency if necessary.

Electrical interference from certain dental devices is a potential concern for patients with implanted cardiac pacemakers or cardioverter/defibrillators. In particular, electronic apex locators (EAL) and electric pulp testers (EPT) are commonly used in root canal therapy. Manufacturers of EAL and EPT devices warn against the use of these devices in patients with cardiac pacemakers. However, current cardiac pacemakers are well shielded from external electrical fields and the possibility for electrical interference seems to be very low.[93–97] Recent clinical studies support previous *in vitro* research and a case report in concluding that EAL and EPT devices should be safe to use in patients with cardiac pacemakers and cardioverter/defibrillators.[98,99] One caveat is that a mucosal lip clip was used in one study to complete the circuit with the EPT device instead of the common clinical practice of having the patient hold the EPT wand in his or her hand.[98] This lip clip technique is recommended if one elects to use an EPT device on a patient with a cardiac pacemaker since the practice of using the patient's hand to complete the circuit may allow for electrical current to pass through an area of the body in closer proximity to the pacemaker. The safety of this variation of the EPT technique has not yet been tested.

Heart Failure

Heart failure (HF), also known as congestive heart failure (CHF), is an increasingly common diagnosis in an aging population and represents the end-stage of other common cardiovascular diseases such as coronary artery disease, hypertension, cardiomyopathy, valvular heart disease, and MI.[100] HF results in an inability of the heart to efficiently pump blood that can involve one or both ventricles. Patients with HF typically present with significantly diminished reserve capacity for handling stress (including dental treatment) and are often taking multiple medications with the potential for drug interactions.

Patients with well-managed HF should tolerate routine dental treatment with possible minor treatment modifications, similar to those recommended for patients with ischemic heart disease. In addition, the underlying causes (coronary artery disease, hypertension, valve disease, etc.) and medications should be considered and managed appropriately. Patients with moderate to advanced HF may require a more upright chair position due to the presence of pulmonary edema. In these settings, poorly controlled HF may present with shortness of breath and peripheral edema. The clinician should be alert for orthostatic hypotension when making adjustments in chair position. Uncompensated, advanced HF requires medical consultation prior to dental treatment and vasoconstrictors should be avoided. These patients may require treatment in special care facilities or hospital based clinics. The New York Heart Association (NYHA) has developed a classification system for HF that can be adapted to assist in assessing risk for dental treatment (Table 31-7).

TABLE 31-7 New York Heart Association Classification System for Patients with Congestive Heart Failure and Dental Management Considerations

Heart Association CHF classification	Signs and symptoms	Dental management considerations
Class I	No limitations or physical activity; no dyspnea, fatigue, or palpitations with ordinary physical activity	Should be able to tolerate routine dental treatment; stress reduction protocol as needed
Class II	Slight limitation on physical activity; comfortable at rest but may experience fatigue, palpitations, and dyspnea with ordinary physical activity	Should be able to tolerate routine dental treatment; *stress* reduction protocol as needed; possible medical consultation
Class III	Significant limitation of activity; comfortable at rest but minor activity results in symptoms	Medical consultation; consider treatment in hospital dental clinic or similar facility; avoid vasoconstrictors
Class IV	Symptoms present at rest; symptoms exacerbated by any physical activity	Medical consultation; conservative treatment only; treatment in hospital dental clinic; avoid vasoconstrictors

(Adapted from: Little JW, et al. [32])

Diabetes

Diabetes mellitus (DM) is a complex metabolic disorder characterized by abnormalities in carbohydrate, fat, and protein metabolism resulting either from a deficiency of insulin (type 1) or from target tissue resistance to its cellular metabolic effects (type 2). Hyperglycemia is the most clinically important metabolic aberration in DM and the basis for its diagnosis. Chronic hyperglycemia is associated with ophthalmic, renal, cardiovascular, cerebrovascular, and peripheral neurological complications. DM is defined as a fasting blood glucose level greater than 125 mg/dl and normal fasting blood glucose level is considered to be less than 110 mg/dl. Patients with fasting plasma glucose levels greater than 110 mg/dl but less than 126 mg/dl represent a transitional condition between normal and DM and are considered to have impaired glucose tolerance.[101] Identification of patients at this stage can allow for earlier preventive interventions and possibly delay or prevent progression to DM.

Gylcated hemoglobin (HbA1c) is considered to be a more accurate test for diabetes since it measures the average level of blood glucose over the previous 3 months. Normal HbA1c is less than 5.7%; pre-diabetes is 5.7 to 6.4%; and diabetes is defined as 6.5% or greater. The HbA1c goal for most diabetic patients is less than 7%.

Approximately 26 million children and adults in the United States, or 8.3% of the population, have DM and over one-third are unaware that they have the disease.[102] The epidemic of obesity in the United States is anticipated to result in an increase in the prevalence of diabetes.

When reviewing medical histories, the clinician should be aware of cardinal symptoms of DM, such as polydipsia, polyuria, polyphagia, weight loss, and weakness, and should be referred to a physician for diagnosis and treatment.[102] In diabetic patients, the clinician should ascertain how well-controlled the condition may be. Dental appointment scheduling should take into account the importance of nutritional consistency and the avoidance of appointments that will overlap with or prevent scheduled meals.

Symptoms of hypoglycemia may range from mild, such as anxiety, sweating, tachycardia, to severe, such as mental status changes, seizure, and coma. Severe hypoglycemic episodes are a medical emergency and should promptly be treated with 15 g of oral carbohydrate, such as 6 oz orange juice, three to four teaspoons of table sugar, five Life Savers, or three glucose or dextrose tablets. If a patient is unable to cooperate or swallow, 1 mg glucagon may be administered by subcutaneous or intramuscular injection. Side effects of glucagon include nausea, vomiting, and headache.

It has been well-established that hyposalivation, gingivitis, periodontitis, and periodontal bone loss are associated with DM, especially when poorly controlled.[103,104] The well-controlled diabetic may not be at increased risk of postoperative infection than the nondiabetic.[105] Therefore, surgical procedures in well-controlled diabetics do not require prophylactic antibiotics. However, when surgery is necessary in the poorly controlled diabetic, prophylactic antibiotics should be considered due to altered function of neutrophils in diabetics. Surgery may also increase insulin resistance such that a diabetic may become hyperglycemic in the postoperative period. Pre-operative antibiotics should be considered in these instances.[106] Furthermore, delayed alveolar healing following dentoalveolar surgery should raise the suspicion of osteomyelitis, for which prompt surgical consultation should be arranged. Oral complications may include dry mouth, and increased risk of candidiasis, particularly if an antibiotic is prescribed. The potential of diabetic neuropathy to cause oral symptoms should be considered in the diagnosis of orofacial and dental pain. Finally, patients with DM may present with systemic complications, each of which should be taken into account prior to dental procedures.

Pulmonary Disorders

Asthma

Asthma is a chronic inflammatory respiratory disease with recurrent episodes of chest tightness, coughing, dyspnea, and wheezing resulting from hyper-responsiveness and inflammation of bronchiole tissue. Overt attacks may be provoked by allergens, upper respiratory tract infections, genetic and environmental factors, certain medications, and highly emotional states such as anxiety, stress, and nervousness.

The endodontist should obtain a good history to determine the severity and stability of disease. Patients should be instructed to bring their inhalers (bronchodilators) to each appointment and inform the endodontist at the earliest sign or symptom of an asthma attack. During dental treatment, the most likely times for an acute exacerbation of asthma are during and immediately after local anesthetic administration and with stimulating procedures such as pulp extirpation.[107] Because stress is implicated as a precipitating factor in asthma attacks sedation may be beneficial. While nitrous oxide may be used in patients with mild-to-moderate asthma, its use is contraindicated in patients with severe asthma due to its potential to cause airway irritation.[108] Alternatively, oral premedication may be accomplished with small doses of a short-acting benzodiazepine. In patients taking theophylline, macrolide antibiotics should be avoided, as they have the potential to develop toxic levels of theophylline. In addition, it is important to note that aspirin and other NSAIDs may trigger asthma attacks in a proportion of patients.[109]

Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease (COPD) is a term for pulmonary disorders characterized by chronic irreversible obstruction of airflow from the lungs and represents the fourth most common cause of death in the United States.[110] The three most common forms of COPD are chronic bronchitis, emphysema, and bronchial asthma. Patients with pulmonary diseases typically present with one or more of the

following symptoms: cough, dyspnea, sputum, hemoptysis, wheezing, or chest pain.[111] Patients should be placed in a semi-supine position. Since use of a dental dam may induce a feeling of airway constriction, careful application of the dental dam and administration of humidified low-flow oxygen, generally between two and three liters per minute, may be considered. Nitrous oxide should not be used in patients with severe COPD.

Tuberculosis

Tuberculosis (TB) is an infectious disease that is spread by way of bacilli-containing airborne droplets, typically by coughing, sneezing, or talking. The signature lesion of TB is the tubercle, a granuloma formed by the continuing ingress of macrophages and lymphocytes to the site of the infection.[112] In the lung, tuberculous granulomas are frequently associated with regions of tissue necrosis, termed *caseous necrosis* due to its gross appearance. After the infection is established, symptomatic individuals will show pulmonary manifestations of the disease, often limited to the periphery of the middle and lower regions of the lung,[112] though reactivated disease is most commonly found in the lung apices.[113] Oral tubercular infections are rare, occurring in 0.05% to 5% of patients with TB, though when lesions are present, they typically consist of ulcers, fissures, or swellings on the dorsum of the tongue.[114] Despite the declining incidence of TB in the United States, health care workers including endodontists and their staff remain at elevated risk for contracting the disease.

It is imperative for the endodontist to educate office staff about TB prevention and recognition of symptoms and oral manifestations of the disease to protect the staff and other patients from becoming infected. A thorough medical history should be obtained, and any dental procedures on a patient with established or suspected active TB should be delayed until the individual can be treated and subsequently proved noninfectious.

Routine dental treatment is appropriate after it has been established that the patient has been adequately treated and there are no signs or symptoms of active disease. The clinician should be aware of potential drug interactions when managing dental patients undergoing antitubercular treatment. In patients taking medications such as rifampin and isoniazid, acetominophen should be avoided due to the potential for liver damage. The use of aspirin is discouraged in those individuals taking streptomycin due to a heightened risk of ototoxicity and vestibular disturbances.[115] Streptomycin is also known to cause facial paresthesia and pancytopenia.

Cerebrovascular Accident

Stroke

A cerebrovascular accident (CVA), or stroke, is subclassified into ischemic insults, occurring secondary to thrombosis or embolization, or hemorrhagic that usually indicate an arterial process. The lack of blood flow leads to deprivation of oxygen and glucose in a localized area of the brain. The endodontist should be aware of how to identify a patient having a stroke in his/her office. A useful mnemonic for early identification of a possible stroke is: F.A.S.T.:

F = Face: Ask the patient to smile. Does one side of the face droop?
A = Arms: Ask the patient to lift both arms. Does one arm drift downward?
S = Speech: Ask the patient to repeat a simple phrase. Is their speech slurred or strange?
T = Time: If you observe any of these signs, call your emergency operator (in the U.S.A. it is 911) (www.stroke.org).

Regardless of the procedure, a patient's blood pressure should be checked before treatment to identify a patient whose blood pressure is elevated and who might be at risk for a stroke if subjected to stress. Slurred speech, loss of motor control over a portion of the body, unilateral facial droop, unilateral visual changes, and unilateral severe headache are all potential signs of a stroke or transient ischemic attack (TIA). Should any of these events occur, the patient should have his/her vital signs checked, be placed in a supine position, have vital signs monitored, and be transported to an emergency facility immediately, as treatment must be activated in a timely manner. Patients with a history of stroke may be at risk for aspiration due to swallowing abnormalities, so they should be positioned in a semi-supine position, and dental dams should be carefully applied and should always be used. Post-stroke patients may be on oral anticoagulants, so if surgical intervention is planned, the endodontist should contact the physician to determine whether or not the risk of a thromboembolic event outweighs the benefits of postoperative hemostasis (please refer to subsection on *anticoagulant therapy and bleeding disorders*). The endodontist should also be aware that the post-stroke patient may experience emotional problems, including anger and depression and behavior inappropriate to the situation.

Seizures Disorders

Seizures are one of the most commonly encountered neurological disorders and can manifest as an isolated incident with unknown etiology or as a symptom of a condition that requires long-term treatment. A seizure is a temporary involuntary disturbance of brain function that results in synchronous, excessive, abnormal electrical discharges of the neurons in the central nervous system.[116] This manifestation can take the form of motor disturbances, altered feelings, or changes in the patient's level of consciousness. The two main categories of seizure classification are partial and generalized. Most people who have seizures have good control and are capable of receiving routine dental care.

The endodontist should be aware of the patient's seizure medications, since many antibiotics are contraindicated. Should a seizure occur in the dental office, the procedure should be stopped immediately and all instruments should be removed from the oral cavity. The patient should be placed

in a supine position and low to the ground. Basic life support should begin immediately, including opening the airway, obtaining vital signs such as heart rate and blood pressure, and contacting emergency medical services.

The risk of shunt infection following invasive dental treatment for patients with hydrocephalic shunts (ventriculoperitoneal and ventriculoatrial) is believed to be very low,[117] and because of this there is a lack of consensus regarding the need for antibiotic prophylaxis. An expert panel of the American Heart Association stated that antibiotic prophylaxis prior to dental treatment in patients with nonvalvular cardiovascular devices (including hydrocephalic shunts) was generally not recommended.[118] However, antibiotic prophylaxis was recommended if treatment involved incision and drainage of an abscess. One study found that pediatric dentists were more likely to be concerned about streptococcal microorganisms and neurosurgeons were more concerned about staphylococcal microorganisms in shunt infection.[119] The majority of both groups recommended penicillin prophylaxis although there are more appropriate antibiotics since staphylococcal microorganisms are responsible for most shunt infections.[118,119] (Refer to the discussion in the *Prosthetic joints and other prosthetic devices* section of this chapter.) Consultation with the patient's neurosurgeon is advised and close attention to any changes in prophylaxis guidelines is important.

Renal Disease and Dialysis

Chronic renal failure is a slowly progressive condition characterized by irreversible reduction in glomerular filtration rate (GFR). The progression of this disease begins with an asymptomatic decrease in kidney function and eventually results in end-stage renal disease (ESRD). Throughout the decline in function, multiple systems are affected, directly related to the kidney dysfunction. ESRD is potentially fatal unless the patient undergoes dialysis or kidney transplantation (please see the section: *solid organ transplantation* regarding kidney transplantation). Dialysis may take the form of hemodialysis that represents 90% of dialysis treatment,[120] or peritoneal dialysis. This treatment removes fluid and wastes and equilibrates electrolytes and acid-bases via diffusion and osmosis across a semi-permeable membrane.

The clinician must be aware of the ESRD patient's type and days of dialysis treatment as well as co-morbid conditions such as hypertension and/or diabetes. Mechanical trauma to platelets and anticoagulants such as heparin used during hemodialysis may increase the renal patient's tendency for bleeding. While it is recommended that dental procedures be performed on non-dialysis days, typically the day after dialysis,[121] the endodontist should be aware that abnormal platelet function may cause a greater risk of bleeding during surgical procedures. In addition, patients with ESRD require aggressive treatment of odontogenic infections. Antibiotic premedication has been recommended by some authors for hemodialysis patients with shunts who undergo invasive dental procedures,[122–124] and other authors have recommended antibiotic prophylaxis for all hemodialysis patients undergoing procedures that cause mucosal bleeding to prevent vascular access infections, bacteremia, and infective endocarditis.[123] However, an expert panel scientific statement from the American Heart Association does *not* recommend routine antibiotic prophylaxis for this group of patients prior to dental treatment, except in cases that involve incision and drainage of an abscess.[118] The majority of vascular grafts infections are associated with *Staph aureus*.[118]

Renal osteodystrophy and secondary hyperparathyroidism may occur in late-stage disease due to disorders in calcium, phosphorous, and abnormal vitamin D metabolism. Such a manifestation may increase a risk of jaw fracture during surgical procedures.

Because many drugs are metabolized via the kidney, renal dosing should account for the drug's extended half-life by lengthening the interval between medication doses and/or altering the dose of required medications. In particular, antibiotic medications should be adjusted for renal dosing. Nonsteroidal anti-inflammatory drugs should be avoided in patients with renal insufficiency due to their nephrotoxic effects, but no longer need to be avoided when the patient has ESRD.

Oral Cancer

Oropharyngeal cancer encompasses a variety of malignant diseases. More than 90% of oral cancers are squamous cell cancers, 9% are salivary gland tumors, sarcomas and lymphomas, and the remaining 1% are metastatic cancers originating in other parts of the body.[125] In the year 2015, the American Cancer Society projected 39,500 new cases of oral and oropharyngeal cancers and 7800 deaths from this disease.[126] Numerous risk factors have been implicated in the etiology of oropharyngeal cancer, including tobacco, excessive alcohol, ultraviolet light exposure, immunosuppression and viruses, specifically human papilloma virus (type 16).

Oral cancer has a variable appearance, including white or red patches, an exophytic mass, an ulceration, a granular raised lesion, a submucosal mass, or a combination thereof. Treatment of oropharyngeal cancer is composed of surgical intervention, radiation treatment, and chemotherapy, targeted therapy and immunotherapy is being investigated.[127] Multimodality therapy is now more commonly used for oropharyngeal cancer in order to obtain increased survival rates.

Cancers that are amenable to surgery, and that do not affect the oral cavity, require few treatment plan modifications. On the other hand, oropharyngeal cancer treatments and complications may cause significant changes in the oral cavity. Preceding cancer treatment, all sources of inflammation and potential infection must be eliminated. Whenever possible, nonrestorable teeth and teeth with a poor long-term periodontal prognosis (i.e., not expected to be retained for the patient's lifetime) within the field of high dose radiation, should be extracted preferably more than 2 weeks prior to radiation therapy. Symptomatic nonvital teeth can be endodontically treated at least one week before initiation of head and neck radiation or chemotherapy. Dental treatment

of asymptomatic teeth, even with periapical involvement, can be delayed, particularly if treatment can be limited to intracanal therapy. Within a high dose radiation field, teeth may be considered for extraction or definitive management. Many cancer patients have indwelling catheters that may be susceptible to infection. While controversial, the CDC does not recommend routine antibiotic prophylaxis before invasive dental procedures.[128] If an individual is receiving chemotherapy, the endodontist should be familiar with the patient's white blood count (WBC) and platelet status and may consult the physician and/or experienced oral care specialists. Endodontic procedures may be performed if the neutrophil count is greater than 1,000 cells/mm3 and platelets are greater than 50,000 cells/mm^3. Post-radiation osteonecrosis (PRON) results from radiation-induced changes in the jaws, may arise in bone exposed to high dose radiation, and is characterized by asymptomatic or painful bone exposure. Protocols to reduce the risk of osteonecrosis include endodontic therapy over extractions. Expert atraumatic surgical procedures, under local anesthetics that contain no or low concentration of epinephrine, plus prophylactic antibiotics and antibiotics during the week of healing are in order.[129]

Antiresorptive Agent-Induced Osteonecrosis of the Jaw (ARONJ)

Bisphosphonates are bone resorption inhibiting medications that are commonly used in cancer therapy and may be used in conjunction with cancer chemotherapy (e.g., multiple myeloma and metastatic bone, breast and prostate cancer) and management of hyperkalemia in cancer. They may also be used in the prevention and treatment of osteopenia, osteoporosis, and Paget's disease of the bone. Recent reports have suggested that bisphosphonates (e.g., pamidronate, zoledronic acid) increase the risk of osteonecrosis of the maxillary and mandibular bones, either spontaneously or following dental surgical procedures or oral trauma.[130–134]

The new name (ARONJ) for the condition previously known as bisphosphonate associated osteonecrosis (BON) reflects current knowledge that some non-bisphosphonate antiresportive medications also increase the risk of osteonecrotis of the jaw (e.g., denosumab).[135] Although the mechanism of action is not completely understood, it has been suggested that inhibition of osteoclastic bone resorption and remodeling, inhibition of angiogenesis, and the presence of infection and/or inflammation could contribute to the development of ARONJ.[132,136] There appears to be a dose-response relationship in that patients taking IV formulations appear to be at a greater risk for ARONJ.[137] The highest estimated risk for developing ARONJ in a patient who is taking an antiresorptive drug for a non-cancer related indication is about 0.10%.[135,136] The risk for developing ARONJ following an invasive dental procedure such as extraction is estimated to be about 0.5%.[136,138] The risk of ARONJ is significantly higher for patients with a history of IV bisphosphonate use and cancer. Although no data exists to objectively assess the relative risk of various invasive dental procedures, it is reasonable to assume that any procedure that requires exposure and manipulation of bone (e.g., implant placement, endodontic and periodontal surgery) has an increased risk of necrosis, although more extensive procedures are expected to have higher risk.[133,136] Unfortunately, previous suggestions that serum levels of C-terminal cross-linking telopeptide of Type I collagen (CTX) might be a useful predictor of which patients would be at higher risk for ARONJ has not been confirmed or supported by recent research.[135,136]

There are currently no scientific data to support any specific treatment protocol for the management of patients at risk for ARONJ, though minimally invasive procedures have been recommended.[133] Discontinuation of bisphosphonate therapy for several months before and after invasive dental treatment may be reasonable, but research to support this approach is currently absent and the agent has a multi-year presence in bone once administered; however the potential of reversible effects of denosumab in bone is possible and is being investigated. Before initiation of antiresporptive agents, aggressive preventive treatment should be performed including oral hygiene, caries control, and extraction of teeth with a poor long-term prognosis. For patients who have been taking bisphosphonates or denosumab, preventive care for high-risk patients is important to reduce the risk of developing ARONJ.

Nonsurgical endodontic treatment of teeth that would otherwise be extracted should be considered. Teeth with extensive carious lesions might be treated by nonsurgical endodontic therapy possibly followed by crown resection and restoration similar to preparing an overdenture abutment.[139] Another possible approach for management of non-restorable endodontically involved teeth is slow (four to six weeks) orthodontic extrusion of the root(s) (Figure 31-4 A–E). For patients at higher risk of developing ARONJ, surgical procedures including surgical endodontic procedures should be avoided if possible. Teriparatide, an osteoanabolic drug approved by the FDA for management of osteoporosis, has been suggested as a possible treatment for ARONJ, but this is only at the case report level of evidence and needs to be validated with larger scale controlled studies.[140] A case series has suggested the use of pentoxifylline and vitamin E for treatment of necrosis.[141] Informed consent for endodontic procedures should involve a discussion of risks, benefits, and alternative treatments with the patient.

Bone Marrow and Solid Organ Transplantation

Hematopoietic Stem Cell Transplantation

Hematopoietic stem cell transplantation (HSCT) may be indicated in patients with hematologic malignancy, nonhematologic malignancy, and some nonmalignant disorders. Patients may undergo an autologous (self) or allogeneic (non-self) transplantation, each of which has its own pros and cons. The goal is to treat bone marrow disease or to intensify therapy that would destroy bone marrow, following which the patient

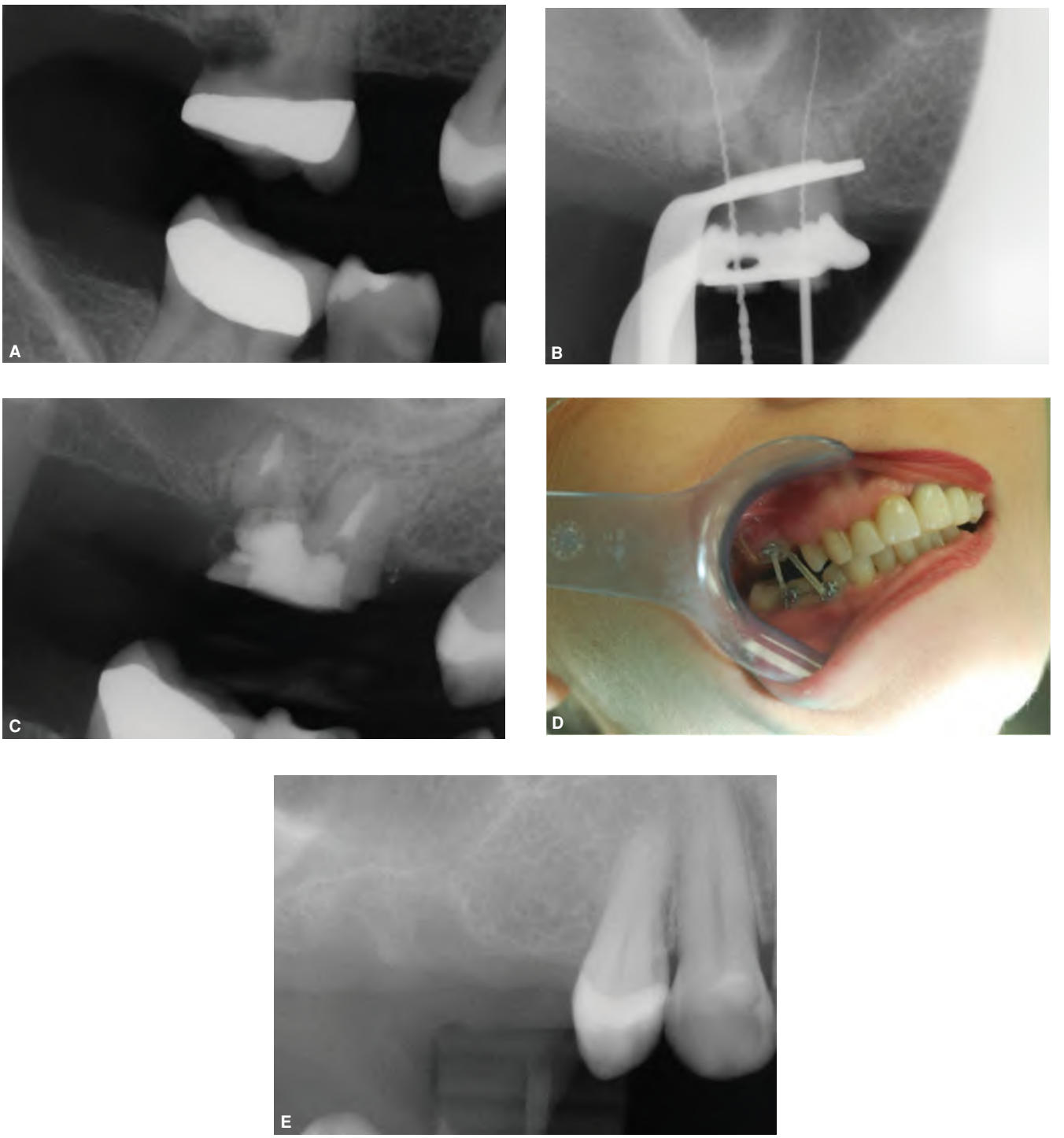

FIGURE 31-4 Orthodontic extrusion (extraction) of a symptomatic, non-restorable tooth in a patient with recent history of IV bisphosphonate use ***A.*** Pre-operative bitewing radiograph of tooth #3. ***B.*** Working length radiograph. ***C.*** Immediate postoperative radiograph. ***D.*** Three months after atraumatic extrusion. The tooth has been atraumatically extracted. (Case courtesy of Dr. Robert Lee.)

is "rescued" by infusion of previously stored autologous hematopoietic stem cells or related or unrelated hematopoietic stem cells from a matched donor. Prior to transplantation, patients should undergo a thorough dental examination and treatment to permit adequate healing before the HSCT.[127] Pretreatment endodontic therapy should be completed at least 10 days prior to initiation of cancer therapy. Teeth with poor prognoses should be extracted, utilizing the 10-day window as a guide. Prophylactic antibiotics are also recommended in patients who are neutropenic (< 1,000 neutrophils/mm^3).

During and following high-dose chemotherapy/HSCT, aggressive oral hygiene measures should be instituted.

Numerous oral complications may develop, including mucositis, graft-versus-host-disease, infection, taste changes, and bleeding. Patients should not resume routine dental treatment, including dental scaling and polishing, until adequate immunologic reconstitution has taken place; this typically occurs no less than 1 year post-transplant. The aerosolization of debris and bacteria during the use of high-speed rotary cutting instruments can put the patient at risk for aspiration pneumonia; additionally, bacteremias occur as a result of dental treatment and can cause serious outcomes. Should treatment be deemed necessary within 1 year post-transplant, the endodontist must consult with the oncologist and/or expert/experienced dental provider to determine appropriate treatment.

Solid Organ Transplantation

It is important to reduce the risk of infection in the immunosuppressed recipient of a transplant.[142] Pre-transplant, patients should undergo eradication of dental disease, including endodontic procedures as warranted to remove any potential sources of infection, and deferral of any elective treatments. Of course, the endodontist should take into account the underlying condition for which the transplant is required. In the immediate post-transplant period, emergency dental procedures may be necessary. At this stage, patients are highly immunosuppressed to prevent organ rejection, so the AHA regimen of antibiotic prophylaxis with possible post-operative antibiotics may be recommended for invasive procedures.

Should a patient experience transplant rejection, dental care should be limited to emergency care only until stabilization is again achieved. After the post-transplantation patient has stabilized, indicated dental procedures may be performed after consultation with the patient's transplant team. Postoperative guidelines regarding prophylactic antibiotics have not been established but, if recommended, AHA guidelines may be used.[142] Finally, the endodontist should be aware that post-transplant recipients will likely be on immunosuppressant therapy and should determine the dose and potential impact upon the planned dental care.

Prosthetic Joints and Other Prosthetic Devices

The long-standing practice of recommending antibiotic prophylaxis prior to dental treatment for patients with prosthetic joints has been questioned and based upon high-quality evidence-based reviews is no longer recommended.[143,144] The current best evidence supports the following recommendation: "In general, for patients with prosthetic joint implants, prophylactic antibiotics are not recommended prior to dental procedures to prevent prosthetic joint infection."[144]

Several risk factors for developing prosthetic joint infection were identified and were also determined to be independent of dental treatment. The risk factors included: postoperative wound drainage, hematoma, or urinary tract infection, and previous surgery on the index joint, diabetes, or immunocompromised status. The potential risks of routine antibiotic prophylaxis, including risk of anaphylaxis, development of antibiotic resistance, and opportunistic infections, were judged to be greater than the potential benefits. As always, clinical judgment and informed patient preferences should be considered as part of the clinical decision making process. If consultation with the patient's orthopedic surgeon is warranted, discussion should be based upon up-to-date evidence-based guidelines. Antibiotic prophylaxis is not indicated for dental patients with pins, plates, screws, and penile or breast implants. A patient with a total joint replacement or other surgically placed prosthesis with acute orofacial infection should be aggressively treated as any other patient to eliminate the source of infection, and appropriate antibiotics should be administered as warranted for the dental infection but this is not related to risk of prosthesis infection.

Pregnancy

Few procedures are contraindicated during pregnancy, however medication use should be carefully considered. There is no contraindication to using necessary diagnostic procedures, such as appropriate radiographs, as long as normal safety precautions are followed. If dental caries is the source of pain or infection, invasive care such as endodontic therapy should be provided regardless of the patient's phase of pregnancy.[145] Elective dental procedures may, often, be performed in the second trimester, when the pregnancy is mostly devoted to maturation. While some medications may be harmful to the fetus, safe alternatives are often available.

Lidocaine and prilocaine local anesthetics have an FDA category B rating and consequently should be first-line choices for use with pregnant women. A common misconception is concern over the use of local anesthetics containing epinephrine. Local anesthetics containing epinephrine should be relatively safe for use during pregnancy[146] and allow for greater depth and duration of anesthesia as well as reduction of any potential systemic effect of lidocaine. In addition, the only commonly available alternative to local anesthetics with a vasoconstrictor is 3% mepivicaine, an FDA category C drug. If antibiotics are warranted, many commonly used first line choices are rated by the FDA as category B for pregnancy risk (e.g., amoxicillin, azithromycin, cephalexin, clindamycin, and penicillin).[146] Pregnant women may be more susceptible to infection[147] that may be partly due to physiologic changes as well as alterations in pharmacokinetics.

Human Immunodeficiency Virus (HIV)

HIV is a blood-borne retrovirus infection transmitted primarily by blood and bodily fluids by intimate sexual contact and parenteral means. Upon infection, a viral enzyme reverse transcriptase allows the virus to integrate viral DNA into the genome of an infected cell and replicate using the infected cell's ribosomes and protein synthesis. Initially, immune seroconversion with antiviral antibody

production occurs followed by a significant decrease in CD4+ lymphocytes.

The most effective management in the progression of HIV infection and AIDS is a combination of antiviral agents known as highly active antiretroviral therapies (HAART) that has significantly increased the lifespan and quality of life of individuals infected with HIV. Upon initial assessment of an HIV-infected individual, the patient's physician should be contacted to determine CD4+ counts and viral load, as well as baseline kidney and liver function. It is safe and desirable to assure that comprehensive dental care is available to HIV-positive patients. No modification of irreversible procedures or surgical treatment is recommended unless patients have reduced platelet count (<50,000 cells/ml) or neutrophil counts <1,000 cells/ml, at which time a patient may require antibiotic prophylaxis. Routine antibiotic use is contraindicated.

The prognosis for successful healing of necrotic teeth with chronic apical periodontitis following root canal treatment is essentially the same for HIV-positive patients as non-infected patients.[148,149] The endodontist should examine oral tissues, as oral conditions associated with HIV may identify a person who is unknowingly infected with HIV, may be used in staging and classification, and/or may denote progression to AIDS. Surgically treated teeth do not show delayed healing. Antibiotics are only used if warranted by the clinical infection, and in a neutropenic patient.

Sickle Cell Anemia

Sickle cell anemia (SCA) affects approximately 1 in 400 African-Americans and as much as 30% of the population of some Central and West African countries. Clinical concerns in endodontic practice include the propensity for painful vaso-occlusive episodes and bacterial infections.[150] Since patients with SCA may be considered immunocompromised and infections can trigger a sickle cell crisis, these patients usually require aggressive treatment of infections, including the use of systemic antibiotics.[150]

The vaso-occlusive aspects of the disease can result in tissue and bone necrosis and pulpal necrosis in an otherwise intact and healthy tooth.[151] A recent clinical study found that pulpal necrosis in clinically intact permanent teeth was 8.33 times higher in patients with SCA compared to non-SCA controls.[152] Since teeth with asymptomatic necrotic pulps can become infected, patients with SCA require careful pulpal evaluation. Nonsurgical root canal treatment of asymptomatic necrotic teeth prior to the development of acute symptoms and infection is indicated.

There is a lack of consensus regarding the value of prophylactic antibiotics for patients with SCA although the majority of pediatric dentistry program directors and pediatric hematologists recommend antibiotic coverage for invasive procedures such as extraction or other surgical procedures.[153]

Use of a local anesthetic with no or minimal vasoconstrictor may be advisable for nonsurgical procedures since the microvasculature is often already compromised by SCA.

Osteomyelitis is much more common in patients with SCA[150] and the risk/benefit ratio for surgical endodontic procedures should be carefully considered for patients with SCA.

Liver Disease

End-stage chronic liver disease (cirrhosis) is the result of hepatocellular injury and necrosis that leads to fibrosis and nodular regeneration. Cirrhosis may be asymptomatic for long periods of time and may be undiagnosed until systemic signs are apparent. Ultimately, chronic liver disease affects multiple body systems. For more information on the pre-liver transplant patient, please see the subsection *solid organ transplantation* earlier in this chapter.

Performing any surgery in the pre-liver transplant patient involves the risk of severe hemorrhage due to thrombocytopenia or reduced hepatic synthesis of coagulation factors. Preoperative evaluation should include a complete blood count with platelet count, prothrombin time (PT) or international normalized ratio (INR), and partial thromboplastin time (PTT) to ensure an intact coagulation system.

Patients with cirrhosis have an increased susceptibility to infection. Odontogenic infections should be treated aggressively that may require the addition of appropriate antibiotic treatment. Antibiotic prophylaxis prior to dental procedures is only recommended if the patient has a history of spontaneous bacterial peritonitis (SBP), ascites (accumulation of excess fluid in the abdomen), another medical indication for antibiotic prophylaxis, or whose medical condition would drastically deteriorate should SBP develop. When antibiotic prophylaxis is indicated in the patient with end-stage liver disease, a recommended oral regimen is 2.0 grams of Amoxicillin plus 500 mg of metronidazole 1 hour before the dental procedure, or patients may be given 2.0 grams of ampicillin plus 500 mg metronidazole intravenously 1 hour before the procedure.[154] Finally, the pharmacokinetics of drugs commonly used in dentistry can be altered in patients with end-stage liver disease. Alteration of medication dosage based upon hepatic compromise, additional medications, and site of metabolism of the medication may require consultation with the patient's physician or experienced dental specialist or referral to hospital environment.

Adrenal Suppression and Long-Term Steroid Use

The adrenal cortex produces mineralocorticoids, such as aldosterone, and glucocorticoids, such as cortisol, which are important in maintaining fluid volume. Adrenocortical insufficiency may result primarily from Addison's disease, an autoimmune condition, or secondarily from hypothalamic or pituitary disease or from the administration of exogenous corticosteroids (30 mg/day or more of cortisol equivalent for more than five days [about 5 mg prednisone]). Supplemental steroids are often recommended before and possibly following surgery to prevent adrenal crisis in patients who receive chronic daily steroid therapy; however, adrenal crisis

during or after dental treatment is very uncommon and most patients probably do not require steroid supplementation.[155] If supplementation is indicated for a minor surgical procedure (such as routine endodontic surgery), the glucocorticoid target is about 25 milligrams of hydrocortisone equivalent (5 mg of prednisone) on the day of surgery. If a moderate risk surgery is to be performed, the glucocorticoid target is about 50 to 75 mg per day of hydrocortisone equivalent on the day of surgery and for 1 postoperative day.

Nonsurgical dental procedures, including nonsurgical root canal treatment, generally require no supplementation; however, this should be reviewed on a case-by-case basis and consideration given to the anticipated procedural stress and patient tolerance for dental treatment.[155] As a rule of thumb, a patient who recently discontinued use of exogenous corticosteroids for more than five days should wait 2 weeks before undergoing surgical procedures. Patients on alternate day steroids do not likely require steroid supplementation, and is often a goal of therapy for those requiring chronic steroid use. Efforts to control pain and infection can decrease the risk of an adrenal crisis.

Allergies

The prevalence of allergies and allergy-related diseases has increased significantly in recent years.[156] Approximately 15 to 20% of dental patients report some form of allergy on their medical history questionnaire and approximately 5% report allergy to one or more drugs.[157] In fact, allergy is the single most common positive finding on the medical history questionnaire.[2] Fortunately, with the exceptions of latex and certain antibiotics, the majority of reported allergies are to substances not typically used in dental treatment. Even so, many materials used in root canal therapy have the potential for eliciting an allergic reaction. The medical history questionnaire serves as the first stage in screening for allergies but should always be supplemented with direct patient questioning about history of allergic reactions to any drugs or substances. True allergic reactions are characterized by one, or more, of the following: skin rash, swelling, urticaria, throat/chest tightness, shortness of breath, rhinorrhea, and conjunctivitis.

Type I (immediate or anaphylactic, IgE-mediated) and Type IV (delayed, cell-mediated) are the two types of allergic reactions most likely to be encountered as a result of exposure to a substance used in endodontic treatment. Type I hypersensitivity requires previous exposure to the antigen and can occur after a single prior exposure or multiple prior exposures to the allergen. The reaction occurs shortly after exposure and can rapidly progress to life threatening anaphylaxis. Type IV hypersensitivity typically appears 48 to 72 hours after exposure and is mediated by T lymphocytes in contrast to the humoral immune system (antibody) mediated Type I reaction. Contact dermatitis is a classic Type IV reaction. When materials used in endodontic treatment come in contact with the periapical tissues (either intentionally or inadvertently), there is the potential for a delayed Type IV hypersensitivity reaction. The allergic potential of various materials commonly used in endodontic treatment is summarized in Table 31-8.

TABLE 31-8 Allergic Potential of Materials Commonly Used in Endodontic Treatment

Category	Material	Allergic potential
Barriers	Natural rubber latex	+
	Vinyl (polyvinyl chloride)	–*
	Nitrile (acrylonitrile and butadiene)	–*
	Polycholoroprene (Neoprene)	–*
Irrigating solutions	Sodium hypochlorite (0.5%–5.25%)	+
	Hydrogen peroxide (3%–30%)	–
	Chlorhexidine (0.2%–2%)	+
	Iodine potassium iodide (2%–5%)	+
	Ethylenediamine tetra acetic acid (EDTA) (10%–17%)	–
	Citric acid (10%–50%)	–
	MTAD (mixture of tetracycline, citric acid, and detergent)	?
Intracanal medications	Phenols	+
	Aldehydes	+
	Calcium hydroxide	–
	Iodine containing pastes	+
Sealers and filling Materials	ZnOE materials (various sealers and temporary filling materials)	+
	Epoxy resins	+
	Glass ionomers	–
	Composite resins	?
	Mineral trioxide aggregate (MTA)	–
	Calcium chelate/polyvinyl resin	–
	Gutta percha (trans 1, 4-isoprene polymer)	?

(Adapted in part from: Hensten A, Jacobsen N. [193] and Zehnder M. [184]). (obtain permission)

Allergy to Local Anesthetics

True Type I allergy to an amide local anesthetic is extremely rare. Nevertheless, patients reporting a history of allergic reaction to a local anesthetic require thorough evaluation prior to proceeding with treatment (assuming a local anesthetic is needed for root canal treatment) since true allergic reactions have been reported.[158–167] Perhaps the most common response elicited upon exploration of the presumed allergic reaction is a report of tachycardia, syncope or general uneasiness following local anesthetic injection. Such a response almost certainly represents a psychogenic reaction rather than true allergy.[166,168] Careful aspiration during injection can help prevent an inadvertent intravascular injection and subsequent increased toxicity and potential for adverse reaction.

In a prospective study of 5,018 dental patients who received a local anesthetic, 25 (0.5%) adverse reactions were recorded. Twenty-two of the reactions were mild, quickly reversible, and considered to be psychogenic in nature. Only two of the reactions were initially viewed as possible allergic reactions and both of these were excluded as true allergic reactions after provocative challenge tests. In another study of 236 patients who experienced an adverse reaction after

injection of local anesthetic, all tested negative following intradermal injection of local anesthetic containing epinephrine and preservative.[169]

Sulfite preservatives used in local anesthetics containing epinephrine and latex allergen released into the anesthetic solution from the vial stopper are both potential causes of allergic reactions. Although reaction to the sulfite preservative is also believed to be rare,[169] allergic reactions to preservatives used in local anesthetics have been reported.[170–172] Since preservatives are only used in local anesthetics containing a vasoconstrictor, the risk of allergic reaction from this potential source can be eliminated by using an anesthetic without vasoconstrictor and preservative (e.g., 3% mepivicaine). Local anesthetic cartridges contain two potential sources of latex allergen that could possibly leach into the anesthetic solution—the rubber stopper and the diaphragm. A review of the literature found no case reports or controlled studies demonstrating that the latex present in a dental local anesthetic cartridge could cause an allergic reaction.[173] However, this same review found several case reports of allergic reactions attributed to trace amounts of latex found in other medication vial stoppers and intravenous tubing. Even though there is no strong evidence to support avoidance of local anesthetic cartridges in patients with a known latex allergy, experts in the area have recommended using local anesthetic from glass enclosed vials to avoid any potential risk of exposure to latex allergens.[167] If it can be determined that the local anesthetic cartridge contains non-latex materials for the diaphragm and stopper, then there is no need for concern.

It should also be noted that a documented allergy to one type of amide local anesthetic does not necessarily imply allergy to all amide local anesthetics and often a readily available alternative can be found after testing.[162,166] As a practical matter, patients referred for allergy testing should be given sample cartridges of at least two different local anesthetics so the allergist can test with the same solution that will be used for dental treatment. Options for patients with a documented allergy to all commonly used local anesthetics include sedation, general anesthesia, electronic anesthesia[166,175] and injectable diphenhydramine. A solution of 1% diphenhydramine with 1:100,000 epinephrine can be compounded by a local pharmacist and used for infiltration or mandibular block injections. The dosage should be limited to a maximum of 50 mg at each appointment.

Allergy to Latex

Of the many materials used in the dental office with the potential for initiating an allergic reaction, natural rubber latex (NRL) is the most common.[176] Reports of allergic reactions to NRL began in 1987 coincident with the widespread adoption of universal precautions, including the use of latex gloves for practically all medical and dental procedures.[177,178] Type IV sensitivity is the most common type and is related to the various chemicals used in processing NRL. The potentially much more serious Type I sensitivity to NRL is a reaction to proteins found in NRL. Approximately 6% of the general population is believed to have Type I sensitivity to NRL and this number increases to as much as 17% for health care workers.[179] Urticaria is the most common initial finding in Type I sensitivity reaction to NRL.[177]

Patients with a history of multiple surgeries (especially spina bifida) or atopy (multiple allergies) and health care workers all have an increased risk of sensitivity to NRL. Some food allergies (e.g., avocado and banana) are associated with an increased risk of latex allergy. Considering the multiple potential sources of exposure to NRL in the dental office (e.g., dental dam material, gloves, local anesthetic cartridges, rubber mouth props, rubber tubing, and even some blood pressure cuffs), history of allergy to NRL requires special treatment modifications. In addition, clinicians who treat patients with known or suspected sensitivity to NRL should be prepared to provide initial management of an acute allergic reaction if necessary.

Consultation with the patient's primary care physician or allergist is advised to help assess the degree of risk, previous reactions and treatment, and possible premedication with a corticosteroid. All potential sources of NRL exposure in the dental office should be considered. Non-latex gloves and dental dam material are now readily available from commercial sources and these items may be easily substituted for NRL containing products. Since latex allergens can be transferred by contact with powder from latex gloves and other sources, it may be prudent to schedule the patient as the first of the day to decrease the chance of contact with residual latex allergens on environmental surfaces, clothing, and room air.[180] The potential for cross-reaction with gutta-percha in NRL sensitive patients has not been demonstrated although caution should be exercised to avoid extrusion of any filling material beyond the confines of the root canal space. Also, as previously discussed, the potential for reaction to latex allergens present in the local anesthetic cartridge stopper or diaphragm should be considered.

Antibiotics and Analgesics

Allergy to penicillin is one of the most common drug allergies and affects approximately 5% to 10% of the population.[157] Although many reported allergic reactions cannot be confirmed, unless the patient is willing to undergo testing to rule out allergy to penicillin, it is safest to assume to the allergy is real and select an alternative antibiotic. In the case of allergy to penicillin, it should be presumed that the patient is also allergic to the synthetic penicillins. In addition, cephalosporins show cross-reactivity but only in approximately 5 to 10% of penicillin allergic patients.[157] Clindamycin is an appropriate alternative to penicillin for treatment of endodontic infections and bacterial endocarditis prophylaxis.[67,181]

Nonsteroidal anti-inflammatory drugs are the usual drugs of first choice for management of endodontic related pain and are tolerated well by most patients. However, caution should be used in prescribing NSAIDs in patients with asthma and/or known allergy or sensitivity to aspirin.

Reports of allergy to codeine are most commonly related to gastrointestinal side effects rather than true allergy, although allergy to opioid analgesics does occur. If the patient's history suggests an adverse reaction related to gastrointestinal distress (including nausea, emesis or constipation), an alternative synthetic narcotic or combination pain medicine may be considered.

Irrigating Solutions

Sodium hypochlorite, in concentrations varying from 0.5% to 6.0%, is currently the most commonly used canal disinfectant and irrigating solution in endodontics.[182–184] Sodium hypochlorite possesses excellent tissue solvent and antimicrobial properties but also demonstrates concentration related tissue toxicity. Although an allergic reaction and/or hypersensitivity to sodium hypochlorite when used as an endodontic irrigating solution is rare, several cases have been reported.[183,185,186] It has been suggested that some patients may be sensitized by exposure to household bleaching products.[183] Alternatives to sodium hypochlorite include: sterile saline or water, chlorhexidine (0.2 – 2%), iodine potassium iodide (2 – 5%), hydrogen peroxide (3%), ethylenediamine tetraacetic acid (EDTA – 10 – 17%), citric acid (10%), and a recently introduced material, MTAD.[184,187–190] Of these alternatives, iodine potassium iodide and chlorhexidine also possess the potential for stimulating an allergic reaction. A recent review article provides an excellent overview of the relative advantages and disadvantages of these selected irrigating solutions.[184]

Intracanal Medications, Cements and Filling Materials

Intracanal medications such as formocreosol, formaldehyde, eugenol, camphorated phenols, and cresatin are all known to be potential allergens.[191,192] Fortunately, these canal medications are not frequently used in current endodontic therapy. Calcium hydroxide paste, a commonly used intra-appointment medication, is not allergenic. Temporary filling materials containing zinc oxide and eugenol (ZnOE) have the potential for allergic reactions and, unlike materials contained exclusively within the confines of the root canal space, are likely to have contact with mucosal tissues.[193]

ZnOE, a potential allergen, is a common component of many root canal sealers and two common root-end filling materials (IRM® and Super EBA®). Sealers containing formaldehyde or paraformaldehyde (such as N2 paste and Endometazone), especially when extruded beyond the apex, have been demonstrated to stimulate often severe allergic reactions.[194–197] Resin-based sealers such as AH26® and AHPlus® also have the potential to stimulate an allergic response[191] although this is believed to be rare. Calcium hydroxide-based sealers such as Sealapex® or glass ionomer sealers such as Ketac-Endo® could be reasonable alternatives for patients with known allergy to any of the components of ZnOE or resin based sealers. As always, one should carefully read the list of ingredients since at least one sealer marketed as a calcium hydroxide based sealer contains a significant ZnOE component.

Dentin-bonded resin-type root-end filling materials have demonstrated excellent biocompatibility in long-term clinical studies with no evidence of allergic reactions in treatment failures.[198–200] However, the choice of resin filling material is important since some resins are known to release formaldehyde when setting. Mineral trioxide aggregate (MTA) and newer bioceramic materials used for root-end fillings, apexification, perforation repair, and pulp capping have demonstrated excellent biocompatibility and no suspected allergic potential.[201–203]

Although there have been case reports of suspected allergic reactions to gutta-percha in patients who were allergic to natural rubber latex (NRL),[204,205] the possibility that the reactions were due to another material used in root canal treatment could not be ruled out. In fact, cross-reactivity between commercially available gutta-percha and NRL has not been demonstrated.[206,207] In addition, gutta-percha is normally well contained within the confines of the root canal space and therefore should not have the potential to elicit an immune response. Gutta-percha manufactured for root canal treatment contains other ingredients such as barium sulfate, zinc oxide, waxes and coloring agents so potential allergy to any of these materials should be considered, especially if there is potential for extrusion of filling material. In patients with multiple allergies (atopy) and suspected allergy to any of the components of gutta-percha, consultation with the patient's physician is advised.

If safe use of the standard obturating material cannot be confirmed, one alternative is to fill the root canal space with MTA or a bioceramic material. This technique presents some technical challenges, especially in smaller canals, but should be manageable by an experienced clinician. MTA has a relatively long setting time (about four hours) but once final hardening has occurred, removal of the filling material through an orthograde approach is difficult and only possible in large, straight canals.

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Chapter 32

Drug Interactions and Laboratory Tests

S. Craig Rhodes, Paul D. Eleazer

Drug-drug interactions, as well as some food-drug, herbal/diet medicine-drug, and vitamin-mineral/drug interactions, are discussed in this chapter. While dentists are not obliged to treat every patient, they cannot deny treatment based upon a patient's disability, such as a medical condition. For more details, see Chapter 31. Therefore, it is important to be prepared to treat patients taking multiple drugs by becoming aware of dangerous drug interactions.

The drug interaction information in this chapter is derived from the work of a panel of pharmacologists who factor the likelihood of an interaction, with the severity of a possible reaction to arrive at a level of clinical significance.[1] Some less-likely reactions, with serious potential adverse results, are also included. Certainly new reactions, some serious, will be discovered as more knowledge accumulates. On-line resources are available for updated information.[2,3]

Principles of pharmacology and history taking can help clinicians determine probable risk for individual patients. Drug interactions can be classed as pharmacokinetic or pharmacodynamic. Pharmacokinetic reactions include changes of rate or extent of absorption, distribution, metabolism, or excretion. Pharmacodynamic drug interactions involve a change in the patient's response without a change in drug plasma level.

An example of a pharmacokinetic absorption is the antidiarrheal Kao-Pectate (kaolin and pectin) that decreases the absorption of tetracycline antibiotics. An example of pharmacokinetic drug interaction of distribution is epinephrine and the beta-blocker drugs competing for the same binding site on albumin. Metabolism-type pharmacokinetic interactions include the macrolide family of antimicrobials, competing for the breakdown liver enzyme pathway with drugs such as Tagamet (cimetidine). Elimination drug reactions include the competition of methotrexate and non-steroidal anti-inflammatory drugs (NSAIDs) for removal by the kidney.

An example of a pharmacodynamics reaction is ethanol and a benzodiazepine combining to increase central nervous system (CNS) sedation without a detectable difference in plasma levels in either of the drugs from their levels if administered alone.

Interviewing the patient may provide valuable clues about possible drug reactions or interactions. When drug action is plotted against response in a large patient population, the graph is almost always a bell-shaped curve, meaning that a few patients develop an exaggerated response and some show very little effect. Most people have the expected response. If a patient has a history of over- or under- response to a drug, the clinician should be alert for a similar response. Drug interactions for members of the same drug class are likely to be analogous, but there can be exceptions. A second major consideration in history taking is the possibility of inaccurate reporting by the patient. Such may be the case with a patient who denies a medical problem, based on a sense of bravado, or one who vainly wants to postpone admission of the encroachment of age. Furthermore, patients may simply forget important details of their drugs or specific dosages. In addition, many lay persons not well versed in medical conditions may not grasp the importance of reporting all of their specific conditions or chronic medications.

Some authors have suggested that generic medications may not be as effective as brand name drugs, especially for certain categories of drugs. An example is illustrated by the discovery of imperfections with enteric coatings that would subsequently not protect the medication from the adverse effects of acid stomach contents.[4] A more recent study demonstrated a lack of bioequivalence between brand name and generic levothyroxine for use in children with severe congenital hypothyroidism. There appears to have been an insensitive FDA pharmacokinetic standard in the production of the generic version of the drug, as compared to the brand name version.[5]

The prudent practitioner should observe drug interaction reports for newly introduced drugs, and be cautious about prescribing these drugs until sufficient time has elapsed to elucidate all reactions. Often, drug interactions are discovered after a drug has been introduced to the market, even though many lab tests, animal tests, and human trials were performed before release of the drug for sale. An example of such a situation is the non-opiate drug ketorolac (Toradol). It was found to be very effective in controlling postoperative dental pain and was widely prescribed before being associated, through post-market surveillance, with stomach bleeding and kidney problems. This eventually led to a brief withdrawal of the oral form from the market, except for brief follow-up to parenteral use.[6] Ketorolac is currently indicated for short-term (≤ 5 days in adult patients) management of moderately-severe acute pain requiring analgesia at an opiate level.

Dentists are fortunate that they seldom need to prescribe drugs for chronic use. Taking medications for relatively shorter periods of time minimizes the potential for drug interactions and side effects that can potentially occur more often when these medications are taken for extended periods of time. Even so, some important drug interactions occur rapidly.

For many drug interactions, the net result is simply a change in reaction to one or both drugs. This change can be an increased response or a decreased effect. Sometimes, a combination of drugs results in an unexpected interaction. An example of a surprising increased drug effect occurs when administering benzodiazepine drugs to patients taking the calcium channel blocker diltiazem (Cardizem). The combination causes little change to diltiazem action, but results, however, in a rapid increase in benzodiazepine sedation, apparently because the calcium channel blocker decreases the metabolic breakdown of the benzodiazepine. In one study, the area under the curve of benzodiazepine over time nearly tripled.[7]

Individual variation plays an important role in much of what we know about drug interactions. Generally, those affected more are the elderly, whose metabolic systems are less robust. Often, those with chronic systemic disease, and those taking multiple medicines, are more likely to have drug interactions. Unknown reactions may also be at work. Patients taking herbal medicines and those on atypical diets may also be more likely to have an unexpected drug reaction.

Some patients may not be greatly affected by a drug interaction and might not report it to their prescribing doctor. Even among observant patients, the number of dramatic drug interactions that affect every patient every time is probably very low. This inconsistency of effect may allow the practitioner to gain confidence in prescribing drugs if they see their patients having no problems. Even for a specific individual, past experience with combining drugs is no guarantee of safety.

The most serious reactions of concern to dentists are listed in Tables 32-1 to 32-12. With theophylline, the margin between therapeutic dose and toxic level is narrow. With international travel now commonplace, drugs removed from the market in one country may be brought in from another. One needs to know overseas trade names as well as the generic labels. Such is the case with the non-sedating antihistamines astemizole (Hismanal) and terfenadine (Seldane), which can cause life-threatening *torsade de pointes* cardiac arrhythmias if combined with macrolide antibiotics.

Systemic epinephrine can adversely affect a patient without drug interaction by increasing anxiety and causing a frightening tachycardia. Keeping the local anesthetic in the local area minimizes the possibility of drug interaction. However, Lipp et al.[8] experimented with labeled epinephrine. They found a 22% incidence of intravascular injection without a positive aspiration. That means that clinicians should not derive a false sense of security, from a lack of aspiration of blood, during an anesthetic injection. Avoidance of systemic interaction can best be assured by a slow injection. Observation of the patient, for signs of systemic injection of epinephrine, such as pallor, tachycardia, and anxiety, are essential. For more details, see Chapter 18.

The concept of decreased drug effect from orally administered antibiotics has received attention in the lay press. Careful research has led to the belief that antibacterial drugs, commonly used by dentists, are quite unlikely to cause failure in oral contraceptives. Research suggests that azole antifungal drugs, such as ketoconazole, may reduce the therapeutic efficacy of hormonal contraceptives. The anti-tuberculosis drug rifampin may also reduce the therapeutic efficacy of hormonal contraceptives. Yet it is prudent for clinicians to suggest alternative methods of contraception to female patients of childbearing age prescribed antibiotics. Birth control pills are not 100% effective along with certain antibiotics.

Over-the-counter drugs can cause interactions of concern to the dentist. Herbals as well as vitamin and mineral supplements can also cause drug interaction problems. Many patients consider these non-prescription drugs to be innocuous dietary supplements, and fail to report taking them in their histories. Recent studies have elucidated many drug interactions with herbal medications and supplements (addressed later in this chapter).

Dentists commonly use antimicrobial drugs, pain relievers, and local anesthetics. Antimicrobial drugs have eliminated many risks of life. The first true antibiotic, penicillin, came into common use among dentists following World War II. Because so many patients have subsequently taken antibiotics, it is of little wonder that many drug interactions with these drugs have been identified.

Tetracyclines were developed after penicillin. As they were gaining in popular usage, penicillin drugs were undergoing structural modifications to afford different forms, in order to counter the emerging antibiotic resistance issues. Erythromycin, the original macrolide antibiotic, was introduced after the tetracyclines. For a time, erythromycin was widely popular among dentists, who chose it primarily because it eliminated the risk of anaphylaxis to penicillin and because antibiotic resistance to this drug originally seemed to be relatively limited. While severe allergic problems have not arisen, bacterial resistance and serious drug interactions have limited the use of erythromycin and its congeners, clarithromycin (Biaxin) and azithromycin (Zithromax).

The many drugs now known to interact with macrolides do so because of shared metabolic pathways that delay metabolism. Higher levels of one or both drugs can commonly result in severe cardiac rhythm problems, some life threatening. Surprisingly, even anti-arrhythmic drugs may cause such arrhythmias when administered with macrolides. One should note that combinations do not always cause a predictable reaction, meaning that prescribing a certain dose for a particular individual without an untoward reaction does not guarantee a similar result for subsequent prescriptions.

Serious reactions of severe muscle wasting may occur with macrolides when combined with "statin" drugs, very commonly used for lowering cholesterol. These drugs inhibit HMG-CoA reductase, a key enzyme in the production pathway of the "bad" or low-density cholesterol. Muscle aches are among the first symptoms that can occur with the drug alone, but occur much more often when the macrolide antibiotic effectively increases its concentration, by impeding

metabolism through their common liver pathway. The symptoms may take a few days to appear. Damaged muscle leads to increased creatinine phosphokinase enzyme levels that are typically used to confirm a clinician's suspicion of this potentially very serious drug interaction.

Ergot derivatives, such as Migranal, mitigate vascular headaches and may interact with macrolide antibiotics, inducing peripheral ischemia secondary to vasospasm. Not all patients respond to reversal therapy for this drug interaction, making this a very serious potential problem.

Yet another category of classic interaction, with drugs slowing metabolic pathways, occurs with macrolides. To reach therapeutic levels, theophylline is often administered in doses close to the toxic threshold. When paired with erythromycin, the decreased elimination may push the concentration of the bronchodilator into a danger zone. Obviously, the higher the patient's therapeutic dose, the greater the risk. Early studies indicate azithromycin probably acts similarly, while clarithromycin (in a study of five patients) did not affect theophylline pharmacotherapeutics.[9,10]

Table 32-1 contains more serious and more probable reactions of drugs with macrolide antibiotics. Dentists who prescribe macrolides should maintain vigilance for signs of any interaction.

Table 32-2 ranks potential reactions to metronidazole (Flagyl), a drug many dentists prescribe. This drug's antibacterial spectrum covers obligate anaerobes, however, some of these microorganisms demonstrate resistance to the drug. Clinicians often use this DNA-impeding agent along with a penicillin or cephalosporin bactericidal drug. Research has shown that most endodontic infections contain multiple organisms, with many facultative microbes.[11] Outcome analysis of microbial susceptibilities of cultures from endodontic infections shows that metronidazole alone is not effective for these infections.[12]

Metronidazole shares the adverse interaction of anticoagulant drugs with all antibiotics. By killing normal gut flora, the production of vitamin K decreases, thus altering the balance between the normal clot-promoting vitamin and the clot-preventing anticoagulant. The result is that a clinical bleeding problem is more likely to occur. Metronidazole has an additional interaction of importance to the overall clotting balance. It further increases bleeding proclivity by a direct inhibition of the metabolism of warfarin (Coumadin).[13]

Also of note, metronidazole shows interaction with ethanol, just as with disulfiram (Antabuse), resulting in nausea and vomiting in many patients. The clinician should caution patients to avoid ethanol while taking metronidazole and for 1 full day thereafter to ensure no untoward drug interaction. Another drug interaction of metronidazole is, when combined with Antabuse, patients taking metronidazole have experienced acute psychotic reactions.

TABLE 32-1 Potential Reactions between Macrolide* Antibiotics and All Drugs

Rapid Reactions	Delayed Reactions
Very significant reaction	
Established reaction	*Established reactions*
▲ Carbamazepine (Tegretol) (→ toxicity) (468)	▲ Cisapride (Propulcid) (increased arrhythmia) (522) ▲ Digoxin (→ toxicity) (792)
Possible reaction	*Probable reaction*
Ergot derivatives (→ peripheral ischemia (884)	▲ Anticoagulants (→ bleed) (187) ▲ "HMG-COA Reductase Inhibitors"(→ myopathy) (rhabdomyolysis) (1070) ▲ Pimozide (Orap) antipsychotic (→ cardiotoxicity) (1642)
	Suspected reactions ▲ Antiarrhythmics (→ arrhythmia) (112) ▲ Eplerenone (Inspira) (hyperkalemia) (→ arrhythmia (875) Grapefruit = ▲ absorption (→ toxicity) (1339) Some quinonolones = (→ arrhythmia) (1341) Verapamil, ▲ macrolide (→ cardiotoxicity) (2286)
Somewhat less significant reaction	*Established reactions* ▲ Theophylline (→ toxicity) (2119) ▲ Corticosteroid (→ possible toxicity) (627) ▲ Cyclosporine = (→ nephrotoxicity amp; neurotoxicity) (676)
	Probable reaction Rifampin (→ ▼ antibacterial/▲ gut effects) (1342)
Suspected reaction	*Suspected reaction*
▲ Benzodiazepines (→ sedation) (328)	Tacrolimus (Prograf) (→▲ tacrolimus toxicity) (2039) Repag (Prandin) (→▲ Repag = ▼ blood glucose (1811)

Numbers indicate page number in Facts and Comparisons, Drug Interactions.[1]

*Macrolides share liver metabolic pathways with many other drugs, generally resulting in delayed metabolism of both drugs, which increases drug levels.

▼ Drug action is probably diminished.

▲ Drug action is probably increased.

TABLE 32-2 Metronidazole* (Flagyl) and All Drugs

Rapid Reactions	Delayed Reactions
Very significant reaction	▲ Anticoagulants (bleeding) (194)
Somewhat less significant reaction	*Suspected reaction*
Suspected reaction	Barbiturates speed metabolism of metronidazole (→ ▼ antibacterial) (1434)
Ethanol (→ disulfiram reaction) (830)	Disulfiram (→ acute psychosis) (830)

Numbers indicate page number in Facts and Comparisons, Drug Interactions.[1]

*This antibiotic decreases metabolism of warfarin. It also may cause nausea if combined with ethanol or disulfiram (Antabuse), but reaction is inconsistent.

▼ Drug action is probably diminished.

▲ Drug action is probably increased.

TABLE 32-3 Penicillins/Cephalosporins and All Drugs

Delayed Reactions
Suspected reaction
Tetracyclines (→ bactericidal drug) (1604)
Methotrexate (→ toxicity) (1411)
Somewhat less significant
Suspected reactions
Allopurinol (Zyloprim) (→ absorption) (1597)
Food → decreases absorption (1602)
Beta-blockers (▼ antihypertensive + antianginal effects) (155)
Warfarin, ▲ bleeding (208)
Aminoglycosides are inactivated by parenteral Penicillin (PCN) (85)
Aminoglycosides + cephalosporin (increased nephrotoxicity) (81)

Numbers indicate page number in Facts and Comparisons, Drug Interactions.[1]

▼ Drug action is probably diminished.

▲ Drug action is probably increased.

Except for potentially fatal allergic reactions (anaphylaxis), the penicillin families of antibiotics, and the cephalosporins, have remained relatively free of serious drug interactions (Table 32-3). The penicillins are very effective against common endodontic pathogens.[12] Most cephalosporins are not effective against the strict anaerobes found in endodontic infections. There are varying levels of cross-allergenicity seen between penicillins and cephalosporins, depending upon the specific chemical structure of the latter.

Methotrexate, a powerful anti-metabolite for some types of cancer and for refractory arthritis patients, when combined with penicillins, vancomycin, trimethoprim, tetracyclines, or sulfonamides has resulted in severe toxicity, including renal failure, myelosuppression, neutropenia, thrombocytopenia, and skin ulcerations. Cephalosporins have not been reported to cause this drug interaction. Metronidazole can also interact with NSAIDs to effect severe toxic reactions resulting from the reduced clearance of methotrexate.

Quinolone antibiotics have potential to cause severe interactions with several drugs (Table 32-4). As with macrolides, quinolones can cause serious cardiac rhythm problems in the presence of many other drugs. The macrolide-quinolone combination is one of the drug interactions that can cause fatal arrhythmias. Macrolides or quinolones plus cisapride (Propulcid) may interrupt normal nerve impulse conduction within the heart that may threaten life. Yet another problem that quinolones have is the drug interaction with theophylline. The shared metabolic pathway similarly causes theophylline concentrations to increase to toxic levels. These similarities occur even though the molecular structures of the two drug classes are quite different.

Coumadin plus quinolone may produce a cardiac rhythm problem.[14] All antibiotics can cause bleeding problems for patients taking anticoagulants because the antibiotic kills gut flora that produce vitamin K, a natural substance that enhances clotting. Lack of this balance means the patient can take longer for his or her blood to clot. When healthy patients take an antibiotic, the reduction in vitamin K causes no clinically apparent change in clotting parameters.

TABLE 32-4 Quinolone Antibiotics and All Drugs

Rapid Reactions	Delayed Reactions
Very significant reaction	*Probable reaction*
	Anticoagulants ▲ bleed (216)
	Suspected reaction
	Serious arrhythmias with phenothiazines (1629)
	Cisapride (Propulsid) (1765)
	Macrolide antibiotics (1341)
	Tricyclic antidepressants (2244)
	Ziprasidone (Geodon) (2341)
	Antiarrhythmics (113)
Somewhat less significant reaction	*Suspected reaction*
Antacids, ▼ Quinolone absorption (1763)	Cyclosporine, nephrotoxicity (698)
Probable reaction	Theophylline ▲ Theo → toxicity (2127)
Sucralfate (Caragate) = ▼ Quinolone absorption (1777)	
Heavy metal salts = ▼ Quinolone absorption (1770)	
Suspected reaction	
Sevelamer (Ranagel) = ▼ Quinolone absorption (1776)	
Tizanidine (Zanaflex) = ▼ Tizanidine metabolism → toxicity (2186)	
Food = ▼ Quinolone absorption (1768)	

Numbers indicate page number in Facts and Comparisons, Drug Interactions.[1]

▼ Drug action is probably diminished.

▲ Drug action is probably increased.

The well-informed dentist should be able to order and evaluate blood-clotting tests such as bleeding time and International Normalized Ratio (INR) with the patient's physician.

Tetracyclines have seen resurgence in popularity among dentists because of their actions against collagenase and their effectiveness against periodontal pathogens, many shared with endodontic diseases. Both of these advantages carry over into endodontics. While there is a long list of drugs that interact with tetracycline, the reactions tend to be inconsistent and rarely life threatening (Table 32-5). The digoxin-tetracycline interaction arguably has the most serious potential. In a small portion of the population, the gut flora metabolizes a significant percentage of their digoxin dose, meaning that they require a fairly high dose to achieve the proper steady-state digoxin level. Tetracyclines can induce significant microflora changes, such that greater digoxin absorption leads to toxic levels. As noted above, macrolides interact with digoxin in a different way, inhibiting renal excretion of digoxin, also reaching potentially toxic levels, and this change can last for many days after the macrolide is stopped.

While it may be nice to contemplate treating endodontic patients without antibiotics, such is an unrealistic dream. The rapid discovery of new interactions mandates that those who treat infected patients keep abreast of contemporary information. No antibiotic is without the potential for interaction with other drugs.

Pain control drugs fall into a similar situation. Certainly, modern endodontic techniques and better appreciation of the value of "not" harming tissues adjacent to root canals have helped reduce the need for pain-relieving drugs. Yet, postoperative pain will remain a factor in treating some endodontic maladies. Clinical experience of many dentists reinforces the experimental observations that NSAIDs are effective pain relievers. Most practitioners find NSAIDs as effective as codeine or hydrocodone. Chronic NSAID use has resulted in stomach and kidney problems, especially among the elderly, but only a few problems have been associated with short-term use.

TABLE 32-5 Tetracyclines and All Drugs

Delayed Reactions

Suspected reaction
Pens/Cephs, cidal/static (1604)
Digoxin, ▲ dig. (812)
Somewhat less significant reaction
Suspected reaction
Activated charcoal, absorbs (490)
Heavy metal salts = chelation (2078) (2080) (2086) (2087)
Retinoids (Accutane) risk of benign intracranial hypertension (1814)

Numbers indicate page number in Facts and Comparisons, Drug Interactions.[1]
▼ Drug action is probably diminished.
▲ Drug action is probably increased.

Serious drug interactions with NSAIDs are relatively rare (Table 32-6). The effect of Coumadin is potentiated by NSAIDs. The prescription of NSAIDs for only a few doses limits the clinical appearance of this interaction. On the other hand, a very serious NSAID reaction is with methotrexate, resulting in kidney failure. This reaction is more likely with high-dose methotrexate, typically used for anti-neoplastic therapy, as opposed to the lower doses used for rheumatoid arthritis refractory to less powerful drugs.

Patients taking beta-blockers may experience hypertension breakthrough when taking NSAIDs. Sulindac (Clinoril) apparently does not have the propensity to cause this reaction.[15]

Lithium toxicity induced by concurrent NSAID use has occurred. The decrease of lithium metabolism has not resulted in clinical problems in healthy patients.[16]

NSAIDs and the very popular antidepressants that work by selective serotonin re-uptake inhibition (SSRI) drugs within brain synapses can interact adversely, as can similar drugs that act at synapses. Increased gastrointestinal (GI) bleeding has been reported, although the problem has also occurred when an SSRI drug was used alone.[17]

Narcotic pain relievers will continue to play a useful role for dentists, albeit less necessary because of improved understanding and better instruments and techniques. Demerol (Meperidine) is the most troublesome for potential interactions. The most serious, sometimes resulting in death, is less likely now because mono-amine oxidase inhibitors are rarely prescribed. Other reactions are also listed in Table 32-7.

Control of intra-operative pain is a standard by which patients judge their endodontic caregiver. Pilots are judged by how smoothly they can land a plane even though this is

TABLE 32-6 Nonsteroidal Anti-inflammatory Drugs (NSAIDs) and All Drugs

Delayed Reactions

Very significant reaction
None
Probable reaction
Anticoagulants ▲ bleeding (203)
Suspected reaction
Methotrexate ▲ Methotrexate (→ toxicity) (1408) (1414)
Somewhat less significant reaction
Probable reaction
Beta-blockers = ▼ hypertensive effect (377)
Suspected reaction
Aminoglycosides, ▲ antibiotic effect (84)
▲ Lithium (→ Li toxicity) (1283)
Selective serotonin re-uptake inhibition (SSRI), ▲ GI bleeding (1556)

Numbers indicate page number in Facts and Comparisons, Drug Interactions.[1]
▼ Drug action is probably diminished.
▲ Drug action is probably increased.

TABLE 32-7 Narcotics and All Drugs

Rapid Reactions	Delayed Reactions
Very significant reaction	
Demerol and Mao inhibitors (1372)	
Somewhat less significant reaction	
Probable reaction	
Demerol + Phenothiazines (1373)	
Suspected reaction	*Suspected reaction*
Barbiturates (1369)	Demerol + Ritonavir (Norvir) (1374)

Numbers indicate page number in Facts and Comparisons, Drug Interactions.[1]

Note that death has been reported from giving Demerol to patients taking monoamine oxidase inhibitors (MAO).

*Most reactions are due to additive central nervous system effects.

but a small part of a pilot's overall skill set. Similarly, patients will judge us by our skill at giving an "easy shot."

There are two separate drug interaction considerations with local anesthetics, the anesthetic itself and the vasoconstrictor. Epinephrine is very rapidly metabolized, so any new drug interaction becomes rapidly apparent to the clinician (Table 32-8). Tolerance of the vasoconstrictor in one dose means that an additional dose after about 5 minutes can be given with similar result, given that neither is injected intravascularly. Toxicity of the antibiotic drug is another matter.

Hypertensive events have been documented in patients taking beta-blockers, furazolidone (Furoxone), tricyclic antidepressants, Methyldopa, and the anti-hypertensive drugs guanethedine (Ismelin) and Rauwolfia alkaloids. Beta-blocker interactions are potentially the most serious. It is advisable to take a preoperative blood pressure reading to establish a baseline for that patient. Furthermore, intravascular injection can occur without a positive aspiration of blood.[8]

The anesthetic itself does not cause any serious drug interactions (Table 32-9). The problem with anesthetics is that repeated injections can build to overdose levels because, unlike the very rapidly metabolized vasoconstrictor, the anesthetic agent is metabolized over hours. This is especially important to note for pediatric patients whose body mass cannot tolerate as much anesthetic as an adult.

Beta-blockers may interact with lidocaine, lowering the number of doses to reach the toxic range. This drug interaction is believed to occur via inhibition of hepatic metabolic enzymes.[18]

Cimetidine (Tagamet) increases lidocaine levels too, probably by the same general effect on liver enzymes. Yet, the reaction does not occur every time the drugs are combined.[19] Research on the other histamine H2 antagonists failed to show this interaction.

Benzodiazepine reactions are typically one of the increased drug-drug effects. This can be accounted for by beginning with a lower dose, perhaps half a pill, until the patient's own reaction is defined. The protease inhibitor reactions, however, have been reported to induce severe sedation and respiratory depression. As noted in Table 32-10, no drug

TABLE 32-8 Epinephrine-Containing Local Anesthetics and All Drugs

Rapid Reactions
Very significant reaction
Established reaction
Beta Blockers → hypertension then bradycardia (871)
Suspected reaction
Furazolidone (Furozone) antibiotic → hypertension (2008)
Somewhat less significant reaction
Established reaction
Tricyclic antidepressants → hypertension (2018)
Suspected reaction
Rauwolfia alkaloids → hypertension (2016)
Methyldopa → hypertension (2014)
Guanethidine → hypertension (1011)

Numbers indicate page number in Facts and Comparisons, Drug Interactions.[1]

Epinephrine is rapidly metabolized, so delayed reactions do not occur.

Minimize risk by injecting and watching patient reaction.

TABLE 32-9 Local Anesthetics and All Drugs

Rapid Reactions
Somewhat less significant reaction
Established reaction
Beta blockers (→ (Lido. Toxicity)) (1255)
Histamine H2 Antagonists (Tagamet) (→ Lido. Toxicity) (1257)
Suspected reaction
Succinylcholine, ▲ succinylcholine half-life (1949)

Numbers indicate page number in Facts and Comparisons, Drug Interactions.[1]

▼ Drug action is probably diminished.

▲ Drug action is probably increased.

TABLE 32-10 Benzodiazepines* and All Drugs

Rapid Reactions	Delayed Reactions
Somewhat less significant reaction	
Established reactions	*Suspected reactions*
Ethanol (907)	Carbamazepine (Tegretol) (303)
Azole antifungals (299)	Hydantoins (Dilantin) (1091)
	Modanfinil (Provigil) (329)
	Non-nucleoside reverse transcriptase inhibitors (331)
	Protease inhibitors (336)
	St. John's Wort (341)
	Macrolide antibiotics (325)
	Rifampin (339)
Probable reaction	
Diltiazem (Cardizem) (314)	
Food (319)	

Numbers indicate page number in Facts and Comparisons, Drug Interactions.[1]

*In most reactions, the benzodiazepine effect is increased, but is decreased with Rifampin. Generally, titration of dosage is indicated.

TABLE 32-11 Drugs Prescribed by Dentists that May Interact with Warfarin Anticoagulant

Delayed Reactions

Very significant reaction
- *Established reactions*
  - Sulfonamides, bleed (233)
  - Metronidazole, bleed (194)
  - Vitamin E, bleed (250)
  - Aspirin (ASA), bleed (223)
- *Probable reaction*
  - Nonsteroidal anti-inflammatory drugs (NSAIDs)/(203) CoX_2 inhibitors (204) = bleed
  - Quinolones = bleed (216)
  - Macrolides = bleed (187)
- *Somewhat less significant reaction*
  - *Established reaction*
  - *Suspected reactions*
    - Acetaminophen = ▲ Vitamin K, thus ▼ bleed (119)
    - Carbamazepine (Tegretol), ▼ bleed (137)
    - Vitamin K = ▼ bleed (251)
    - Clopidogrel = ▲ bleed (146)

Numbers indicate page number in Facts and Comparisons, Drug Interactions.[1]

Most drug interactions result in increased bleeding. Tylenol, Tegretol, rifampin, and vitamin K increase bleeding. The Tylenol reaction is apparently not consistent and, if reported, exceeded six doses/week. Vitamin K reverses the action of warfarin. However, there is currently no antidote for Plavix.

▼ Drug action is probably diminished.

▲ Drug action is probably increased.

TABLE 32-12 Drug Prescribed by Dentist that May Interact with Herbal Medicines

Significance Levels	
1	Amoxicillin + Khat →▼ amoxicillin (1603)
1	ASA or NSAIDs + Ginkgo biloba → increased bleeding via decreased platelet aggregation (1550)
1	Benzodiazepines + Kava →▲ benzodiazepine levels (sedation) (325)
	Benzodiazepine + St. John's Wort →▼ benzodiazepine (sedation) (341)
2	Corticosteroids + Licorice →▲ corticosteroid (626)
1	Ciprofloxacin + Calcium-supplemented orange juice → ▲ Cipro (1772) ▼antibiotic absorption
1	Cipro + Zinc →▲Cipro (1778) ▼ antibiotic absorption (1778)
1	Acetaminophen + Ethyl Alcohol → hepatotoxic metabolite (20)
1	Macrolide antibiotics + Grapefruit →▲ absorption of macrolides (toxicity) (1339)
2	Tetracycline + Zinc →▲ Tetracycline ▼ antibiotic absorption (2089)
1	Tetracycline + dairy products → ▼ Tetracycline due to chelation (2085)
	Levofloxacin + orange juice (plain or with Ca^{++}) →▼Levoflox (1772)

Numbers indicate page number in Facts and Comparisons, Drug Interactions.[1]

Significance Level 1 = herbal drug should not be combined.

Significance Level 2 = herbal drug may be continuously allowed.

▼ Drug action is probably diminished.

▲ Drug action is probably increased.

interactions with benzodiazepines have been ranked in the very significant category.

Table 32-11 lists drugs that may be used by dentists that can interact with Coumadin (Warfarin). This extensive list calls for careful consideration by the dentist and alerting the patient to watch for signs of bleeding. Notably, the rapid reaction category is empty, indicating that short-term use carries with it less potential for harm.

DRUG INTERACTIONS WITH DIETARY SUPPLEMENTS

The U.S. Food and Drug Administration (FDA) defines a dietary supplement as a product intended for ingestion that contains a "dietary ingredient" intended to add further nutritional value to (supplement) the diet. Such "dietary ingredients" may include but are not limited to vitamins, minerals, and herbs or other botanicals. Such supplements may be obtained in various forms such as tablets, capsules, softgels, gelcaps, liquids, or powders.[20]

As consumers live longer, take an increasing number of medications, and more commonly experience medical conditions such as diabetes and obesity, it is likely that the use of dietary supplements will likewise increase. Many patients are hesitant to disclose and/or discuss their use of dietary supplements with their dentist, unless specifically asked to provide such information. Many of these supplements have the potential to adversely interact with some of the prescribed medications patients ingest. One study found that 45% of herbal supplement users were exposed to at least one potential drug–herb interaction.[21] It behooves all dental practitioners to query patients about their use of dietary supplements. What specific supplements, in what dosages, by what regimens, and in what forms are appropriate questions. It is also helpful to note if the patient's physician has recommended the supplements and for what specific medical conditions the supplements are being taken.

DRUG INTERACTIONS WITH FOOD AND HERBALS

Licorice is the root of *Glycyrrhiza glabra*, a legume native to India, Southern Europe, and parts of Asia. It is commonly used as a flavoring agent in tobacco and confectionary foods. The purported medicinal uses of it include anti-inflammatory, antiviral, antimicrobial, and blood-pressure

increasing effects. The principal active ingredient in licorice is glycyrrhizin that has been shown to alter levels of corticosteroids, either increasing or decreasing the drug.[22] The mechanism is thought to be an inhibition of the conversion of cortisol to cortisone, by interfering with the enzyme 11-betahydroxysteroid dehydrogenase. The overall effect can be one similar to hyperaldosteronism.[23] The interaction could be more serious in patients on a high-dose steroid.

Grapefruit juice mediated drug interactions have been shown to occur due to an irreversible inhibition of cytochrome P450 enzymes (specifically CYP3A4) involved in phase I drug metabolism. The result may be damaging to high levels of the drug, even in the absence of other drugs. This action contradicts the typical finding, that food binds with drugs to decrease absorption, or at least slows drug absorption.

Orange juice, particularly when calcium-fortified, lowers the levels of the fluoroquinones Cipro (ciprofloxacin) and Levaquin (levofloxacin). Both orange and apple juices have been shown to inhibit organic anion transporting polypeptide 2B1 (OATP2B1), an uptake transporter that is associated with the oral absorption of drugs, notably fexofenadine (Allegra).[24]

Dairy products all contain the heavy metal ion calcium that can hinder the absorption of tetracyclines (demeclocycline, tetracycline, doxycycline, and minocycline). Doxycycline is the least affected of the tetracycline family to bind with heavy metal ions in a process called chelation.[25] Calcium in dairy products and supplements may also impair the absorption of fluoride. Therefore, patients should be advised to avoid intake of dairy products for at least 1 hour before, or 2 after, the administration of these drugs.

Ethyl alcohol was discussed previously in this chapter, but there is a brief review here.[26] Perhaps, because it is so often used or abused among patients, alcohol is sometimes considered a food. Generally, its interactions serve to increase the effect of drugs that act on the CNS. It delays gastric emptying, so can delay absorption of almost any drug taken by mouth. It specifically interacts with metronidazole in a disulfiram class reaction, inducing nausea and vomiting. All the above reactions are dose-related, so the astute clinician should caution patients accordingly. The interaction with Tylenol (acetaminophen) is different and potentially lethal. The chronic abuse of ethyl alcohol induces the CYP P450 enzymes that metabolize acetaminophen, resulting in the rapid metabolism of acetaminophen, with consequent high levels of a hepatotoxic acetaminophen metabolite, known as NAPQI, N-acetyl-p-benzoquinone.

Khat (*Catha edulis*) is a stimulant plant from East Africa and Southern Arabia that is usually chewed to reduce fatigue and appetite. It has been imported into the United States and other countries as a recreational euphoric agent. The euphoric effect is attributed to cathinone, a sympathomimetic amine similar to amphetamine.[27] It is sometimes smoked to achieve higher concentrations that may induce hallucinations.

Khat has been shown to decrease the absorption of amoxicillin and its close congener, ampicillin. This may lead to persistence of an infection. A small study demonstrated the maximum decrease to occur 2 hours after chewing khat.[28] Chronic khat use can result in arrhythmias, hypertension, and ischemic cardiomyopathy. As such, it has been recommended to use minimal amounts of sympathomimetic drugs for such patients.[29]

Ginkgo biloba, commonly used as a memory enhancer, interacts with salicylates and NSAIDS to increase bleeding. The apparent mechanism is by lowered platelet aggregation that decreases blood coagulation.

Kava (*Piper methysticum*), a perennial shrub native to regions of the South Pacific, is drunk as a tea and also can be obtained in pill or liquid forms. It is used to relieve stress and produce a sense of well-being. A single, but serious drug interaction-induced coma has been reported. While the reaction occurred when combined with alprazolam (Xanax), experts anticipate that the danger likely extends to the entire benzodiazepine family.[30] Based upon concerns regarding hepatotoxicity, Kava has been either restricted or withdrawn from several countries since 2002.[31]

St. John's wort (SJW) (*Hypericum perforatum*), a herbaceous perennial plant native to Europe, is the leading complementary and alternative medicine (CAM) treatment for depression and related psychiatric disorders in the United States.[32] It has the potential for causing several drug interactions. Chief among them are the drugs that are cytochrome P450 (3A4, 2C19, 2E1) and/or intestinal P-glycoprotein substrates. The metabolism of such drugs as erythromycin, prednisone, tetracycline, several benzodiazepines, clarithromycin, warfarin, cyclosporine, statins, and clindamycin is induced by SJW, leading to a potentially reduced overall effectiveness of these drugs.[33] Also, serotonin syndrome may result when SJW is taken along with the anti-depression selective serotonin reuptake inhibitors (SSRIs), or with 5-HT receptor agonists within the brain.[34]

Evening primrose (*Oenothera biennis*) is an herbaceous flowering plant native to the Americas. It is commonly marketed in an oil form as a purported remedy for rheumatoid arthritis, premenstrual syndrome, and eczema although these claims are lacking in scientific evidence. Its seed extract contains polyphenols that are attributed with having antioxidant properties. It has been reported to have cariostatic effects by inhibiting the growth of *Streptococcus mutans* and glucosyltransferase activities.[35] It has also been demonstrated to have anti-inflammatory activity.[36] Evening primrose oil interacts with salicylates and NSAIDs, potentially resulting in an additive anticoagulant effect.

Valerian (*Valeriana officinalis*) is a perennial flowering plant native to Europe and portions of Asia and has been introduced into North America. It is commonly provided in a capsule form and is advocated for use as a sedative or anxiolytic. As with the drug interactions seen with SJW, valerian has influence on the activity of the cytochrome P450 enzymes involved in phase I drug metabolism. Interactions

of valerian with drugs such as benzodiazepines, opiates, antihistamines, and other sedative hypnotics such as zaleplon and zolpidem can lead to additive therapeutic and adverse effects (increased sedation).[33]

DRUG INTERACTIONS WITH VITAMIN AND MINERAL SUPPLEMENTS

An adequate diet should provide all the necessary vitamins and minerals required by a healthy adult. Yet vitamins and minerals continue to be purchased in ever-greater numbers. In 2010, Amercian Association of Retired Persons (AARP) and the National Center for Complementary and Alternative Medicine (NCCAM) conducted a telephone survey aimed at assessing the discussion of complementary and alternative medicines (CAM) with their healthcare providers.[37] The survey indicated 47% of adults over age 50 reported using CAM in the previous 12 months and 53% reported using them at some time in their lives. Of CAM users, nearly 70% admitted not discussing their supplement use with their healthcare providers. It is important for dentists to ask their patients about their use of CAMs in order to be able to accurately assess the potential for drug interactions.

The excessive use of zinc-containing denture adhesives can lead to a deficiency of copper that in turn can cause symptoms such as paresthesias, gait and balance problems, and anemia.[38] Cipro (ciprofloxacin) absorption is also diminished in the presence of zinc, as are tetracycline concentrations. This essential heavy metal additive is common in multivitamin preparations. The net result of the interactions is lowering of antibiotic levels in the 25% to 40% range.[39,40]

Vitamin D may induce cytochrome P450 (CYP3A4) that could result in reduced availability of its substrate drugs (midazolam, erythromycin, ketoconazole, itraconazole, triazolam, clarithromycin, and erythromycin).

Vitamin E can interact with anticoagulant and antiplatelet medications (salicylates, clopidogrel, heparin, low-molecular-weight heparins, warfarin, and NSAIDs) to increase bleeding via the inhibition of platelet aggregation. It also has the potential to antagonize the effects of vitamin K-dependent clotting factors with 800 units/day or more. Vitamin E also poses the theoretical risk of increasing the metabolism of substrate drugs for CYP3A4, as already mentioned for vitamin D.[41]

Excessive intake of vitamin K can lower the anticoagulant effect of warfarin.[41]

DRUG INTERACTIONS WITH WEIGHT-LOSS MEDICATIONS

As the incidence of obesity continues to escalate among children and adults, many people turn to weight-loss medicines, as their efforts toward lifestyle modification prove insufficient. Medications used to treat obesity have the potential to interact with drugs commonly used in dentistry.

Orlistat inhibits the absorption of dietary fats by up to 30%. It has the potential to enhance the anticoagulant effect of warfarin. Careful monitoring of the patient's INR should be done to make sure it is within an acceptable range (2.0–3.5).[42]

Phentermine, as an amphetamine derivative, has the potential to enhance the sympathomimetic response to epinephrine and levonordefrin in dental local anesthetics, leading to increased blood pressure and cardiotoxicity, especially in existing cardiac patients.[43] The same caveat applies to patients taking the phentermine-topiramate combination pill (Qysmia). This combination medication also has the potential to impact with sympathomimetics or benzodiazepines.[44]

Phendimetrazine, diethylpropion, and benzphetamine are also sympathomimetic amines with properties similar to those of the amphetamines. They have the same potential to modify the CNS effects of dopamine, serotonin, or norepinephrine. The same cardiac and blood pressure precautions should be employed when these drugs are reported by patients, and especially so for those with existing cardiovascular conditions.[42]

LAB TESTS OF POTENTIAL IMPORTANCE IN ENDODONTICS

Laboratory tests are commonly helpful to dentists. The list of such lab tests is not exhaustive, and dentists should not be limited to those tests listed below. Dentists may be uncomfortable in ordering laboratory tests due to unfamiliarity or perceived patient resistance.

A recent survey was conducted to assess patient attitudes towards chair side medical screening in a dental setting.[45] The survey results indicated the majority of respondents were willing to have a dentist conduct screening for heart disease, hypertension, diabetes, HIV, and hepatitis infection. Referral to a physician is always appropriate, but there is no reason for a dentist to be intimidated by the process of ordering lab tests for their patients.

Dentists are often the first healthcare provider to identify systemic disease. Oral tissues are often the first to be affected. Moreover, many patients do not regularly visit their physician, or many receive only cursory information on subtleties of oral changes from disease. Leukemia, bacterial endocarditis, various cancers, and many other diseases are first suspected in the dental office. Ordering a laboratory test to confirm or rule-out suspicions is perfectly appropriate for any dentist. Culture and sensitivity testing, for identification of a cause of infection and the most likely effective antimicrobial, should be included in every dentist's armamentarium.

Typically, dentists will ask patients to complete a medical history questionnaire. Prior to providing consultations, examinations, and treatment, results should be reviewed during a dialogue between the dentist and patient prior to the physical examination. In order to obtain all the necessary

information for the formulation of an adequate risk assessment,[46] patients are then assigned to a physical status category, commonly the one based on the classification system developed by the American Society of Anesthesiologists (ASA).[47] Medical consultations with the patient's personal physician(s) are often necessary when the dentist is uncertain of the accurate assignment of physical status category and/or the anticipated degree of risk to the patient.

Problems with this recommended protocol can arise when patients are either unaware of their health status, are hesitant to provide this information within the dental office setting, or are less than truthful regarding medical conditions they may have for concerns of privacy and/or fees. It is this first group of patients, the ones who may be unaware of their health status, who may benefit the most from medical screenings involving lab tests within the dental office setting. One recent study provided medical screening tests to patients within a dental setting in order to examine the relationships between the laboratory test results and the patient's self-reported medical health status.[48] The authors found 83% of the participants had one or more abnormal test results and did not disclose a relevant medical condition in their history. The results suggest that many patients would benefit from screening within the dental office.

The dental profession has always taken a leading role promoting prevention. As healthcare reform has rapidly evolved within the United States over the past decade, a renewed emphasis has been placed on prevention, along with the integration of healthcare practitioners involved in primary care across professions and disciplines. The Affordable Care Act (ACA) includes recommendations designed to shift the focus of healthcare away from the traditional treatment of illness toward a system more aligned with overall patient wellness. The expansion of the dental profession, to incorporate the medical screening of patients for undiagnosed conditions, is a logical extension of our historical emphasis on preventive health care. In time, this may prove to play an important role in attempts to control the epidemic rates of incidence of such chronic maladies as cardiovascular disease and diabetes mellitus.

Cardiovascular disease (CVD) and diabetes mellitus (DM) are important public health concerns, and assessing disease risks for these conditions can readily be accomplished within a dental setting. For CVD the Framingham-based risk score is widely used.[49] Use of this algorithm can assess the patient's risk of experiencing coronary heart disease within the next 10 years. In assessing the risk for coronary heart disease, screening tests such as blood pressure, high- and low-density lipoproteins, triglycerides, and body mass index, over a 1-year period, could be utilized. DM-appropriate screening tests could include hemoglobin A1c (HbA1c), random plasma glucose, body mass index, urine glucose, and glucose challenge testing.

Other screening tests, including liver function tests such as aspartate aminotransferase (AST), hematocrit, hemoglobin, herpes simplex virus (HSV), complete blood cell count (CBC), urinalysis, and a standard blood chemistry panel, have also been utilized in previous studies conducted in a dental setting.[48,50]

Disorders of inflammation, perhaps related to a chronic dental infection, may cause an increased erythrocyte sedimentation rate (ESR). Blood tests may be helpful identifying blood dyscrasias, from cellular imbalances to inflammatory system evaluation, to clotting factors. Most classic hemophiliacs are diagnosed early in life and can provide the dentist with historically important information. Every dentist should be aware of the risks associated with altered blood clotting profiles of their patients. Newly identified risk assessment studies have shown the mortality and morbidity of decreasing anti-clotting parameters. The dentist should be comfortable interpreting these tests, to determine if a patient is a candidate for a surgical procedure.

The prothrombin time (PT) test has been largely replaced by the INR that uses a standardized control and expresses the result in a percent of normal clotting time. This test evaluates the extrinsic clotting mechanism. With an INR of 1 being the normal level, minor dental alveolar surgery can be carried out to an INR level, <3.5 with local methods of hemostasis sufficing. For other than minor surgery, consultation should be obtained with consideration for modification of the anticoagulation or hospitalization.

The partial thromboplastin time (PTT) test remains valuable to determine function of the intrinsic clotting pathway that includes the clotting factors in blood plasma.

Bleeding time, the number of minutes required for a standard wound to clot, may help diagnose later onset problems of importance to the dentist such as von Willebrand's disease, where the intrinsic factor VIII is diminished by this hereditary defect. Also, bleeding time identified disorders of the platelet system, including those induced by salicylates or NSAIDs.

The CBC test enumerates platelets, RBC and WBC counts, and can confirm or rule out many suspicious findings. An increased number of red blood cells, polycythemia vera, can reveal itself clinically as darker gingiva, due to the engorgement of the vascular system. An increase in the number of white blood cells can manifest as bleeding gingival that is seen in many forms of leukemia. Petechiae due to sickle cell anemia can be diagnosed by the occasional characteristic shaped red blood cells clogging oral capillaries.

While it is easier to direct patients and staff, who are accidentally exposed to blood-borne pathogens, to someone trained in counseling for such risks, tests for such infectious agents may be ordered by the dentist who suspects a disease.

Patients with unexplained radiolucencies of their jaws may need testing for certain disorders. For example, a multiple myeloma patient will have elevated abnormal antibod-ies known as Bence-Jones protein in his or her urine. A patient with hyperparathyroidism-caused bone radiolucencies will have elevated blood calcium and decreased blood phosphorous level. Urinary levels of calcium may

be lower, with increased phosphorous found in the urine. Serum alkaline phosphatase (ALP) is elevated in hyperparathyroidism, although not as markedly as with Paget's disease.

Allergies can make life unpleasant for many. They may stem from food allergies that can cause nutritional imbalances and may be suggested by changes in the appearance of oral tissues. This may include commonly used dental materials, such as nickel or eugenol, making the patient less likely to have a favorable treatment outcome. The dentist who suspects such a problem will probably refer the patient to an allergist for skin or other tests and then help manage the dental aspects of the patient's care.

SUMMARY

There is a limited understanding of drug interaction incidence because of under-reporting. Also, animal studies may not directly transfer to the human. Animals cannot express mood changes effectively, and there are some metabolic differences between animals and man.

The astute clinician should explain possible adverse or undesirable reactions with patients so that they may watch for an adverse outcome. This chapter emphasizes drug reactions that are potentially very serious and/or are very likely to occur. Some practitioners will see interactions of drugs beyond those covered here. It is important to report suspected drug interactions to the FDA for evaluation through their Medwatch program.[51] The FDA periodically sends information about newly discovered, serious drug interactions, to practitioners registered by state license to practice dentistry. The dentist should report suspected drug interactions to the appropriate administrations in their countries. Furthermore, the reader should remember that information about drug interactions is constantly changing, mostly with newly discovered interaction reports.

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Chapter 33

Outcomes of Endodontic Therapy

Kishor Gulabivala, Yuan-Ling Ng

HISTORICAL CONTEXT OF EVALUATING ENDODONTIC OUTCOMES

The development of medical and dental practices has been guided by prevailing philosophies and consensus of expert opinions. Fears of fatal oral sepsis from deficient root canal treatments at the beginning of the last century led to widespread extraction of pulpless teeth. Endodontics virtually disappeared from many dental schools as the focal infection theory reigned for about 50 years[1] until around 1940.

Rescue of the Endodontic discipline was attributed to individual diligent dental practitioners in Europe and United States, who meticulously recorded their treatments, as well as their outcomes to demonstrate their effectiveness in controlling root canal infection. Such individual endeavors of merit restored the discipline of endodontics that was granted its specialist status in 1952 in the United States.

The costs of health care have risen alarmingly as improvements in general health have driven the survival of western populations, as well as their teeth, much later into life, prompting a host of re-evaluations of the altered economic burdens on society. The *cost-effectiveness of treatment procedures* for management of diseases, therefore, came under close scrutiny and thus began the era of evidence-based medicine and dentistry, with its emphasis on costs, benefits, and outcomes of treatment. Individual studies proved too small to provide confident estimates of intervention outcomes in the wider population. Attempts to pool outcome data for greater power brought the realization that the collective pool of data originating from different centers was heterogeneous and precluded definitive conclusions. Characterization of data types and their quality naturally led to the realization of the need for standardization of approaches in measuring outcomes to enable more meaningful pooling.

A modern re-visitation of the "focal infection era" in the last 20 years, rather than threaten the endodontic discipline, as in the past, prompted the urgency for establishing a better evidence-base for judging the impact on systemic health. Ironically, the modern threat to the endodontic discipline 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. Once again, the science of evidence-based practice has helped to avert irrational options-appraisals that lean towards extraction of savable teeth.[2,3]

By the time of writing, the pendulum had swung firmly back in favor of maintaining predictably restorable teeth rather than to extracting and replacing them with implant-retained crowns. The clinician must, therefore, be ready to play a critical and influential role in restorative treatment planning decisions. Endodontists must not be relegated to the subordinate role of "root canal therapists" providing technical execution of the procedure on prescription. It follows, therefore, that the endodontist must have a sound knowledge of the basic science underpinning the clinical discipline, as well as a rounded clinician with good general treatment planning skills.

Types of Disease and Its Treatment

Endodontic treatment is aimed at treating pulpal disease caused by inflammation and/or infection that generally commence in the pulp tissue and progress to the periradicular tissues *via* portals of communication conveying the neuro-vascular supply. Direct spread of disease across the cementum is precluded by its nature.

Incipient or established-but-limited pulpitis may be managed by vital pulp therapy when it is believed that a sufficient body of pulp tissue remains healthy and unaffected. The direct and uncontrolled spread of pulp inflammation is buffered by specific neuro-vascular mechanisms, distinct from those in the remainder of the body to prevent the pulp from self-strangulation. More advanced pulpitis judged to be on the verge of involving the apical tissues, may require root canal treatment. Where pulpal necrosis and infection have already ensued with consequent apical inflammation, root canal treatment is required to manage the resident intra-radicular infection, unless some natural or iatrogenic obstruction precludes access to the apical anatomy, in which case periradicular surgery may be required for such access. Persistent apical periodontitis following technically inadequate root canal treatment may require root canal retreatment. Persistent apical periodontitis following technically adequate root canal treatment may require root canal retreatment or periradicular surgery to manage the source of the persistent disease.

Recently, a new treatment modality, the "regenerative endodontic procedure" (also called pulp regeneration or revascularization) has been introduced for "*de novo* synthesis" of pulp or connective tissue inside the immature teeth with pulp necrosis and apical periodontitis leading to arrested root development. "Endodontic therapy" is therefore a collective and non-specific term for a range of different procedures

directed at managing disease of different degrees of severity and extent within the pulp tissue or extending beyond the apical confines of the root(s).

Endodontic treatment encompasses the following distinct procedures:

1. Vital pulp therapy (indirect pulp therapy, direct pulp capping, pulpotomy);
2. Nonsurgical root canal treatment;
3. Surgical root canal treatment;
4. Nonsurgical root canal retreatment;
5. Surgical retreatment;
6. Regenerative endodontic procedure.

The ideal outcome for endodontic treatment consists of controlled reduction of infection and 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 have to be employed to evaluate the presence or absence of inflammation or its resolution. This process is further complicated by the lack of direct correlation between measures of chronic inflammation and its clinical manifestation, as well as the sensitivity of methods to image such tissues.

TYPES OF OUTCOME MEASURES

Chronic inflammation is typically clinically asymptomatic, unless superimposed by acute exacerbation. Clinical detection of chronic inflammation of pulpal and periradicular tissues, that are hidden from direct view, is consequently challenging and requires indirect or associated (proxy) measures. The process calls upon the clinician to triangulate various sources of information to form a judgment about the presence or absence of disease.

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, microbial load reduction, and technical quality of root filling may all be regarded as outcome measures by this definition. The ultimate *clinical* measures, however, assess the prevention or resolution of disease (or inflammation).

The outcome of endodontic treatment may be assessed in four dimensions as in other medical disciplines. The first dimension is physical/physiological and related to presence or absence of pulpal or 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; whilst the fourth dimension examines psychological aspects involving perceptions of Oral Health Related Quality of Life (OHRQoL) and esthetics.

PURPOSE OF EVALUATING OUTCOMES

Evaluation of treatment outcomes enables the effectiveness of interventions to be determined. In association with the characterization of the properties of the intervention protocol, the specific factors affecting the outcome may be determined, that in turn may help to estimate the prognosis of impending treatments.

Effectiveness of Procedures

Treatment procedures must be effective for them to be recommended to patients as an option. This must form part of the informed consent process, wherein the risks, benefits, and potential outcomes are articulated to the patient. 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. Pooled average outcome data may not, however, always placate patients that the particular endodontist offering the treatment does indeed have the requisite experience, skills, and personal outcomes matching at least the average figures of performance. It behooves individual practitioners to observe and develop their own personal outcome data; a growing practice in modern surgery.

Personal outcome data offers the patient more precise measures for comparison and expectation. It also aids the practitioner in refining their technique and knowledge to further improve their outcomes, ultimately to help improve the overall pooled data.[4]

Factors Affecting Outcomes

Pooling homogenous data offers the potential to evaluate and prioritize the factors that exert a dominant influence on outcomes. In this way, protocols for treatment may be refined in a progressive fashion so as to reach for the perfect meld of technical performance, clinical outcome, and biological effect necessary for the most predictable and favourable result. Evaluation of factors (microbiota, immune and healing response, intervention, host, operator) affecting treatment outcomes arguably provides the strongest driver and hub for generation of translational *hypotheses* to improve outcomes. Weighing the relative importance of individual factors should help to pinpoint the key biological and clinical factors at play and how best to manage them technically and clinically.

Value for Prognostication

The science of prognostication (predicting, projecting, prophesising, or foretelling the likely outcome of treatment) for an individual tooth is not well advanced in endodontics as it is at least partly based on factors affecting outcomes of endodontic treatment. The latter knowledge and its biologic correlates are still in the process of elucidation. Furthermore, the overall prognosis of a tooth is dependent

upon three individual prognoses and their summative effect. These include the endodontic, periodontal, and restorative prognoses. Each subsumes a set of subsidiary factors that will influence the 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 and overall dental function.

The complexity of the problem of analyzing the factors affecting outcomes of endodontic treatment is highlighted by comparison with the randomized controlled trial of a drug as a treatment intervention. Drugs have a clearly prescribed and standardized dose delivered by specified timings to affect an anticipated blood or target tissue concentration for a defined duration. Treatment data recording is confined to compliance with the prescription, possible checking of blood or tissue levels and the final outcome measure.

In stark contrast, a surgical intervention, regardless of standardisation of treatment protocol, is subject to great variation in its delivery, dependent upon interpretation and execution of the protocol by each operator. To add to the levels of complexity and variation, surgical protocols are often multi-step, sequential procedures, each prospective step dependent upon the previous for its efficacy. Even characterizing and accurately recording any variations in a putatively standardized protocol, therefore, becomes a challenge, since many aspects and steps must be recorded. This leads to the next challenge, that of demonstrating compliance with the accuracy of such complex data gathering. Furthermore, it becomes important from an analytical perspective to not only consider the effect of individual steps (factors) alone but also the effect of any interaction between the multiple steps inherent in the procedure. Although these issues could theoretically be controlled in well-designed and executed randomized controlled trials (RCTs), the potential bias related to recruitment and drop-out is inevitable, in particular within small scale trials. The use of strict participant selection criteria may limit the generalization of findings. In addition, the effects of pre-operative factors and some treatment steps cannot be randomized and can only be investigated in observational studies. Well-designed RCTs and population-based observational studies can therefore serve as complementary forms of research to ensure that the results of clinical trials translate into tangible benefits in the general population. Even when such comprehensive observational prospective outcome data is available, its utilization 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 extreme; (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 the effect of variations within the system. At present, the evidence-base is insufficient to allow such a sophisticated approach.

Value for Future Development of Treatment Protocols

Evaluation of outcomes and the factors that affect them are fundamental to the creation of a suitable data foundation for future application to endodontic prognostication. Endodontic treatment options compete with alternative dental therapies and, consequently, the survival of the discipline demands a suitable blend of efficacy, efficiency, utility, predictability, and cost-effectiveness of its principal procedures. Evidence-based practice, in turn, demands relevant data to support these outcomes. Achievement of this goal will require diligence and conscientious application by multiple centres of teams. Analyses of such powerful datasets will ultimately yield proper insight into the procedures used. Coupled with an evolving biological insight of the problem, it will become possible to derive new therapeutic solutions from first principles, as well as the technical and clinical skills necessary to deliver them through a broad spectrum of clinicians.

OUTCOME MEASURES FOR ENDODONTIC TREATMENT

Vital Pulp Therapy Outcome Measures

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 two-step (stepwise) caries excavation; (2) direct pulp capping of exposed pulps; or (3) partial/full pulpotomy procedures for more extensively involved pulps. For more details, see Chapter 27.

The surrogate outcome measures adopted in studies have included: (1) clinical success (positive response to pulp tests; and absence of pain, soft-tissue swelling, sinus tract, periradicular radiolucency, or pathologic root resorption); (2) patient satisfaction; and/or (3) adverse events (pain, swelling, tooth fracture); or (4) tooth extraction. Although not providing a specific follow-up strategy, the 2006 European Society of Endodontology quality guidelines suggested "initial review at no longer than 6 months and thereafter at further regular intervals." They did, however, provide the criteria for judging the favorable outcome of vital pulp therapy: (1) normal response to pulp sensitivity tests (when feasible); (2) absence of pain and other symptoms; (3) radiological evidence of dentinal bridge formation; (4) radiological evidence of continued root formation in immature teeth; (5) absence of clinical and radiographic signs of internal root resorption and apical periodontitis. Guidelines on pulp therapy for primary and immature permanent teeth provided by the American Academy of Paediatric Dentistry in 2009 suggested the following criteria: (1) tooth vitality maintained; (2) absence of

post-treatment 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; and (5) teeth with immature roots should show continued root development and apexogenesis. There are substantial differences in the criteria adopted in published clinical studies on outcomes of vital pulp therapy with few studies adopting all of the criteria. The duration and frequency of radiological review varies substantially from first review at one month followed by three-monthly reviews to an initial assessment at 6–12 weeks, followed by six and twelve-monthly reviews. The latter is more widely accepted because of the relative benefit of information yield *against* unnecessary radiation to patients from early and short interval radiological reviews. Follow-up over 10 years suggests declining success rates inferring probable slow spread of pulpal inflammation and latent disease in some cases, presumably because it is difficult to accurately judge the initial state of the pulp. The conclusion is that longer follow-up periods are merited.

Nonsurgical Root Canal (Re)Treatment Outcome Measures

Root canal treatment may be employed either to prevent or resolve periapical disease. Given that periapical lesions result from *interaction between* microbiota (and their products) and the host defenses, their prevention or resolution depends upon preventing or terminating such *interaction*.

Prior absence of apical periodontitis implies (in most instances) an absence of apical microbial colonization coupled with vital, healthy pulp tissue in the apical part of the root. The purpose of chemo-mechanical debridement would then be to extirpate the pulp (necrotic, inflamed or healthy) to prevent apical infection; thus, asepsis is the prime requirement. The precise technical details of the protocol may not matter as long as it is effective in aseptic removal of the pulp tissue. This is validated by the high chance of *retaining periapical health* (90%-99% judged by conventional radiography), regardless of the clinical protocol used.[5]

In the case of teeth with established periapical lesions, the challenge is different; the prime purpose in such cases would be to remove the established microbial biofilm to switch off the periapical host response. The challenge is greater still when the periapical lesion is larger as it is associated with a more diverse infection,[6] that is more difficult to eradicate.[7] A number of approaches (protocols) have been used to achieve this general aim.

The healing process after root canal treatment has not been deeply researched. Nevertheless, ideal healing should eventually result in regeneration and formation of cementum over the apical termini, isolating the root canal system from the periapex but this is not an inevitable end-result. Incomplete removal of the apical infection would reduce but not eliminate the inflammatory area and in fact this is generally what does happen.[8] This implies that residual infection in the apical area of the canal is the norm following completion of root canal treatment with ongoing microbial-host interaction beyond the end of treatment, influenced to varying degrees by the prior root canal treatment procedure and now resident root filling material.[9]

The culture test has been used during root canal treatment as an interim measure of the efficacy of the chemo-mechanical procedure, however, it has fallen out of favor in contemporary practice for a variety of reasons. The *immediate* outcome measures at the end of the root canal treatment procedure are the technical quality of root-filling and absence of clinical signs and symptoms of persistent periapical disease.[10,11] The *definitive* outcome measure (in conjunction with absence of signs and symptoms), however, is periapical healing, since the treatment is aimed at resolution of periapical disease.[12] Clinical judgment of periapical healing 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 and radiographic demonstration of reduction in the size of the periapical lesion (if sufficient time has lapsed), with a completely normal periodontal ligament space as the end-point. Although the majority of periapical lesions show radiographic evidence of healing within one year,[13,14] healing can actually take anything up to four years or longer.[15] On the other hand, the longitudinal follow-up[16] of 14 cases presenting with widened apical periodontal ligament for 10 years revealed unfavorable future healing in a small proportion of the cases (28%, 4/14).

Absence of signs and symptoms of periapical disease with persistence of radiographic evidence of periapical radiolucency may indicate either fibrous repair or persistent chronic inflammation. Only time or acute exacerbation would identify the latter, whereas the former should remain asymptomatic. Although some patients do anecdotally report discomfort in cold, damp weather, that is consistent with discomfort from scars in other parts of the body, commonly joints.

Longevity measures include survival of the root canal fillings or treatment and tooth retention or survival. The term *functional retention* was coined by Friedman and Mor[17] 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 absence of specific signs and symptoms of infection or inflammation, they find it impossible to use the tooth because it "feels" weak or vulnerable.

Modern definitions of health embrace a broader spectrum of measures including psychological well-being. 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. There is no definitive instrument for measuring these aspects

in endodontics as yet. Studies published so far have mainly adapted versions of Oral Health Impact Profile (OHIP).

The periapical status of root-treated teeth has traditionally been assessed using two-dimensional conventional radiographic imaging. Such imaging suffers from low sensitivity, particularly in the posterior parts of the mouth due to anatomical superimposition. Digital imaging technology enables image manipulation, including digital subtraction, densitometric analysis, correction of grey values and the manipulation of brightness and contrast but these do not address the main deficiencies. For more details, see Chapter 9B.

A three-dimensional imaging technique, cone-beam computed tomography (CBCT), requiring only 8% of the effective dose of conventional computed tomography, has been proposed as a means of overcoming the problem of superimposition of tissue layers and structures. For more details, see Chapters 9C and 10. CBCT has been found to be significantly more accurate in detecting small bone lesions in an animal model. Other evidence to support the superior sensitivity of CBCT is available from studies using a variety (size, shape) of simulated defects created in bone as gold standards by chemical or mechanical means in pig or human jaws. Although the validity of clinical outcome data derived using conventional radiographic technique has been questioned, the *routine use* of CBCT is not recommendedowing to its higher radiation dosage (×2-3). The use of CBCT that may be more sensitive for detecting periapical healing may give lower healing rates and longer durations to complete healing.

Many studies adopt a threshold for judging successful periapical healing that is passed only when *both* radiographic and clinical criteria are satisfied. Comparison of success rates estimated with or without clinical examination revealed no[18] or only a very small difference (1%),[13] although crucially, a small proportion of cases present with persistent symptoms despite complete radiographic resolution of the periapical lesion.[19]

The definition for success/failure by Strindberg[15] embracing both radiographic and clinical findings have been widely adopted or adapted. Success was defined by absence of symptoms and radiographic evidence of normal periodontal ligament structure, accepting some widening around excess filling material. Failure was conversely defined by presence of symptoms and radiographic evidence of decrease, stasis, increase in the periradicular rarefaction; or appearance of a new rarefaction. Friedman & Mor,[17] preferred the terms "healed," "healing," and "diseased" instead of "success/failure" because of the potential of the latter to confuse patients. The "healed" category corresponds to "success" as defined by Strindberg,[15] whilst "healing" corresponds to "success" as defined by Bender et al.[10,11]

The long duration of the periapical healing process coupled with the reduced recall rates at longer follow-up periods has prompted setting of the thresholds for success at partial healing (reduced lesion size) rather than complete healing. Setting the threshold of success at "*reduced periapical radiolucency size*" has been described as either a "*loose*" or a "*lenient*" measure[17]; whilst, setting the threshold at "complete resolution" has been described either as a "*strict*"[12] or "*stringent*" measure.[17] 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 difference varies from 4% to 48%[5,12].

A five-point periapical index (PAI) for measuring the periapical status has been used in some studies.[20,21] The studies though only reported the increase or decrease in mean scores for the clinical factors under investigation coupled with the proportion of successful cases. This approach unfortunately precludes direct comparison of their data with studies reporting conventional dichotomised outcomes. Other studies using the index dichotomized the scores into "healthy" (PAI 1 or 2) or "diseased" (PAI 3–5) categories[22] to overcome this problem. This system of designation is debatable, given that the PAI score 2 represents periodontal ligament widening or in fact a persistent lesion, albeit incipient.

Periapical Surgery Outcome Measures

Non-surgical root canal treatment alone may fail to resolve apical periodontitis in a small proportion of cases because of lack of access to the infection or non-microbial etiology. Microbial persistence may be localized intra-radicularly to the apical canal anatomy or the apical dentinal tubules; or extra-radicularly on the root surface or in the periapical lesion. In these instances, a surgical approach to the periapex may be required, *in addition* to the non-surgical approach.

The outcome of root-end management *via* a periapical surgical approach has been assessed by clinical and radiographic criteria as for non-surgical root canal treatment. However, the radiographic criteria for successful periapical healing are different from those for non-surgical root canal treatment.[23,24] Complete healing was defined by: (1) reformation of a periodontal ligament space with normal width & lamina dura around the apex, or a slight increase in the width of the apical periodontal space (less than twice the width of non-involved parts of the root), or a tiny defect in the lamina dura (maximum 1 mm^2) adjacent to the root filling; or (2) complete bone repair with the deficiency that either the bone bordering the apical area lacks the density of the surrounding non-involved bone, or that there is an absence of apical periodontal space. Incomplete healing (scar tissue) was defined by: (1) a decreased or static rarefaction; or (2) an isolated scar tissue in the bone. Either of these conditions may be found with: (1) bone structure within the rarefaction; (2) a well demarcated but irregular periphery to the rarefaction; (3) asymmetrical distribution of the rarefaction around the apex; or (4) angular connection between the rarefaction and the periodontal ligament space. Periodontal

TABLE 33-1 Studies Investigating the Clinical/Radiographic Outcome of Indirect Pulp Capping of Permanent Teeth

Study	Study Design	Type of indirect pulp capping	No. of teeth	Success (%)	Age (years)	Pre-treatment status	Capping material	Criteria for success	Duration after treatment	Notes
More 1967	Case series	Stepwise	8	100	11 to 18	Carious Vital Apical pathosis	1st visit: ZOE. 2nd visit: CH/Amal	Vital/ Asymptomatic/ Intact PDL	6 to 36 m	
Jordan et al. 1971 (in Hayashi et al. 2011) & 1978	Case series	1-step	243	97.1	8 to 37	Deep caries without pulpitis	CH, CH+crestatin, or ZOE / ZOE+amalgam	Vital/ Asymptomatic/ Intact PDL	Not given	24 teeth were followed up for 11 y, 11 remained vital with no evidence of pathosis.
Sawusch 1982 (in Hayashi et al. 2011)	CCT	Stepwise	48	100	14 or younger	Deep caries without pulpitis	CH (Dycal vs improved Dycal)/ ZOE or ZnPO4	Clinical	6 m	
Fitzgerald & Heys 1991	Case series	1-step	46	84.8	20 to 60	Deep caries Vital	CH (Dycal vs Life)	Asymptomatic	1 y	The brand of CH did not have significant effect.
Nagamine 1993 (in Hayashi et al. 2011)	CCT	Stepwise	23	91.3	17 to 46	Deep caries without pulpitis	Polycarboxylate cement with tannin-fluoride vs hydraulic temporary sealing material /GIC	Vital	3 m	
Leksell 1996	RCT	Stepwise	57	82.5	6 to 16	Not given	CH/ZOE then CH/ GIC	Asymptomatic/ intact PDL	1 to 11 y (mean = 43 m)	47/57 without exposure; duration of CH dressing had no significant influence on exposure.
		1-step	70	60.0						42/70 without exposure
Bjorndal & Thylstrup 1998	Case series	Stepwise	94	92.6	Not given	Deep caries	CH/temporary filling	Absence of perforation, asymptomatic	1 y	88/94 without exposure
Bjorndal et al. 2010	RCT	Stepwise	143	74.1	29 (25-38)	deep caries Only provoked pain Vital	CH/GIC	Vital/intact PDL	1 y	Stepwise better than complete. Pre-operative pain, older patients reduce success
		1-step	149	62.4						

TABLE 33-1 Studies Investigating the Clinical/Radiographic Outcome of Indirect Pulp Capping of Permanent Teeth (*Continued*)

Study	Study Design	Type of indirect pulp capping	No. of teeth	Success (%)	Age (years)	Pre-treatment status	Capping material	Criteria for success	Duration after treatment	Notes
Gruythuysen et al. 2010	Case series	1-step	34	97.1	upto 18	Carious (>2/3) No spontaneous, persistent pain No apical pathosis	GIC	Survive/ asymptomatic/ intact PDL/no resorption	3 y	
Maltz et al. 2011	Case series	1-step	26	61.5		Vital Deep caries	CH/ZOE then composite	Vital	10 y	
Maltz et al. 2012a&b	RCT (multi-centre)	1-step	112	99.1 (18 m) 91.7 (3 y)	≥ 6	Vital Carious (>1/2) No spontaneous, persistent pain No apical pathosis	GIC/amalgam or composite	Vital/intact PDL Vital/intact PDL	18 m, 3 y (clinical & radio-graphic outcome)	Indirect pulp capping was associated with significantly higher success rate than Stepwise, regardless of duration after treatment.
		Stepwise	101	86.1 (18 m) 69.3 (3 y)			CH/ZOE then GIC/amalgam or composite			The low success rate of stepwise was attributed to patients not returning for completion of treatment.

TABLE 33-2 Studies Investigating the Clinical/Radiographic Outcome of Direct Pulp Capping of Permanent Teeth

Study	Study Design	No. of teeth	Success (%)	Age (years)	Pre-treatment status	Hemostasis	Capping material	Base/ Restorative material	Criteria for success	Duration after treatment	Notes
Weiss 1966	Case series	160	88.0	16 to 67	Not given	Not given	CH+Cresatin	ZOE	Vital/ Asymptomatic/ intact PDL	3 y	
Shovelton et al. 1971	RCT	154 (1-step)	74.7	15 to 44	Carious or traumatic exposure Vital Asymptomatic Molar/premolar	Saline	Corticosteriod + antibiotic, Glycerrhetinic acid + antibiotics, ZOE, or CH	ZOE/amalgam	Vital/ Asymptomatic/ Intact PDL	24 m	There was no significant difference in success rates amongst the various capping materials
		53 (2-step)	64.7								
Haskell et al. 1978	Case series	133	88.0	8 to 74	Carious exposure Asymptomatic	Not given	CH or penicillin	ZOE	Vital/ Asymptomatic/ Intact PDL	> 5 y	5 to 22 year survival; Success rate was not affected by age and tooth type
Gillien & Schuman 1985	Case series	17	76.4	6 to 9	Carious exposure	Not given	CH	Base material notgiven amalgam orcrown	Asymptomatic/ Intact PDL	6 to 12 m	
Horsted et al. 1985	Case series	510	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	5 y	Type of exposure and tooth type had no significant effects. Older age had lower survival.
Fitzgerald & Heys 1991	Case series	8	75.0	20 to 60	Vital Exposure during caries removal	Sterile cotton pellets	CH	$ZnPO_4$/ amalgam or composite	Asymptomatic	12 m	The brand of setting CH paste did not have significant effect.
Matsuo et al. 1996	Case series	44	81.2	20 to 69	Carious exposure No intense pain	10% NaOCl & 3% H_2O_2	CH	ZOE/GIC	Vital/ Asymptomatic/ Intact PDL	3 y	
Santucci 1999	Case series	29	51.7	Not given	Exposure due to caries, or its removal Sensitive to cold or sweatwith no other pain No periapical pathosis	Not given	CH	Composite or castgold restoration	Asymptomatic	4.5 y	
Barthel et al. 2000	Case series	123	23.6	10 to 70	Carious exposure	3% H_2O_2	CH/$ZnPO_4$ or other materials	Not given	Vital/ Asymptomatic/ intact PDL	5 to 10 y	Age, tooth type, site of exposure had no significant effects; Immediate placement of permanent restoration had significantly higher success rate.

TABLE 33-2 Studies Investigating the Clinical/Radiographic Outcome of Direct Pulp Capping of Permanent Teeth (*Continued*)

Study	Study Design	No. of teeth	Success (%)	Age (years)	Pre-treatment status	Hemostasis	Capping material	Base/ Restorative material	Criteria for success	Duration after treatment	Notes
Farsi et al. 2006	Case series	30	93.3	9 to 12	Deep caries Potential exposure Reversible pulpitis Intact PDL	Saline	MTA	ZOE/composite	Vital/ Asymptomatic/ intact PDL/continuous root development	2 y	
Bogen et al. 2008	Case series	49	98.0	7 to 45	Carious exposure (.25 to 2.5 mm) Reversible pulpitis	5.25/6% NaOCl	MTA	Composite	Dentine Vital/ bridge/asymptomatic/root development / intact PDL	1 to 9 y	
Bjorndal et al. 2010	RCT	22	31.8	25 to 38	Exposure during caries removal; Only provoked pain Vital	Saline	CH	GIC	Vital/intact PDL	1 y	
Mente et al. 2010	Case series	122	70.5	8 to 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 to 18 m	Use of MTA for capping and immediate placement of permanent restoration had significantly higher success rate. Age; sex; tooth location & type; exposure site & type; restoration type, size and quality did not have significant effects.
Miles et al. 2010	Case series	51	45.1	21 to 85	Carious exposure	2.5% NaOCl	MTA	GIC / composite or amalgam	Vital/ Asymptomatic/ intact PDL	12 to 27 m	
Hilton et al. 2013	RCT	126	64.3	9 to 90	Carious, traumatic, mechanical exposure	5.25% NaOCl	CH	GIC	Vital/intact PDL/ no resorption/ not requiring extraction or root canal treatment	2 y	MTA was associated with significantly higher success rate than CH. Patient, dentist, tooth, pulp exposure, & pulp capping characteristics did not have significant influence on the results.
		144	80.6	8 to 89			MTA				

TABLE 33-3 Studies Investigating the Clinical/Radiographic Outcome of Partial Pulpotomy of Permanent Teeth

Author	No. of teeth	Success (%)	Age (years)	Pre-treatment status	Exposure size	Hemostasis	Pulp dressing material	Base/ Restorative material	Criteria for success	Duration after treatment	Notes
Cvek 1978	60	96.7	Not given	Traumatic exposure Vital Bleeding wound	0.5 to 4.0 mm	Saline	1st visit: non-setting CH 2nd visit: setting CH	1st visit: ZOE 2nd visit: Composite	EPT/Asymptomatic/Intact PDL/continued root development/hard tissue barrier	14 to 60 m	Size and duration of exposure, root maturity did not affect outcome.
Baratieri et al. 1989	26	100	12 to 44	Exposure due to caries orcaries removal Pulp tissue bleeding withoutsigns of degeneration	Not given	Calcium hydroxide solution	CH powder then hard set CH cement	Zinc oxide cement	Asymptomatic/Vital	1 to 2 y	
Fuks et al. 1993	44	79.5	Not given	Traumatic exposure Vital Bleeding wound	Not given	Saline	CH	ZOE	Asymptomatic/bridge/ root development/vital	0.5 to 4 y	
Mass & Zilberman 1993	35	91.4	7.5 to 25	Molar Deep caries Asymptomatic No apical pathosis	Less than 1 to 2 mm in diameter; 2 to 3 mm deep	Saline	CH	ZOE/amalgam or crown	Asymptomatic/Intact PDL/root development	1 to 2 y	
Mejare & Cvek 1993	31 (2-step) 6 (1-step)	93.5 66.7	Not given	Carious exposure Asymptomatic No apical pathosis	N/A	Saline	CH	ZOE	Asymptomatic/Intact PDL/root development	24 to 140 m	
Barrieshi-Nusair & Qudeimat 2006	28	75.0	7.2 to 13.1	Carious exposure Molar Reversible pulpitis No apical pathosis	2 to 4 mm deep	Saline	MTA	GIC/amalgam or crown	Vital/Asymptomatic/ intact PCL/root development	1 to 2 y	
Qudeimat et al. 2007*	23	91.3	6.8 to 13.3	Carious exposure Molar	2 to 4 mm deep		CH	GIC/Amalgam or crown	Absence of signs and symptoms/intact PDL/ root development	24.5 to 45.6 m	There was no significant difference in outcome between CH and MTA
	28	92.9					MTA				
Bjorndal et al. 2010*	29	34.5	25 to 38	Deep caries Only provoked pain Vital Expose during removal	Not given	Saline	CH	GIC	Vital/intact PDL	1 y	

NB: All the studies were case series except those labelled 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 33-4 Studies Investigating the Clinical/Radiographic Outcome of Full Pulpotomy of Permanent Teeth

Author	No. of teeth	Success (%)	Age (years)	Pre-treatment status	Hemostasis	Capping material	Base/ Restorative material	Criteria for success	Duration after treatment	Notes
Masterson 1966	30	83.3	6 to 39	Not given	Not given	CH	Not given	Vital/Asymptomatic	1 to 70 m	
Russo et al. 1982	30	93.3	9 to 28	Carious exposure No apical pathosis	Not given	CH	Not given	Intact PDL	8 w	
Santini 1983	373	51.4	Not given	Carious exposure or near exposure Symptomatic	Cotton wool pellet	CH or CH+Ledermix	ZOE	Vital/bridge/ asymptomatic	6 m	Sex & medicament had no significant effect; Poor healing was associated with age<7.5
Caliskan 1993	24	91.7	10 to 22	Hyperplastic pulpitis	Saline	CH	ZOE/Amalgam or composite	Vital/Asymptomatic/ dentine bridge/ Intact PDL	1 to 4 y	
Caliskan 1995	26	92.3	10 to 24	Carious exposure Asymptomatic Apical pathosis	Saline	CH	ZOE	Asymptomatic/ bridge/root development/vital	16 to 72 m	
Waly 1995	20	90.0		Carious exposure Molar		CH-glutaraldehyde CH	Not given	Not given	5 y	
Teizeira et al. 2001	41	82.9	6 to 16	Deep caries or exposed pulp With or without apical pathosis	Not given	CH	GIC	Vital/asymptomatic/ dentine bridge/ intact PDL	24 to 32 w	
DeRosa 2006	26	65.4		Not given		CH	Amalgam	Asymptomatic	14 to 88 m	
El Meligy & Avery 2006	15	86.7	6 to 12	Carious or traumatisedteeth Immature apex No apical pathosis Saline		CH	ZOE/Amalgam or composite	Asymptomatic/intact PDL/no resorption/ root development	1 y	
	15	100				MTA				
Witherspoon et al. 2006	19	79.0	7 to 16	Carious or traumatic exposure Irreversible pulpitis	6% NaOCl	MTA	Not given	Vital/Asymptomatic/ intact PDL/root development	1 y	
Asgary & Ehsani 2009	12	100	14 to 62	Carious Irreversible pulpitis	Saline	NEC	Permanent rest	Asymptomatic/Intact PDL	13 to 20 m	

NB: All the studies were case series except El Meligy & Avery (2006), which is a randomized, controlled trial comparing calcium hydroxide and MTA as pulp dressing material.

attachment loss, in the form of marginal gingival recession, is an additional consideration for measuring the outcome of periapical surgery.

OUTCOME DATA FOR 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 Step-wise Excavation)

The most conservative management for extensive caries is indirect pulp capping using either the one-step or the two-step (step-wise) excavation approach. The step-wise excavation approach with initial partial caries removal was advocated to reduce the risk of pulpal exposure; the latter putatively being associated with poorer outcomes. The clinical outcomes of the "one-step" or "step-wise" approaches have been compared in three randomized controlled trials on permanent teeth with deep caries.[25-28] Step-wise excavation gave a lower rate of pulp exposure coupled with higher chance of long-term clinical success than the one-step excavation approach[25,26] (Table 33-1). Maltz et al.[27,28] however, found the contrary; they attributed the low long-term clinical success rate of the step-wise approach to patients not returning for scheduled completion of treatment. Interestingly, comparison of the cost-effectiveness of the "one-step" versus the "step-wise" excavation techniques, using health-economic modeling involving simulated treatment of a molar tooth with deep caries in a 15-year-old patient, revealed the former procedure to accrue lower long-term costs as well as longer tooth retention and pulp vitality.[29]

Meta-analyses of data from studies listed in Table 33-1 gave a weighted pooled success rate of 81.7% for indirect pulp capping using the one-step approach (95% confidence interval [CI]: 72.7%, 90.6%) that was similar to the weighted pooled success rate for the step-wise approach (81.9%; 95% CI: 72.1%, 91.7%).

Calcium hydroxide cement was the preferred lining material for the pulpal surface, whilst 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 (Table 33-1). The age of patients, presence of pre-operative pain, and pulpal exposure during excavation proved to be significant prognostic factors.

Direct Pulp Capping

Direct pulp capping is performed on teeth with pulp exposures caused by caries, caries excavation, traumatic injuries, or root resorption. 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 33-2). Saline, sodium hypochlorite, and chlorhexidine have been reportedly used to irrigate the exposed pulp and to achieve hemostasis, whilst calcium hydroxide paste and mineral trioxide aggregate (MTA) were the commonly used capping materials (Table 33-2).

Meta-analysis of data from studies listed in Table 33-2 gave a weighted pooled success rate of 70.1% (95% CI: 59.9%, 80.2%). 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[30] revealed the former was associated with significantly more successful outcomes.

The type of capping material was another significant prognostic factor, with MTA performing superiorly to calcium hydroxide in a randomized controlled trial[31] and in a systematic review.[30]

Pulpotomy

Earlier studies on the outcome of *partial pulpotomy* (specifying varying definitions of pulpal extent) had only included teeth with vital traumatic pulp exposures but recent studies have also included teeth with carious exposures (Table 33-3). Teeth with spontaneous or severe pain as well as signs and symptoms of apical pathosis were excluded (Table 33-3). On the other hand, the outcomes of *full pulptomies* (removing the coronal pulp and retaining the radicular pulp) had only been investigated in teeth with carious exposures (Table 33-4). Saline irrigation has been preferred for achieving hemostasis, whilst calcium hydroxide or more recently MTA, were the preferred pulp capping materials (Table 33-3).

Meta-analysis of data from studies listed in Table 33-3 and Table 33-4 gave a weighted pooled success rate of 79.3% (95% CI: 66.7%, 91.8%) for partial pulpotomies and 82.4% (95% CI: 69.3%, 95.4%) for full pulpotomies.

The effects of potential prognostic factors for pulpotomy have not been explored systematically except for the pulp capping material. Randomized controlled trials revealed MTA achieved similar outcomes in partial[32] or full[33] pulpotomies when compared with calcium hydroxide.

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: pre-existing health of the pulp; adequate removal of infected hard or soft tissues; careful operative technique to avoid damage to residual tissues; and exclusion 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 pre-operative 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 is reliant upon the correct choice of restorative material and its adequate manipulation to prevent leakage.

Factors such as age and health of the patient, 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, appear to compromise outcomes of vital pulp therapy.

OUTCOMES OF NON-SURGICAL ROOT CANAL TREATMENT

In contrast to other areas of endodontics, the number of studies and extent of investigation of non-surgical root canal treatment is more comprehensive, yielding a much greater breadth and depth of insight, even though the quality and scope of the research does not always reach the highest levels. The following summary is therefore more elaborate than preceding sections.

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 (complete healing) measured by radiographic evidence of periapical health was 83% when vital pulpectomy was performed (Figure 33-1); it reduced to 72% when root canal treatment was performed to eradicate the established infection associated with a periapical lesion (Figure 33-2).

Factors Affecting Periapical Health or Healing Following Root Canal Treatment

The factors influencing the maintenance of periapical health or periapical healing of pre-existing lesions following root canal treatment may be broadly classified into *patient-related* factors (age, sex, general health, tooth anatomy, pre-operative pulpal and periapical status), *treatment-related* factors (operator characteristics, canal shaping/enlargement, irrigation, medication, culture test and obturation), and *restorative-related* factors. Some factors had a profound impact on success rates, whilst others showed a negligible effect. Patient-related factors characterizing the nature of the disease showed the most potent effect (periapical status), whilst most of the treatment-related factors, individually, were found to exert a weak effect; the exceptions were the apical extent of root canal treatment relative to the root canal terminus that showed a strong effect. In addition, the quality of the post-operative restorative care also exerted a profound influence on outcome of treatment.

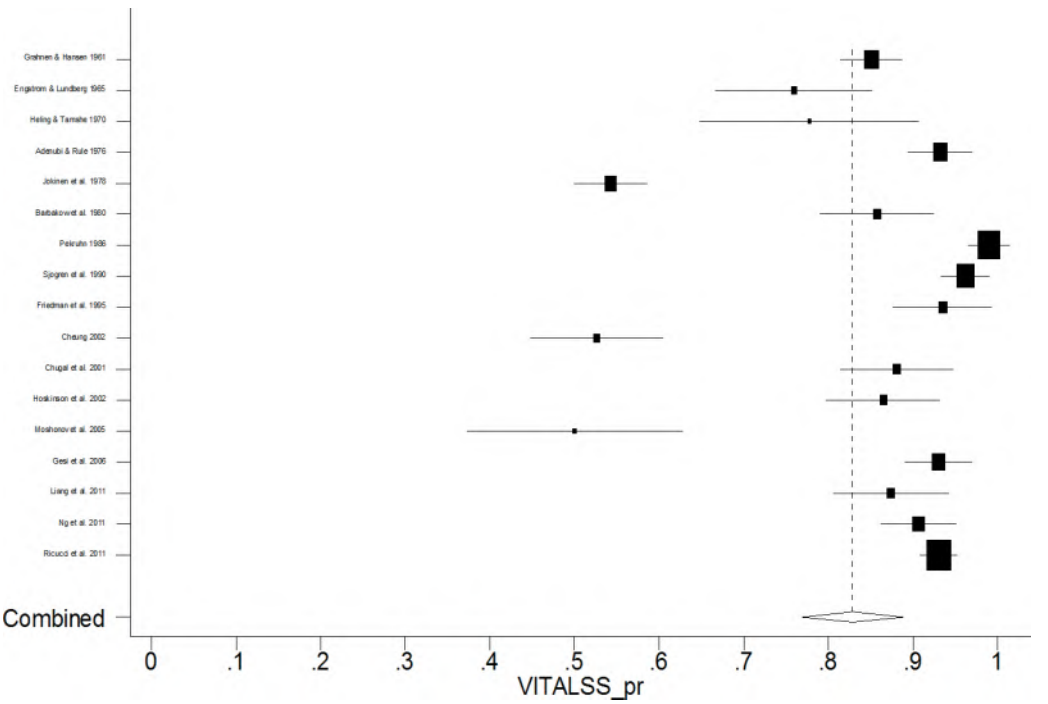


FIGURE 33-1 Forest plot showing results of pooled and individual study's probability of maintained periapical health for pre-operatively vital teeth undergoing root canal treatment. (Pooled probability = 0.83; 95% confidence interval: 0.77, 0.89.)

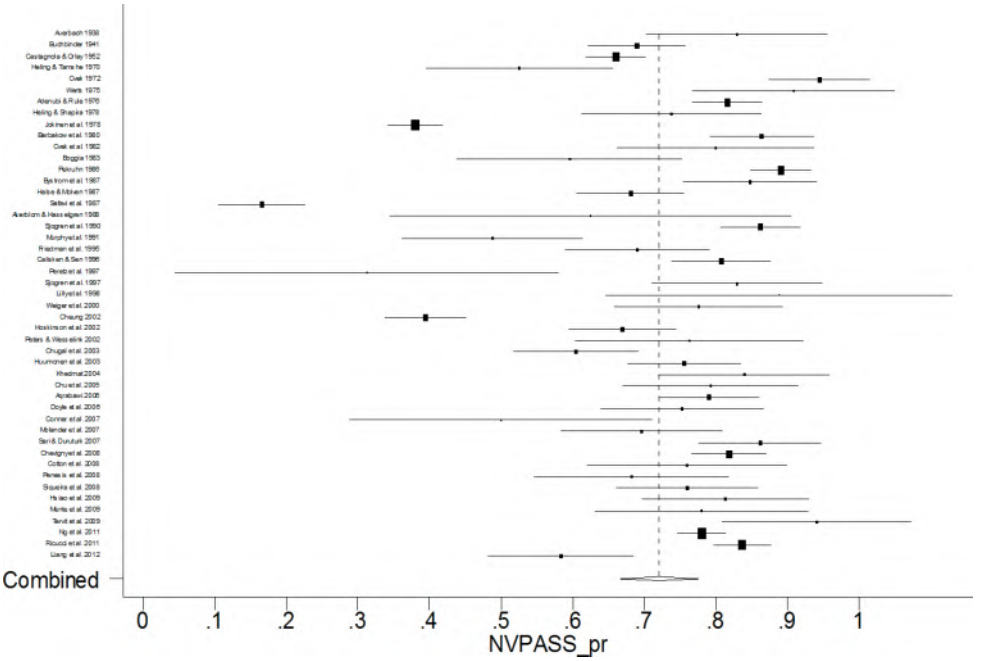


FIGURE 33-2 Forest plot showing results of pooled and individual study's probability of periapical health for teeth associated with non-vital pulps and periapical radiolucencies undergoing root canal treatment. (Pooled probability = 0.72; 95% confidence interval: 0.67, 0.78.)

Patient-related Factors

Patient's age and sex consistently had no significant influence on outcome, whilst some specific health conditions (diabetes, compromised innate immune response) 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. Recent emerging evidence suggests that the host response, characterized by polymorphisms of various genes involved in periapical healing, may have an effect on outcomes. 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.[13]

The perception that single-rooted teeth exhibit simpler canal anatomy and should accrue better and more predictable outcomes proved to be untrue. Having accounted for the potential confounding effect of periapical disease, it is found that tooth type did not exert a strong influence on success rates. This would appear to be counter-intuitive but may be explained by the logical inference that canal complexities in the apical anatomy probably play a more dominant role than canal system complexities more coronally.[14]

The most dominant factor influencing the success of root canal treatment negatively is the presence (Figure 33-3) and size of a periapical lesion (Pooled OR comparing pre-operative large [> 5mm] versus small [< 5 mm] periapical radiolucencies = 2.2; 95% CI: 1.3, 3.7)[5,13]; 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 telling, since it is correlated with the establishment of infection in the apical canal anatomy. This suggests 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 ready and acceptable biological explanation, in that the diversity of microorganisms (by number of species and their relative abundance) is greater in teeth with larger periapical lesions.[6] The infection was more likely to persist in those canals with a higher number of microorganisms pre-operatively.[7] 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[34] where mechanical and chemical decontamination procedures may not readily reach. Larger lesions may also represent cystic transformation.[35] Finally, the host response may also play a part as patients with larger lesions probably respond less favorably to residual infection.[8]

Most of the other investigated pre-operative factors (pain, tooth tenderness to percussion, soft tissue tenderness to palpation, swellings, sinus tracts, periodontal probing defect of endodontic origin, root resorption) are in fact different clinical manifestations of periapical disease. 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 pre-operative pain, sinus tract, swelling and apical resorption have been found to be prognostic factors significantly reducing the success of root canal treatment.

The negative impact of sinus tract and swelling, either in the acute or chronic form, on periapical healing would

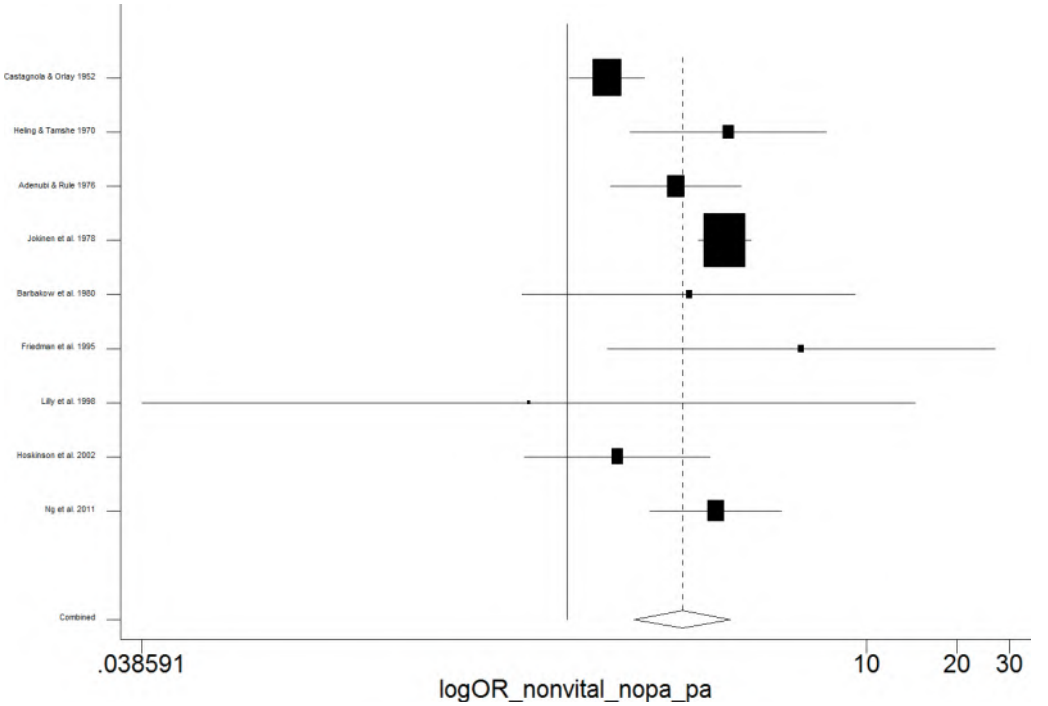


FIGURE 33-3 Forest plot showing pooled and individual study's odds ratio (OR) for periapical health of teeth undergoing root canal treatment with pre-operative non-vital pulps and absence of periapical radiolucencies versus teeth with non-vital pulps and presence of periapical radiolucencies. (Pooled OR = 2.4; 95% CI: 1.7, 3.5.)

seem to be related to suppuration and proliferation of microbiota into the periapical tissues; that is, the host tissues being overwhelmed by the microbiota. The precise mechanisms for reduced success rates remain unclear but must reflect the nature of host-microbial interaction. Clinically, however, endodontically-related sinus tracts (intra-oral and extra-oral) can demonstrate dramatic healing following adequate endodontic treatment.

Treatment-Related Factors

The impact of operator qualification and skill has not been specifically investigated but systematic review demonstrates the involvement of clinicians with different educational and training backgrounds (undergraduate students, general dental practitioners, postgraduate students and specialists).[12] This has allowed partitioning of outcome data to reveal clear trends with superior outcomes associated with greater experience and training. Technical skills would appear to play an important role in the outcome. However, the lack of appropriate tools or methodology to objectively quantify operator skills precludes segregation of the complex constellation of cognitive, technical, and clinical skills. The role of technical refinement must be balanced against the overall understanding of the biological problem by the operator, and crucially, the motivation and integrity with which the procedure is performed.

The use of dental dam in modern root canal treatment is so widely accepted that the absence of systematic data on its influence on root canal treatment outcome is a surprise. One study on retreatment[36] had analyzed the influence of dental dam compared to cotton roll isolation and found significantly higher success rates with the former approach. Another observational study reported a significantly higher success rate of root canal-treated teeth,[37] when dental dam was employed during post placement, compared to when it was not. The principal justification for dental dam use is based on patient safety and the medico-legal implications of root canal instrument inhalation by the patient.

The virtues of magnification and illumination during root canal treatment are universally extolled by endodontists but a systematic review failed to locate objective evidence for the view.[38] A prospective study[13] investigating this factor found an insignificant influence on the outcome. Use of a microscope may sometimes assist location of the second mesio-buccal canal in maxillary molars (64%), but this only made a small difference to the success rates associated with mesio-buccal roots, when a periapical lesion was present.[13] The true benefit of a microscope can only be verified through a randomized controlled trial but current evidence precludes a significant impact on the critical step of apical infection control.

Mechanical preparation

A variety of instruments of different cutting designs, tips, tapers, and materials of construction may be employed to mechanically prepare the root canal system to a defined size and taper. Numerous laboratory studies testify to their properties, efficacy, and utility but investigation of their influence in clinical canal

enlargement appears only in two non-randomized prospective studies.[13,39] In the former study,[13] the better success rates for NiTi instruments (hand or rotary) compared with stainless steel instruments were 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 trainees were more likely to have already established a better understanding of the biological 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, whilst in selected cases, NiTi instruments appear capable of achieving the same in primary root canal treatment undertaken by undergraduates.[40] The importance of biological insight over technical skill development alone in the performance of root canal treatment appears to be reinforced by the observation that improvements in the technical quality of root fillings through training in NiTi instrumentation and single-cone root filling did not accrue a parallel improvement in periapical status.[39]

A key tenet of the European Society of Endodontology (ESE) guidelines is that root canal debridement must be extended to the terminus of the canal system; that is expressed variously as extension to the "apical constriction," or to "0.5 to 2 mm from the radiographic apex,", or to the "cemento-dentinal 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.[13,15,41] Ng et al.[13] reported a two-fold reduction in periapical healing when 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 dentin/organic debris.

The debate on the optimal size of apical canal preparation remains topical in the absence of definitive evidence; the findings from relevant *in vitro* and clinical studies have been reviewed before.[42] So far, six clinical outcome studies have considered the issue or have systematically investigated the effect of apical size of canal preparation on treatment outcome.[13,15,18,43-45] One randomized controlled trial revealed that enlargement of the canal to three sizes larger than the first apical binding file was adequate,[45] the mean final size was ISO 30. Observational studies,[13,15,18] although not designed to investigate the effect of apical canal size as their principal focus, did not find a statistically significant influence attributable to 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 might compromise treatment success by generation of more apical dentine debris, that in the absence of an adequate irrigation regimen may serve to block infected apical canal exits. Continued generation of dentin debris, in the absence of sufficient irrigation may lead to what is termed "dentin mud," that may ultimately create a blockage. The impatient or neophyte dentist/endodontist may fail 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 hypothesized that immature roots present a different debridement challenge, where the canal shape is not amenable to cleaning 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 the above studies, therefore, do not concur with views that more effective microbial elimination may be achieved with larger apical preparations.[46-48]

The issue of apical preparation size should be considered alongside that of the 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 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 on this factor.[13,18,49] Smith et al.[49] 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 confounders were not controlled. In contrast, Hoskinson et al.[18] and Ng et al.[13] using strict criteria did not find any significant difference in treatment outcome between narrow (.05) and wide (.10) canal tapers. The controlled use of stainless steel instruments in a step-back technique may create .05 (1 mm step back) or .10 (0.5 mm step back) tapers, although, of course, uncontrolled use of such instruments may generate a variety of shapes. Ng et al.[13] also compared these (.05 and .10) preparation tapers with .02, .04, .06, and .08, tapers (generally achieved by using non-ISO, greater taper, nickel-titanium 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 confounded by the initial size of canal, type of instrument, and operator experience.

Triangulation of the available data on the effects of canal preparation taper on treatment outcome intuitively suggests that, according to current best evidence, it is not necessary to over-enlarge the canal to achieve periapical healing. An apical preparation size of ISO 30 with a .05 taper for stainless steel instrumentation or .06 taper for NiTi instrumentation may be sufficient for many canals, subject to adequate irrigation. Precisely what biological and hydrodynamic mechanisms underpin such sufficiency is more difficult to define based on available evidence. Although a number of laboratory studies[50] have investigated the interaction between canal dimensions and irrigation or obturation dynamics, the precise physical, chemical, or biological mechanisms that ultimately enable periapical healing remain unknown, although

collaborations with fluid dynamics specialists[50] and (micro) biologists[51] 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. Of these, the effects of changes in canal shape (ledge formation, apical zipping and transportation) have not been specifically investigated and reported, whilst the effect of canal blockage was explored previously.

Iatrogenic perforations during treatment were found to result in significantly lower chance of periapical healing.[13,41,52-55] These studies did not analyze further the specific prognostic factors for teeth with perforation, such as time lapse before defect repair, location and size of perforation, and adequacy of perforation seal. An observational study[56] that had specifically evaluated the outcome of MTA® repair of root perforations revealed substantially lower success rates amongst cases with perforation of larger size or those present in the furcation region. The use of MTA® as perforation repair material may improve the treatment outcome of these cases, attributable to its biocompatible and effective seal.

Instrument separation during treatment has been found to reduce the success rate significantly,[13,15] however, the prevalence of instrument separation was low (0.5% - 0.9%) in these studies, precluding an analysis of causative factors. A case-control study[57] revealed a small but not statistically significant difference in success rates between periapically-involved teeth (with or without retained separated instruments) managed by endodontists. The stage of canal debridement at which instrument separation occurred may have implications on the outcome. The corono-apical location of a separated instrument and whether the instrument was successfully bypassed were found to have no effect on treatment outcome. The positive influence of separated instrument removal on treatment outcome was, however, found in cases managed by undergraduate students.[58]

Irrigation

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, local anaesthetic, sodium hypochlorite, iodine, chloramine, sulphuric acid, EDTA, hydrogen peroxide, organic acid, Savlon®, urea peroxide and Biosept® (a quaternary ammonium compound).[5] Most of the studies had used sodium hypochlorite as an irrigant,[5] regardless of whether it was primary treatment or retreatment. For more details, see Chapter 21.

A prospective study[13] systematically investigated the effect of the irrigant on the success rates of root canal retreatment. Although not a randomized controlled trial, it revealed interesting new findings on the effects of irrigants. Whilst a higher concentration of sodium hypochlorite (5% vs 2.5%) made negligible difference to treatment outcome, the additional use of other specific irrigants had a significant influence on success rates.[13] The use of higher concentration NaOCl solutions did not yield improved periapical healing, that is an observation consistent with previous clinical/microbiological findings.[59,60] Comparing 0.5% to 5.0% NaOCl solution for irrigation, it was found that concentration of solution, *per se*, did not appear to increase the proportion of teeth, either rendered culture-negative[59] or associated with periapical healing.[60] As iodine and sodium hypochlorite are both halogen-releasing agents and attack common key protein groups, 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.[13] This finding was in complete contrast to previous reports[61,62] 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 some years ago[63] and was justified on several grounds, including its substantivity in root dentine, relative lack of toxicity, and broad-spectrum efficacy. Not until recently has alternate irrigation with sodium hypochlorite and chlorhexidine solution raised serious concerns because of their interaction product, an insoluble precipitate containing para-chloro-aniline, that is cytotoxic and carcinogenic. Apart from mutually depleting the active moiety in the two solutions for microbial 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 et al.[13] also found that the additional use of EDTA had a profound effect on improving radiographically observed periapical healing associated with root canal treatment (OR = 1.6 [1.1, 2.1]). The observed synergistic effect of sodium hypochlorite and EDTA had been previously demonstrated in terms of bacterial load reduction[64] but not periapical healing. The long-term (≥ two years) outcome of their cases stratified by canal disinfection protocols of Sunqvist's group[65] did not support their microbiological findings. Given the complexity of their study design (clinical and microbiologic), their sample size was restricted to 11–15 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.[66] EDTA solution assists negotiation of narrow or sclerosed canals by demineralization of canal surface dentine and helps removal of compacted debris from non-instrumented canal anatomy. It may also facilitate deeper penetration of sodium hypochlorite solution into dentin by opening dentinal tubules and removing the smear layer from the instrumented surface, and lastly may help detach or breakup biofilms adhering to root canal walls.[67]

Although the beneficial effects of irrigant agitation (manually or automatically) on irrigant penetration and flushing action have been repeatedly demonstrated in *in-vitro* studies,

a recent randomized controlled trial[68] failed to confirm a significant influence of ultrasonic agitation of NaOCl irrigant on periapical healing. However, their sample size was small (n = 41-43 per group), and the duration of follow-up was less than two years post-treatment.

Medicament

Previous treatment outcome studies have used a variety of root canal medicaments in the inter-appointment period instead of standardizing this variable. These mainly included calcium hydroxide, creosote, and iodine solutions. However, there is an absence of studies investigating the influence of this factor alone 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*.

Root canal bacterial culture results prior to obturation

In the distant past, completion of root canal treatment by obturation would only be done after a negative culture test result, interpreted as confirmation of absence of bacteria in the root canal system. This practice has, unfortunately, fallen out of clinical favor because of the perceived predictability and good prognosis of root canal treatment without microbiological sampling. Sampling procedures are considered lengthy, difficult, inaccurate, requiring laboratory support, and having low benefit/cost ratio. However, a preobturation negative culture result can increase treatment success two-fold (Pooled OR = 2.1; 95% CI: 1.5, 2.9). One large study[69] in particular contributed to the demise of the culture test. Nevertheless, even their 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.

Numerous studies have evaluated the effect of different stages of root canal treatment on the intra-radicular microbiota, both qualitatively and quantitatively (Table 33-5). Some studies merely report positive culture tests, whereas others have identified and quantified intraradicular microbiota before and after various stages of treatment.

The effect of the steps up to and including 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 0–79%). When sodium hypochlorite (concentration range 0.5%-5.0%) irrigation supplemented the steps up to "mechanical preparation," the frequency of negative cultures immediately after debridement increased to a weighted pooled average of 52% (range 13%-95%) (Table 33-5).

Most studies report culture reversals during the inter-appointment period when active antimicrobial dressing is not used in the root canal system between appointments. The reversals may be due to regrowth of residual microorganisms or recontamination by microbial leakage around the access cavity dressing. When active inter-appointment antimicrobial intra-canal dressing is used, the frequency of negative cultures in the subsequent visit were achieved on average in 71% of cases (range 25%-100%) (Table 33-5).

Type of Persistent Microorganisms

Microorganisms in preobturation cultures have included: *Enterococcus*, *Streptococcus*, *Staphylococcus*, *Lactobacillus*, *Veillonella*, *Pseudomonas*, *Fusobacterium* species and yeasts.

TABLE 33-5 Summary of Studies Evaluating the Effect of Root Canal Treatment Procedures on Bacterial Detection by Culture

Study	Year	Sample Size	Percentage of samples with bacterial presence		
			At baseline	After preparation ± irrigation	Next visit (after dressing+/-)
Auerbach	1953	60 teeth	93%	**Chlorinated soda (double strength)**: 22%	-
Ingle & Zeldow	1958	89 teeth	73%	**H_2O**: 70% Some initially –ve became +ve after treatment	-
Stewart et al.	1961	77 teeth	100%	**0.5%NaOCl+Gly-oxide:** 2% **0.5%NaOCl+3%H_2O_2**: 9%	**No dressing:** 0.5%NaOCl+Gly-oxide: 34% 0.5%NaOCl+3%H_2O_2: 39%
Nicholls	1962	155 teeth	100%	**Alkaline chloramine:** 53% **H_2O_2 & 2%NaOCl:** 50% **H_2O & 2%NaOCl:** 71%	-
Grahnén & Krasse	1963	97 teeth	77%	**NaCl:** 72% **Biocept:** 66% **Nebacin:** 36% Some initially –ve became +ve after Tx	**No dressing:** NaCl: 47% Biocept: 47% Nebacin: 18%
Engström	1964	223 teeth untreated or retreated	60%	**Biosept or Iodophor, plus alcohol, chloroform and 0.5%** **NaOCl:** No data	**5% I_2 in 10% IKI:** 2nd visit: 43%; 3rd visit: 22%; 4th visit: 8%; 5th visit: 3%; 6th visit: 2%; 7th visit:16%

TABLE 33-5 Summary of Studies Evaluating the Effect of Root Canal Treatment Procedures on Bacterial Detection by Culture (*Continued*)

Study	Year	Sample Size	Percentage of samples with bacterial presence		
			At baseline	After preparation ± irrigation	Next visit (after dressing+/-)
Olgart	1969	207 teeth	72%	**H_2O_2 & 0.5%NaOCl or H_2O_2 & 1%NaOCl:** 43%	**No dressing:** 34%
Bence et al.	1973	33 teeth	100%	**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:** 48hr 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%	**NaCl:** 65%	**38%CMCP:** 20% 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% 0.5% NaOCl group: 63% 5% NaOCl group: 79%	**NaCl:** 83% **0.5% NaOCl:** 59% **5% NaOCl:** 68%	-
Byström & Sundqvist	1981	15 teeth	100%	**Saline:** 100%	**No dressing:** 47% (5th visit) Where initial bacteria load high, difficult to eliminate.
Byström & Sundqvist	1983	15 teeth	100%	**0.5% NaOCl:** 87%	**No dressing:** 20% (5th visit)
Byström & Sundqvist	1985	60 teeth	100%	**0.5% NaOCl:** No data **5% NaOCl:** No data **5% NaOCl+15% EDTA:** No data	**No dressing;** 0.5% NaOCl: 60% (2nd visit); 40% (3rd visit) 5% NaOCl: 50% (2nd visit); 30% (3rd visit) 5% NaOCl+15% EDTA: 55% (2nd visit); 15% (3rd visit)
Byström et al.	1985	65 teeth	100%	**0.5% NaOCl:** No data **5.0% NaOCl:** No data	**CH:** 0% (1 month), 12.9% (2-4 d) **CP/CMCP** (2 wks): 33.3%
Koontongkaew et al.	1988	15 teeth	100%	**3% H_2O_2/5.25%NaOCl:** No data	**CMCP**: 1 day dressing: 40%; 3 day dressing: 20%; 7 day dressing 10% **No dressing**: 60% after 1 day, 20% after 3 or 7 days
Reit & Dahlén	1988	35 teeth	91%	**0.5% NaOCl:** No data	**CH**: After 14 days: 23%; After 21 days: 26%
Molander et al.	1990	25 teeth	96%	**0.04% iodine:** No data	**Clindamycin**: After 14 days: 16%; After 21 days: 24%
Sjögren et al.	1991	30 teeth	100%	**0.5% NaOCl:** 50%	**CH:** 10 min: 50% at 1 wk later 7 day: 0% (none after 1-5 wks later without dressing)
Ørstavik et al.	1991	23 teeth	96%	**NaCl** irrigation and enlarged to: #20-25: 87% further to #35-80: No data	**CH**: 34%; #35/40: 40% #>40: 25%
Yared & Bou Dagher	1994	60 teeth	100%	**1% NaOCl:** Enlarged to #25: 73% Enlarged to #40: 23%	**CH:** 0%
Gomes et al.	1996	42 root canals. Untreated (n=15) Retreated (n=27)	95%	**2.5% NaOCl:** No data	**Empty canal** (7-10 days): 73%

TABLE 33-5 Summary of Studies Evaluating the Effect of Root Canal Treatment Procedures on Bacterial Detection by Culture (*Continued*)

Study	Year	Sample Size	Percentage of samples with bacterial presence		
			At baseline	After preparation ± irrigation	Next visit (after dressing+/-)
Sjögren et al.	1997	55 teeth (single canal)	100%	**0.5% NaOCl:** 40%	-
Dalton et al.	1998	46 teeth	100%	NaCl+NiTi files: 68% NaCl+K-files: 75%	-
Reit et al.	1999	50 teeth	84%	Enlarged to #35 (curved) or #50 (straight) with **0.5% NaOCl**: No data	**5% IKI** (5-7 days): 44% **Empty** (7 days): 44%
Peciuliene et al.	2000	25 teeth	80%	**2.5% NaOCl & 17% EDTA:** No data	**Medication unknown:** 28%
Shuping et al.	2000	42 teeth	98%	**1.25% NaOCl:** 38%	**CH:** 8%
Lana et al.	2001	31 teeth	87%	**2.5% NaOCl:** No data	**CH:** 13% **Empty for 7 days:** 23%
Peciuliene et al.	2001	40 teeth	83%	**2.5% NaOCl & 17% EDTA:** 30%	**CH (10-14 days)**: 25% **2% I_2 in 10% IKI (10 min):** 5%
Peters et al.	2002	42 teeth	Instrumentation to #20: 100%	**Enlarged to #35 with 2% NaOCl:** 23%	**CH** (4 weeks): 71%; Further irrigation: 43%
Card et al.	2002	40 mandibular teeth/canals	95%	**1% NaOCl** Profile instrumentation (.04 taper): 0/13 of cuspids & bicuspids, 5/27 of mesio-buccal canals; Further LightSpeed instrumentation to size 57.5–65: 3/27 mesio-buccal canals of molars. Only 1/16 of those mesio-buccal canals with detectable communication with the mesio-lingual canals had +ve culture after the first preparation using ProFile instruments.	No data
Kvist et al.	2004	96 teeth	98%	**0.5% NaOCl:** 63%	**CH** (7 days): 36% **IPI** (10 min): 29%
Chu et al.	2006	88 canals	99%	**0.5% NaOCl:** No data	**CH, Septomixine forte, or Ledermix**: 36% Exposure of pulp, tooth type, acute vs chronic condition, size of lesion & type of medication had no significant effect.
Siqueira et al.	2007a	11 teeth (single root)	100%	**2.5% NaOCl:** 55%	**CH/CPMC:** 9%
Siqueira et al.	2007b	11 teeth (single root)	100%	**2.5% NaOCl:** 45%	**CH:** 18%
Vianna et al.	2007	24 teeth (single root)	100%	**Saline+2%CHX gel:** 33%	**2%CHX, CH or mixture:** 54% Type of medication had no significant effect.
Wang et al.	2007	43 canals	91%	**Saline+2%CHX gel:** 8%	**2%CHX+CH:** 8% Size of apical preparation (40 vs 60) had no significant effect.
Markvart et al.	2012	24 teeth	88%	**2.5% NaOCl:** 63%	**17% EDTA irrigation and 10 min 5% IKI medication:** 50% Box preparation (#60): 67% Cone preparation (#25-30): 33%
Xavier et al.	2013	48 teeth (single canal)	100%	**1% NaOCl:** 75% **2% CHX:** 75%	**CH:** 75%

Key; NaOCl = Sodium hypochlorite; CH = Calcium hydroxide; CP = Camphorated phenol; CMCP = Camphorated monochlorophenol; PP = paper point sample; r culture = culture test before obturation

Some studies have found no relationship between individual species and treatment failure but others have. While the overall failure rate for cases with positive cultures was 31%, that for teeth with *Enterococcus* species was 55% and for teeth with *Streptococcus* species was 90%.[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 *Enterococcus faecalis* only achieved a success rate of 66%.[71] These associations cannot be regarded as cause-effect and a relationship should also be sought between microbial diversity and treatment outcome. The success rate for teeth with no detected microorganisms was 80%, whereas for teeth with detected microorganisms in the canal before obturation was 33%.

A monkey-model study[72] used a 4- or 5-strain infection to test the effect of debridement and obturation procedures on outcome. When bacteria remained after chemo-mechanical debridement, 79% of the root canals were associated with non-healed periapical lesions, compared with 28% when no bacteria were found to remain. Combinations of several residual bacterial species were more frequently related to non-healed lesions than were single strains. When no bacteria were detected at the end of chemo-mechanical debridement, healing occurred independently of the quality of the root filling. In contrast, when bacteria were detected in the canal system, there was a greater correlation with non-healing associated with poor-quality root fillings than with 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 for obtaining a sample for culture, the use of a negative culture result to inform progress of treatment has a positive impact on 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. It would be advantageous if future technology could render quick, reliable, and affordable chair-side detection of residual microorganisms. It is, however, important to account for the other factors that influence root canal treatment outcome.

Root filling material and technique

The inter-relationship between the core root filling material, sealer, and technique for their placement complicates the investigation of the effect of root filling material 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).[5] The sealers used may be classified into zinc oxide eugenol-based, glass ionomer-based and resin-based types.[5] Recently introduced materials such as Resilon®, SmartSeal®, and MTA have been adopted but not penetrated the market significantly, except for the use of MTA in apical surgery, immature apices, and perforation repair prior to obturation. In any case, there is no evidence to show that the nature of root filling material and the technique used for placement have any significant influence on treatment outcome.

Apical extent of root filling

Of the many intra-operative factors, this has been the most frequently and thoroughly investigated, presumably because it retrospectively offers a readily measurable outcome. In these previous studies, the apical extent of root fillings has been classified into three categories for statistical analyses. These are: (1) more than 2 mm short of radiographic apex (short); (2) 0-2 mm within the radiographic apex (flush); and (3) extended beyond the radiographic apex (long).[5] The apical extent of root filling was found to have a significant influence on the success rates of treatment, regardless of the periapical status.[5,13] Flush root fillings were associated with the highest success rates (Pooled success rate = 0.81; 95% CI: 0.76, 0.86), followed by short root fillings (Pooled success rate = 0.76; 95% CI: 0.71, 0.82), whilst long root fillings were associated with the lowest success rates (Pooled success rate = 0.66; 95% CI: 0.56, 0.75).

Most previous retrospective studies could not distinguish between the effects of apical extent of instrumentation *versus* the apical extent of obturation; however, the London Eastman study[13] was able to separate the effect of these two factors and found them both to independently and significantly affect periapical healing. The factors though did correlate with each other, consistent with the fact that canals are normally filled to the same extent as canal preparation. A single measure "apical extent of root filling," therefore, conveys information about both the extent of canal cleaning and obturation. The exception is when instruments or cleaning agents are overextended during canal preparation, without root filling extrusion.

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. Magnesium and silicon from the talc-contaminated extruded gutta-percha were found to induce a foreign body reaction, resulting in treatment failure.[73] 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.[74] The inference that perhaps extrusion of large pieces of gutta-percha may not impact on periapical healing was not supported by data from previous studies.[5,13] The discrepancy may possibly be attributable to microbial contamination of the extruded gutta-percha in the clinical scenario.

Radiographic evidence of "sealer puffs" extruding through the main apical foramina and lateral/accessory canals has been pursued with vigor in the belief of its value as "good practice" by some endodontists. Their perception is that

this sign is a surrogate measure of root canal system cleanliness and 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 et al.[75] found that extrusion of a glass ionomer-based sealer significantly reduced success rates. In contrast, Ng et al.[13] 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 to detect small traces of it.[13] It is theoretically possible that, in some cases, the radiographic disappearance of extruded sealer may simply be due to resorption of the radio-opaque additive, barium sulphate, or its uptake by macrophages still resident in the vicinity.[73]

Extruded glass ionomer-based,[75] zinc oxide eugenol-based,[76] silicone-based[76] sealers or Endomethasone®[77] were not found to be resorbed/absorbed by periapical tissues after one year. Traces of calcium hydroxide-based sealer (Sealapex®) could still be detected after three years.[78] 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®)[79] and a resin-based sealer (AH Plus, Dentsply/DeTrey, Konstanz, Germany)[78] was demonstrated in 69% and 45% of the cases after four and five years, respectively. Ng et al.[13] advanced two explanations for the difference between the effect of extruded core gutta-percha and zinc oxide/eugenol sealer; the latter is antibacterial and may kill residual microorganisms, whilst 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 "quality of root filling." The rationale for complete obturation of the root canal system is to prevent re-contamination by colonization from the residual infection or newly invading microorganisms. Both are putatively 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, since good obturation is reliant upon properly executed preliminary steps in canal preparation.

A recent systematic review[5] reported that the criteria for judging the quality of root fillings have not been well defined in previous studies.[18,41,55] An unsatisfactory root filling has been defined as "inadequate seal," "poor apical seal" or "radiographic presence of voids." Despite inadequacies in the criteria for judging root filling quality, satisfactory root fillings, judged by radiographs, were found to be associated with significantly higher success rates than unsatisfactory root fillings (Pooled OR = 3.9; 95% CI: 2.5, 6.2).[5]

Acute exacerbation during treatment

The aetio-pathogenesis of the inter-appointment "flare-up" or pain has not been precisely determined and several hypothetical mechanisms involving chemical, mechanical, or microbial injury to the periradicular tissues, as well as psychological influences, have been suggested as contributory to post-preparation pain and swelling. Although this factor has not been specifically studied in the context of periapical healing, acute "flare-ups" during treatment were found to have no significant association with periapical healing in two studies.[41,43] In contrast, the London Eastman study[13] found that pain or swelling occurred in 15% of cases after chemo-mechanical debridement, and significantly reduced 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 extra-radicular infection, resulting in treatment failure in some of these cases. Alternatively, acute symptoms may have been the result of incomplete chemo-mechanical debridement at the first appointment, leading to a shift in canal microbial ecology, favoring the growth of more virulent microorganisms and hence leading to post-preparation pain, as well as subsequent treatment failure. The exact biological mechanisms of failure in these cases remain obscure and warrant further investigation.

Number of treatment visits

The effect of number of treatment visits on periapical healing remains an on-going controversy, fuelled by debate between specialists and dentists arguing for single visit treatment on the basis of cost-effectiveness and business-sense against academics and some specialists arguing for multiple visit treatments, based on biological rationale. The main thread of argument for multiple visit treatments has been that primary debridement is not completely effective in eliminating all the adherent microbial biofilm[8] and the residual microorganisms may multiply and recolonize the canal system.[59,65] It is therefore considered desirable to use the inter-appointment period to dress the canal with a long-lasting or slow-release antimicrobial agent capable of destroying or incapacitating residual microorganisms, 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 microorganisms, detoxify antigenic material, and act as a slow release agent because of its low solubility-product in an aqueous environment. However, its antimicrobial ability has come under close scrutiny recently, with advocates suggesting that the material is not suitable for this purpose.[80] A final resolution to this debate is awaited based on robust clinical evidence. All the published randomized controlled trials found no significant influence attributable to number of visits, but they all lacked statistical power.[81-84]

The debate about the merits of single or multiple visit treatments will continue unabated given the respective strengths of the motivational drivers amongst the opposing groups. The issue may only be resolved by properly documented, large randomized controlled trials (currently unavailable) because unrecorded confounders (operator skill, biological or technical case complexity, and patient compliance) underlying the factor "number of treatment visits" would continue to play out their biasing effect in non-randomized studies. In any case, regardless of these debates, the long-standing clinical cornerstones for proceeding to obturation, that of absence of exudate, pus, odor and symptoms, should be followed.

Post Root Canal Treatment Restorative-related Factors

Effect of quality and type of restoration

The placement of a coronal restoration after root canal filling is the final step in the management of teeth undergoing root canal treatment and is shown to have a major impact on outcomes. Teeth with satisfactory coronal restorations were found to have significantly better periapical healing compared with those with unsatisfactory restorations (OR = 3.3; 95% CI: 1.1, 10.3).[5] The term "satisfactory restorations" has been defined variously as: (1) a restoration with no evidence of marginal discrepancy; (2) no evidence of marginal discoloration; (3) no evidence of recurrent caries; and (4) absence of a history of decementation.[18,85]

Given that one of the roles of coronal restorations is to prevent post-operative root canal re-infection, the criteria for unsatisfactory restoration given by Hoskinson et al.[18] and Ricucci et al.[85] could not infer coronal leakage when the inner core was still intact. Consequently, the London Eastman study[13] adopted a different classification and definition for unsatisfactory restorations in order to depict obvious and potential coronal leakage more effectively. 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 de-cementation. It is perhaps this strategy that contributed to the finding of an extremely profound effect (OR = 10.7; 95% CI: 3.7, 31.5) of this factor on the 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;[13,55,75] crown *versus* plastic restorations;[13,41,55,75] presence *versus* absence of posts;[13,75] and non-abutment *versus* abutment.[13,41] Teeth that had been permanently restored were associated with significantly higher success rates than their temporarily restored counterparts in some studies[55,75] but not in others.[13,86] The type of permanent restoration[13,41,55,75] 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 sub-seal over the root filling in case of loss of a permanent or temporary restoration; the sub-seal would be glass ionomer (GIC) or zinc oxide eugenol cement. 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 additional antibacterial coronal seal was found to have no beneficial effect on treatment success in a prospective study.[13] However, this may simply reflect a good definitive coronal seal.

In summary, it is advantageous that an adequate restoration should be placed after root canal treatment to prevent subsequent microbial 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 the restoration/tooth complex 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. Such teeth may, therefore, be expected to have lower success rates through development of cracks and fractures due to fatigue. This observation has been confirmed for teeth functioning as bridge abutments compared with those restored as individual units following root canal treatment.[41]

Summary of Factors Influencing Periapical Healing Following Non-surgical Root Canal Treatment

The factors having a major impact on periapical health following root canal treatment were:

- presence and size of periapical lesion;
- patency at the canal terminus (achieving patency significantly increased the odds of success by 2-fold)[13];
- apical extent of chemomechanical preparation in relation to the radiographic apex;
- outcome of intra-operative microbial sample and culture test;
- iatrogenic perforation (if present, the odds of success was reduced by 30%)[13]
- quality of root canal treatment judged by the radiographic appearance of the root filling;
- quality of the final coronal restoration.

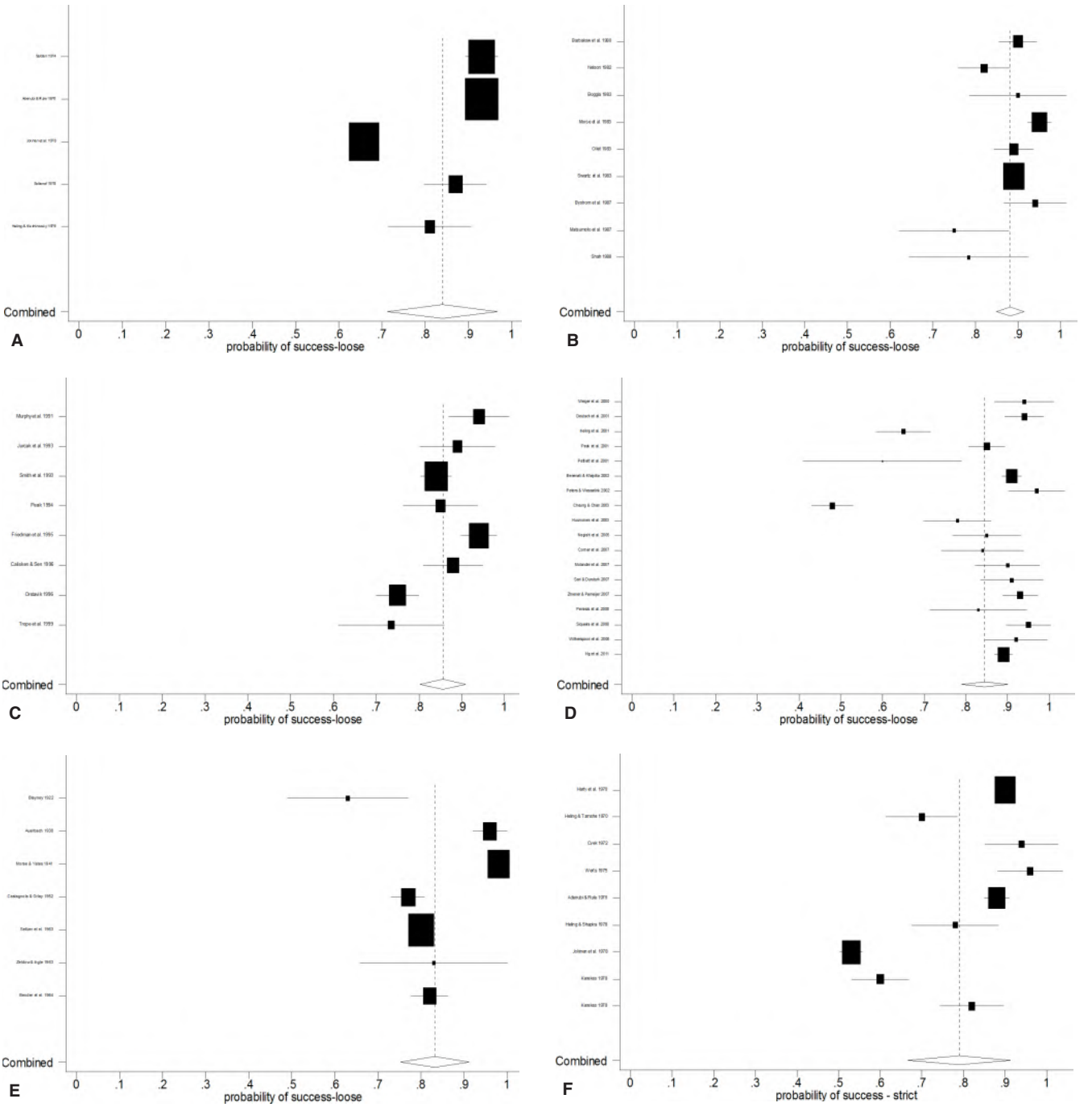
The factors having minimal effect on root canal treatment outcome were:

- age of patient;
- gender of patient;
- tooth morphological type; and

- specific root canal treatment protocol/technique (preparation, irrigation and obturation material and technique).

The improvements in techniques of mechanical and chemical canal preparation have not resulted in increases in success rates for both primary root canal treatment and secondary root canal retreatment over the last century (Figure 33-4). This fact may be explained by the observation that currently available techniques may be ineffective in eliminating the infection in the apical canal anatomy, resulting in ongoing interaction between the residual infection, root filling material and host defense. Such continued interaction probably plays a definitive role in determining the final outcome.

It is notable that all the factors having a strong influence on periapical health after root canal treatment are associated in some way with 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), the manner in which the microbiota is altered by treatment and the nature of its interaction with the host.



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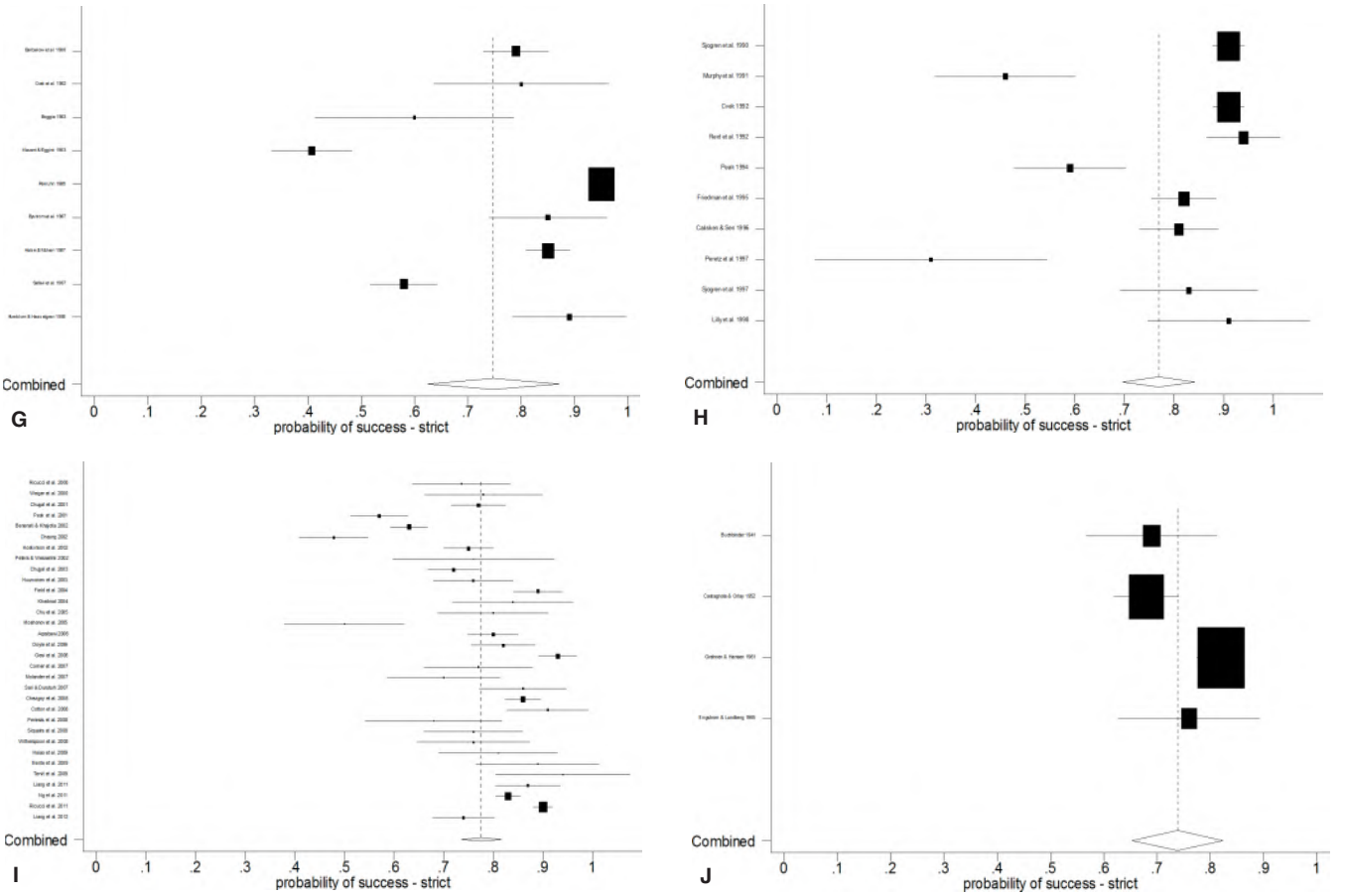

FIGURE 33-4 (Continued) ***A-J.*** Forest plots showing pooled and individual study's probability of periapical health for teeth undergoing root canal treatment by "decade of publication" and "criteria for success."

Factors Affecting Tooth Survival Following Root Canal Treatment

A systematic review and meta-analysis has shown that 93% of root-treated teeth survive at two years post-operatively; but this survival reduced to 88% at 10 years following treatment (Figure 33-5). The common reasons for such tooth loss were due to extractions precipitated by problems of endodontic origin; tooth/root fracture; or restoration failure.[87,88] Consistent with the studies on periapical healing, the prognostic factors for survival of teeth as the outcome measure may be divided into patient-related, intraoperative-related and restorative-related factors.

Patient-related Factors

Medical conditions

Ng et al.[88] found that teeth in patients suffering from diabetes or receiving systemic steroid therapy had a higher chance of being extracted after treatment. The negative influence of diabetes on tooth survival is consistent with the report by Mindiola et al.,[89] whilst the influence of steroid therapy had never been reported previously. It may be argued that patients suffering from diabetes were more susceptible to periodontal disease[90] or had a lower success rate of root canal treatment,[91] that in turn could be the reason for tooth extraction. They, however, 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.[92] It was further interesting to note that systemic steroid therapy is often prescribed to control such chronic pain.[93-95]

Tooth morphological type and location

Tooth types may vary in susceptibility to tooth fracture, a common reason for tooth loss after treatment. Ng et al.[87] found that tooth type had significant influence on survival, with non-molar teeth associated with significantly higher chance of survival than molar teeth (Pooled OR = 1.4; 95% CI: 1.1, 1.6). 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. These data do not confer any insight on the timing of crack origin or its propagation, merely that it was the decisive factor in failure; the cracks/fractures are likely to have pre-dated the root canal treatment.

The factors "proximal contacts" (Pooled OR = 2.6; 95% CI: 1.8, 3.7) and "terminal (last standing) teeth" were found to affect tooth survival significantly,[87,88] but were significantly

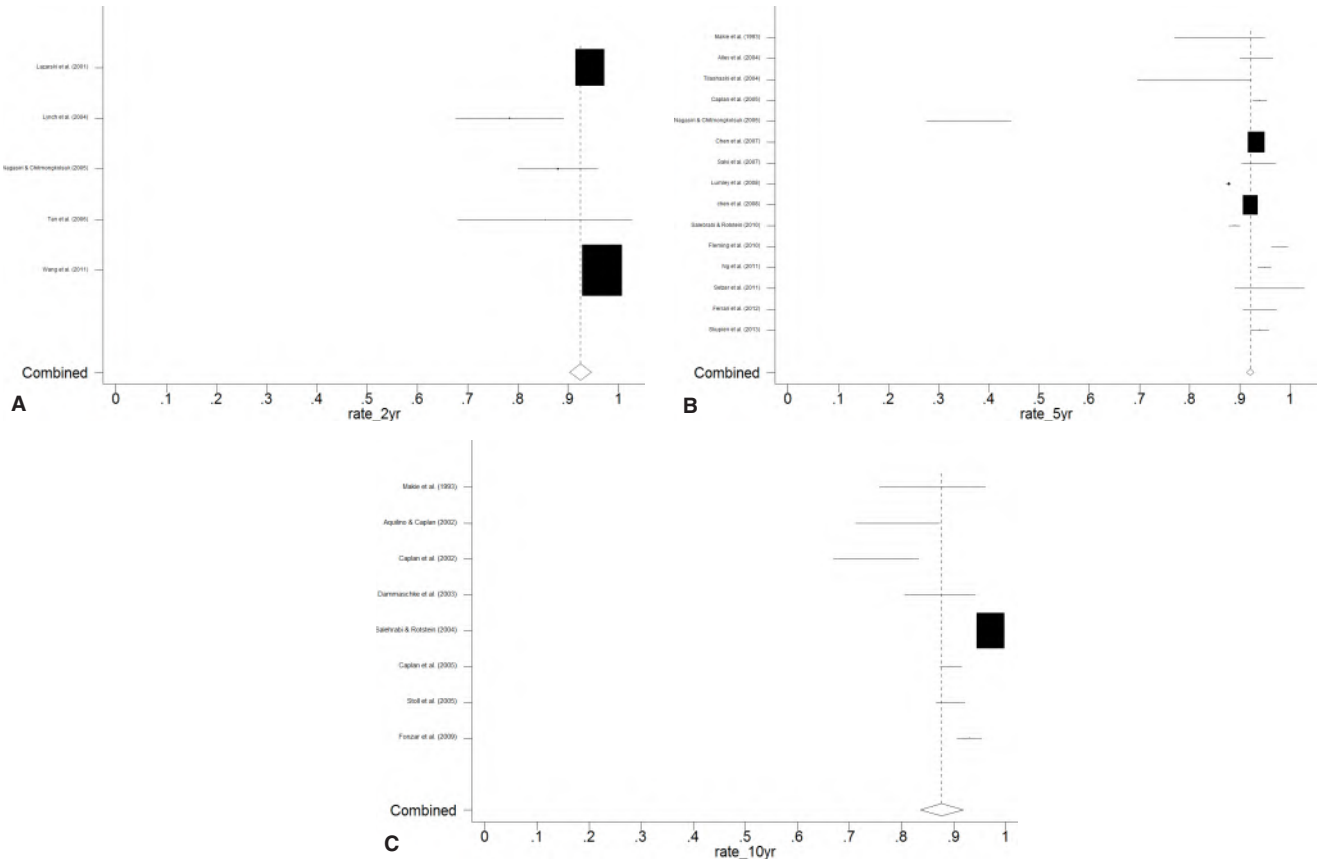

FIGURE 33-5 Forest plots showing pooled and individual study's probability of ***A.*** Two-year (Pooled probability = 0.93; 95% CI: 0.90, 0.95); ***B.*** Five-year (Pooled probability = 0.92; 95% CI: 0.91, 0.93); and ***C.*** 10-year (Pooled probability = 0.88; 95% CI: 0.84, 0.92) survival of teeth undergoing root canal treatment.

correlated to "molar teeth." Most of the extractions of terminal teeth, or teeth with one or less proximal contact, were due to tooth fracture.[88]

The observation may be explained by the unfavorable distribution of occlusal forces and higher non-axial stress on terminal teeth and those with less than two proximal contacts. Other possible reasons explaining their higher rate of loss are that: (1) failure of root canal treatment in a terminal tooth may be accepted more willingly because of little perceived esthetic value; and (2) clinicians may be less likely to offer further treatment on terminal molar teeth due to difficult access. It therefore seems important to ensure favorable distribution of occlusal forces when designing restorations for molar teeth, teeth with one or less adjacent teeth, and terminal teeth following retreatment.

Pre-operative conditions of teeth

The presence of pre-operative periapical lesions (the most significant prognostic factor for endodontic success) was found to have no significant influence on tooth survival.[88] On the other hand, pre-operative periodontal probing defects of endodontic origin, pre-operative pain, and pre-operative sinus tracts were found to reduce tooth survival.[88] These observations are consistent with a previous report that the mere presence of a periapical radiolucency was not a sufficient reason for dentists and patients to seek active treatment.[96] The negative impact of pre-operative pain on survival outcome highlights the importance of accurate pain diagnosis. In some instances, the pain may have been of non-endodontic origin, and therefore would persist after treatment.[19] In other instances, pre-operative 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 with pre-operative pain are crucial.

The presence of pre-operative cervical resorption and perforation were also found to significantly reduce tooth survival.[88] This was as expected because tooth fracture and re-infection due to leakage are likely sequelae in such cases. In the presence of re-infection, clinicians are more inclined to suggest extraction due to the intuitive perception of poor long-term prognosis of such teeth.

Treatment-related Factors

Amongst all the intra-operative factors, "failed to use dental dam,"[97] "lack of patency at apical foramen,"[88] and "extrusion of gutta-percha root filling"[88] were found to reduce tooth survival. It was found that extraction of teeth with the latter two conditions was more likely to be due to persistent endodontic problems as both of them were also prognostic

factors for treatment success based on periapical healing. 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-related Factors

Protection of teeth with crowns or cast restorations had not been found to influence treatment success based on periapical healing although placement of good cores had. In contrast, placement of crowns or cast restorations was found to improve significantly tooth survival (Pooled OR = 3.5; 95% CI: 2.6, 4.7).[87,88,98] This suggests that crowns and cast restorations help prevent tooth fracture, whilst the mere placement of a satisfactory core would be sufficient to prevent re-infection after treatment. Unfortunately, these studies were not able to investigate the inter-relationship between tooth morphological type, the extent of tooth tissue loss after treatment and the type of final restoration.

On the basis of laboratory[99] and clinical findings,[100] posterior teeth with compromised marginal ridges (mesial or distal), together with evidence of heavy occusal loading evidenced by faceting, may benefit from cast cuspal coverage restorations. The restoration design should attempt to preserve as much remaining tooth tissue as possible; the implication is that the so-called non-esthetic but technically demanding partial veneer onlays and partial coverage crowns would be the restorations of choice for root-treated teeth. In anterior teeth, the missing tooth tissue may often be replaced with adhesive restorative material. A crown is only indicated when the remaining tooth structure is compromised. With the advancement of adhesive dentistry, full crowns will probably be fabricated to replace existing old deficient crowns while more conservative cuspal protecting restorations will be used to restore endodontically treated teeth.

The use of cast post and cores for retention of restorations was also found to reduce tooth survival.[87,88] It may be speculated that the presence of posts has different effects on anterior and posterior teeth as they are subjected to different directions and amount of occlusal force. It was reported that only 12% of the extracted teeth with cast post and cores were incisors or canines.[88] Therefore, the inference is that the use of such retention should be particularly avoided in premolar and molar teeth. Alternative treatment options should therefore be considered for molar or premolar teeth lacking sufficient tooth structure. In contrast, a randomized controlled trial[101] revealed that fiber-post retention significantly improved survival of root-treated premolars restored with single-unit metal-ceramic crowns, regardless of the extent of residual coronal dentin with the exception of those teeth with four coronal walls remaining.

Ng et al.[88] observed that teeth functioning as prosthetic abutments had poorer survival but the number of teeth (n = 94) functioning as abutments was too small in their sample to demonstrate a statistically significant effect. As before, the explanation may reside in the excessive and unfavorable distribution of occlusal stresses on abutment teeth.

Summary of Factors Influencing the Survival of Teeth Following Root Canal Treatment

The conditions found to significantly improve tooth survival following root canal treatment were:

- non-molar teeth;
- teeth with both mesial and distal adjacent teeth;
- teeth not located distal-most in the arch;
- teeth (molar) with cast restorations after treatment;
- teeth not requiring cast post and core for support and retention of restoration;
- teeth not functioning as abutments for fixed prostheses;
- absence of pre-operative deep periodontal probing defects, pain, sinus tract, or perforation; and finally,
- achievement of patency at canal terminus and absence of root filling extrusion during treatment.

Impact of Root Canal Treatment on Quality of Life

The impact of root canal treatment on oral health-related quality of life of patients has been evaluated using the short form (OHIP-14) or modified version (OHIP-17) of Oral Health Impact Profile (OHIP-14). The distinctly positive impact of root canal treatment was apparent, regardless of cultural background of the patient group and the measure used. As expected, physical pain, psychological discomfort (feeling tense), and disability (difficulty in relaxing) were the most improved domains following treatment.

OUTCOME OF NON-SURGICAL 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. This requires removal of the previous root-filling material and any other material placed for restorative reasons. Correction of any iatrogenic procedural errors may also be required, if possible. All material must be removed in their entirety to ensure delivery of antimicrobial agents to all surfaces of the root canal dentin. The periapical healing rates of root canal retreatment are generally perceived to be lower, compared to primary treatment because of:

1. obstructed access to the apical infection; and/or
2. a potentially more resistant microbiota.

The outcomes from a range of studies show that the mean weighted success (healed) rate is 66%, about 6% lower than in the case of primary treatment on teeth with apical periodontitis (Figure 33-6).[13,102] On the other hand, the survival rate of

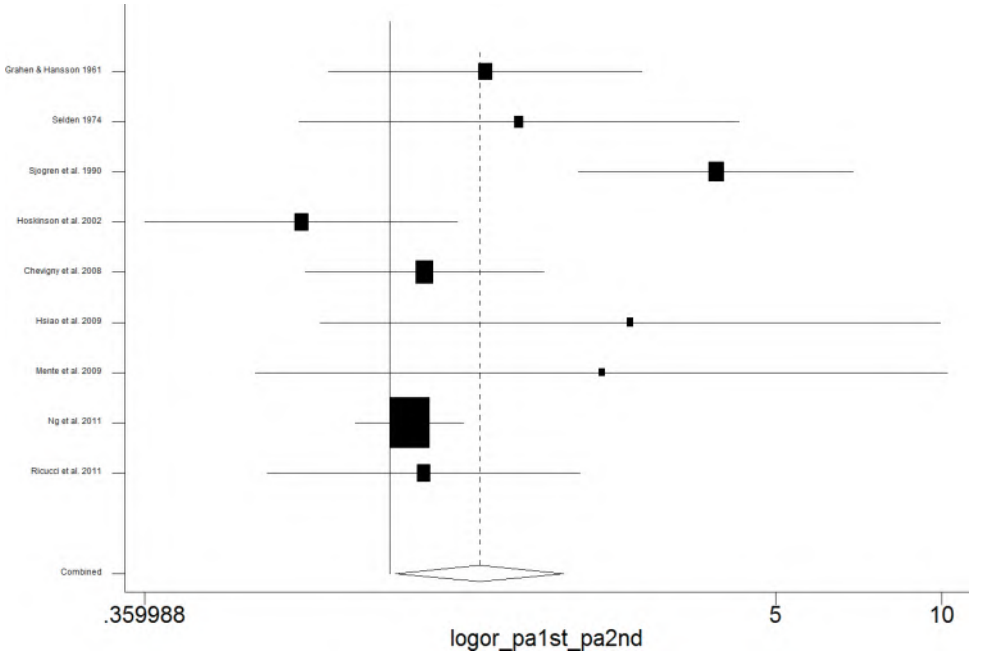

FIGURE 33-6 Forest plot showing pooled and individual study's odds ratio of periapical health for teeth with pre-operative periapical radiolucency undergoing primary root canal treatment versus root canal retreatment. (Pooled OR = 1.5; 95% CI: 1.0, 2.1.)

treatment. The differences between the microsurgical and traditional approaches may therefore have been exaggerated in their review.[107]

The authors' unpublished meta-analyses,[112] stratified by duration, following treatment revealed the pooled probability of success based on complete healing plateaued after two years post-operatively: being 51% (95% CI: 42%, 60%) after six 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; and 74% (95% CI: 66%, 82%) after more than 48 months. It is therefore advisable to follow-up periapical surgery cases for a minimum of two years; and better still for up to four years.

The potential prognostic factors for surgical endodontics may be classified into patient-related factors, surgical treatment-related factors, and restorative-related factors.

Patient-related Factors

Previous reports on the influence of patient's age and sex on root-end surgery outcome have been inconsistent. Meta-analysis of pooled data and the authors' data revealed minimal influence attributable to this factor.[112] As expected, most studies restricted their surgical cases to healthy patients. Only two studies had included both healthy, as well as patients with systemic medical conditions, and found no significant influence attributable to this status.[112,113] Tobacco smoking habits, that have a negative impact on the regenerative potential of human adult stem cells,[114] has been found to have no significant influence on root-end surgery outcome.[113,115,116]

The majority of the root-end surgery outcome studies published up to early 1990s had restricted their cases to single-rooted anterior teeth. Outcome data on posterior teeth had only become available to a greater extent within this millennium. The reported higher success rates associated with anterior (compared to posterior) teeth[115,117,118] or maxillary (compared to mandibular) teeth[119] could be attributed to better surgical access. As an exception, maxillary lateral incisors were often associated with scar tissue healing.[23,120] This healing outcome has been attributed to the palatal curvature of the root apex, that may allow the disease process to perforate the palatal cortical plate more readily, ultimately resulting in a "through and through" defect after surgical access. However, these observations have not been consistently reported. Meta-analyses of pooled published data revealed tooth type had minimal effect on root-end surgery outcome.[112]

The extent and nature of the pre-operative periapical disease may have a profound influence on periapical surgery outcome, and may be characterized by the clinical (swelling, sinus, pain) and radiographic (size of radiolucency) presentation, extent of bone defect, and histopathological diagnosis of the biopsied tissue. The extent of pre-operative periapical radiolucency (or bone defect) has a strong negative impact on root-end surgery outcome. A number of reasons may be advanced to explain this observation. The diseased tissues in larger periapical lesions may be more difficult to remove in their entirety, and require longer duration for complete bone repair; there may also be a greater likelihood of healing by secondary intention (fibrous tissue repair) when both buccal and palatal cortical plates are perforated. In contrast,

teeth having undergone root canal retreatment has been found to be similar to that for primary root canal treatment.[88]

The factors affecting outcomes of periapical health and tooth survival, following root canal retreatment, were otherwise identical to those affecting primary root canal treatment. Of the potential prognostic factors unique to retreatment cases, the main one showing significant influence on outcome after treatment was the ability to remove or bypass preexisting root filling material or separated instrument during retreatment. This is understandable because it would have a direct impact on the ability to achieve canal patency at the terminus.[13] For more details, see Chapter 23.

OUTOME OF SURGICAL ENDODONTICS

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 involving apical filling.[38,103-111] The major limitations of these pooled data sets are the uncontrolled duration of follow-up after treatment and that "loose" radiographic criteria had been adopted. An (as yet) unpublished meta-analysis, by the authors,[112] using data from the studies listed in Table 33-6 revealed that the weighted pooled probability of success defined by complete radiographic healing was 67.5% (95% CI: 62.9%, 72.0%) following periapical surgery with root-e FIGURE 33-7 nd cavity filling (Figure 33-7). The forest plot trend in Figure 33-7 shows higher success rates 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 so-called "modern" technique (*using magnification, root-end resection with minimal or no bevel, root-end cavity preparation with ultrasonic tips, and modern root-end root canal filling*).[109,110] In congruence with the above finding, Setzer et al.[107] 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%). However, the studies included in the latter meta-analysis differed in design, case selection, duration of follow-up, and the provision of pre-operative non-surgical

TABLE 33-6 Studies Investigating Periapical Healing Following Apical Surgery

Author	Design	Examination	Type of radiograph	Radiographic Criteria for success	Sample size			Duration after treatment (months)
					Patients	Teeth	Roots	
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 & 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 & 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
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

TABLE 33-6 Studies Investigating Periapical Healing Following Apical Surgery (*Continued*)

Author	Design	Examination	Type of radiograph	Radiographic Criteria for success	Sample size			Duration after treatment (months)
					Patients	Teeth	Roots	
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 & Kurt 1999	Prospective	C & R	Pa	Von Arx & 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 & 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 & 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
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
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 & Kurt 1999	52	71	95	6-12
Taschieri et al. 2005	Prospective	C & R	Pa	Rud et al. 1972	32	46		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 & DPT	Other	45	45		6-24
Penarrocha et al. 2007	Prospective	C & R	Pa	Von Arx & 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
Taschieri et al. 2007c	Prospective	C & R	Pa	Molven et al. 1987	28	28		12

TABLE 33-6 Studies Investigating Periapical Healing Following Apical Surgery (*Continued*)

Author	Design	Examination	Type of radiograph	Radiographic Criteria for success	Sample size			Duration after treatment (months)
					Patients	Teeth	Roots	
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 & 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 & 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 & 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 & Kurt 1999	75	87		12
Taschieri et al. 2010	Retrospective	C & R	Pa	Molven et al. 1987	76	112		48
Goyal et al. 2011	RCT	C & R	Pa	Rud et al. 1972	25	25		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 & Kurt 1999	23	31		12-19
Song & 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 & DPT	Von Arx & 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		12-84
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
Song et al. 2014	Retrospective	C & R	Pa	Rud et al. 1972		115		12-96

RCT = Randomized controlled trial; C = Clinical; R = Radiographic; Pa = Periapical; DPT = Dental Pantomogram; PAI = Periapical index

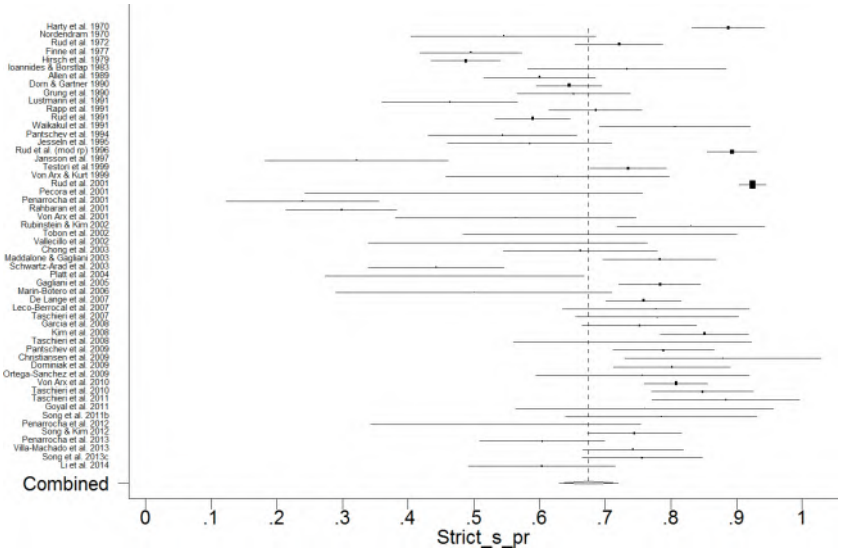

FIGURE 33-7 Forest plot showing pooled and individual study's probability of complete periapical healing following apical surgery.

removal of diseased tissue from a small apical lesion may lead to enlargement of the bony crypt to gain access, resulting in more complete eradication of such tissue.

A further problem of the extent of bone defect manifests when bone absence extends along the root face as a root dehiscence, called an apical-marginal defect; under normal circumstances, the root is expected to have connective tissue attachment to the overlying mucosa. If these soft tissues are detached during flap reflection, healing may be accompanied by apical migration of the junctional epithelium along the bone-denuded root surface and into the periapical bony crypt,[121] this would present radiographically as incomplete, or scar tissue, healing. Such types of healing outcomes have been reported often in teeth with apical marginal defects.[116,122] It has been proposed that such an attachment may be more susceptible to dissemination of marginal inflammation to the apical area,[108] although the periodontal literature does not accord with this view.[123]

The presence of swelling, sinus tract, or symptoms at the time of surgery has been found to be a significant outcome predictor.[116,124] It has been speculated that these presentations signify a (sub)acute phase of disease with possible "extra-radicular" infection, complete eradication of which may be difficult.[111]

The taking of a routine biopsy during root-end surgery to establish a definitive diagnosis has become an accepted standard of care in modern endodontic surgery. The histopathological diagnosis of the biopsied periapical lesion is frequently found to be a "granuloma" or "cyst."[125] Both pathological entities involve inflammatory responses to intra- or extra-radicular infection. Once the pathological tissues have been enucleated and the source of infection controlled, the nature of the pre-existing diseased tissue is unlikely to influence the healing process after its enucleation and root-end surgery.[112,125-127]

The quality of previous treatment, including the coronal restoration and root filling, as well as history of non-surgical retreatment or periapical surgery have been reported to influence surgical outcome. An adequate coronal restoration is considered a prerequisite for teeth undergoing periapical surgery. Only 26% (n=21/81) of the studies, reviewed by the authors, provided information about the restorative status.[112] The significant impact of the pre-operative quality of coronal restoration was reported in one study.[128]

A satisfactorily disinfected and obturated root canal system has also been considered to be a prerequisite for periapical surgery.[129] However, one of the key indicators for surgery includes the scenario where the root canal system could not be accessed to its apical terminus. Therefore, investigation of the effect of this factor may be limited by potential confounders. A number of studies that used multi-variable regression models to control confounding revealed no significant influence of the density[112,128] or extent[112,113,130] of pre-existing root filling. In contrast, the Toronto study[131,132] reported that a pre-operative root-filling of adequate length was associated with an increased odds of disease-persistence after surgery compared with those teeth with short or over-extended pre-operative root fillings, even after adjusting for potential confounding. It is, however, worth noting that in this study a root-end filling was *not* placed in 14% of the cases. It may be speculated that the apical extent of the pre-existing root filling may have no prognostic value as long as a retro-cavity is prepared and filled. Taking the argument further, it is technically even possible, circumstances allowing, to perform root canal treatment from the root-end using files or ultrasonic instruments of adequate length, coupled with a suitable gutta-percha obturation technique.[133] (See Chapter 34 and Figures 34-14 and 34-15)

Root-end surgery performed to an optimal standard may still be associated with persistent periapical disease because of residual intra-radicular infection, new intra-radicular infection, established through coronal leakage or root fracture, or residual extra-radicular infection. Repeat surgery adopting a different strategy or approach is therefore liable to succumb to the same failure, explaining the consistently reported poorer prognosis in such cases.[111,112,134]

Surgical Treatment Factors

The influence of adopting technical advances on surgical outcomes has been extensively investigated since 1990s, including the effect of using enhanced magnification and illumination, micro-surgical instruments and techniques, as well as the root-end filling materials.

Magnification and illumination of the surgical site, using an operating microscope, may allow better appreciation of the periapical tissues, bony crypt and root surface, facilitating more effective management of the crypt and root end. Meta-analysis[112] of data available from two observational studies[113,118] showed that magnification was associated with a significantly higher success rate. Meta-analyses[38,103,110] comparing the type of magnification (magnifying loupes, surgical microscope, or endoscope) showed no significant impact on root-end surgery outcome except an updated meta-analysis by Tsesis et al.[109] The latter suggested that using a surgical microscope, or endoscope, achieved significantly better outcomes than using loupes. However, the observation did not hold true when the analyses were stratified by root-end filling material.

Other contemporary procedures in root-end surgery include the use of back-exhausted air-rotor handpiece for osteotomy and apicectomy, minimal bevel root-end resection, root-end cavity preparation with ultrasonic retro-tips, and modern root-end filling material.

The effect of osteotomy technique on root-end surgery outcome has not been reported previously. The authors' meta-analysis[112] revealed no obvious difference in success rates using the air-rotor or air-motor handpiece. Investigation of the effect of surgical crypt size has been limited by its significant correlation with the pre-osteotomy extent of the lesion.[112,132,135] Therefore, the influence of the amount of bone removal cannot be determined.

Contemporary root-end management with minimal bevel resection and ultrasonic retro-tip cavity preparation (*conventionally with 3mm in depth*) has consistently been associated with higher success rates when compared with the traditional approach (*high respective angle, root-end cavity preparation with bur*).[107,112,113,118,119,136] The better outcome is attributed to more predictable and effective root-end management (capturing apical canal complexities, root-end preparation, and root-end filling).

Other contemporary root-end management techniques include retrograde root canal treatment and the "through and through" surgical approach (orthograde root canal treatment combined with a surgical approach in the same procedure). The latter approach is indicated when exudation into the root canal system cannot be controlled by non-surgical means. Recent unpublished study[112] revealed that the retrograde root canal treatment was associated with the highest success rates (59%, 88%), followed by the conventional ultrasonic retro-preparation approach (53%, 80%), and then the through and through approach (48%, 74%), based on both strict or loose criteria, respectively.

Exploration of the literature on retro-cavity and retro-filling techniques reveals the interesting observation that periapical surgery prior to the mid-1990s only involved root resection without any form of retro-preparation or filling. Periapical surgery adopting a traditional root-end filling technique conferred no advantage to healing when the orthograde root-filling was judged to be satisfactory.[120,135,137-140] In contrast, a randomized controlled trial revealed that teeth treated with "smoothing" of the orthograde gutta-percha root filling after apicectomy (52%) were associated with a significantly lower 1-year healing rate (loose criteria) than those teeth treated with root-end cavity and filled with MTA (97%).[141] The absence of a root-end filling has also been implicated as one of the most common possible causes of failure in periapical surgery.[142]

The types of retro-filling materials used in the studies reviewed by the authors [112] included: amalgam, super ethoxybenzoic acid (super-EBA®), zinc oxide eugenol based intermediate restorative material (IRM®), gutta-percha, composite resin, glass ionomer cement (GIC), and Cavit®. Investigation of the influence of root-end filling material may be confounded by the root-end management approach in observational studies. For example, amalgam had mostly been used in studies adopting a traditional (class 1) root-end cavity. On the other hand, flowable composite (Retroplast™), in combination with a dentin bonding agent,[143] exclusively used a saucer-shaped retro-cavity, designed to increase the surface area for adhesion, that would be enhanced by polymerization shrinkage towards the dentin surface rather than away from a canal wall. The Retroplast™ approach yielded significantly better periapical healing (73%) in a randomized controlled trial compared with GIC (31%).[144] Other randomized controlled trials have found no significant difference in the success rates of periapical surgery when contemporary root-end filling materials were compared: MTA (92%, 92%)[145,146] *vs* IRM (86%, 87%)[145,146], respectively; MTA (96%) *vs* super-EBA (93%)[147]; IRM (91%) *vs* super-EBA (82%)[148]; and IRM (85%) *vs* gutta-percha with AH plus sealer (90%)[149]. Of note, all the above trials had only followed up their cases for one year (with one exception)[145] and also adopted loose criteria for determination of periapical healing outcome. The authors' meta-analyses[112] pooling data from both observational studies and randomized controlled trials concurred with the above findings. Use of amalgam as a root-end filling material was associated with a significantly lower success rate than using IRM or super-EBA, whilst no significant difference was found in the success rate when MTA, super-EBA, IRM or composite resin were used.

The use of hemostatic agents has been reported in the majority of previous studies, including adrenalin-impregnated cotton pellets, epinephrine-containing local anesthetic saturated pellets, ferric sulfate, bone wax, thrombin, calcium sulfate, gelatin-based foam, oxidized cellulose, or bovine-derived collagen. Wang et al.[131] found their use did not have significant influence on periapical healing outcomes, while the effect of type of hemostatic agent had not been investigated.

The use of guided tissue regeneration techniques, such as membranes and/or grafting material, has been advocated

for cases with "through and through" defects (missing buccal and palatal cortical plates) but there are inconsistent reports on their benefits. The authors' meta-analyses[112] did not reveal a significant influence of such approaches on the periapical healing outcome.

Post-operative Factors

The influence of post-operative factors such as the presence of signs and symptoms post-surgery had only been investigated in two studies.[112,135] Rud et al.[135] reported that sero-hemorrhagic exudate under the flap, or an abscess present post-surgery, was not significantly associated with periapical healing. However, another study[112] revealed the presence of tooth tenderness to percussion at the suture removal appointment was associated with a significantly reduced chance of ultimate periapical healing.

Antibiotics had been prophylactically prescribed post-surgery to prevent wound infection but both a randomized controlled trial[146] and a meta-analysis[111] of observational data concluded that there was no beneficial effect.

The influence of the quality of coronal restoration at review has only been investigated by a few studies, a contrast to the literature on non-surgical root canal treatment outcome. Nevertheless, placement of a permanent restoration post-surgery or teeth with satisfactory coronal restoration at recall was associated with better outcome.

Summary of Factors Influencing Periapical Healing Following Root-end Surgery

The factors having a major impact on the outcome of periapical surgery with root-end cavities and fillings in teeth with pre-operative periapical radiolucencies based on the authors' meta-analyses[112] are:

- 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);
- with *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); and
- use of retro-filling material with mineral trioxide aggregate cement (MTA), super ethoxybenzoic acid cement (EBA), 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), whereas 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 pre-operative presence of signs and/or symptoms (risk ratio = 1.2; 95% CI: 1.1, 1.3)[113], periodontal status (risk ratio= 2.1; 95% CI: 1.1, 3.8)[130] and quality of the coronal restoration (risk ratio= 1.6; 95% CI: 1.2, 2.1)[113] have also been revealed as significant prognostic factors by individual studies.

The factors having minimal effect on surgical re-treatment outcome[112] were:

- age of patient;
- gender of patient;
- general health of patient;
- tooth type;
- quality of the pre-existing root canal filling judged radiographically, and
- histological diagnosis of the biopsied periapical lesion (cyst or granuloma).

The periodontal attachment levels after periapical surgery is an additional soft tissue outcome measure. Studies have compared the effect of different soft tissue incision techniques (intrasulcular with or without involving interproximal papilla, submarginal, papilla-base).[150-153] 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 a greater biological awareness of clinicians.[107,111] 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 non-surgical root canal treatment, the outcome measure of tooth survival of surgical cases was investigated in one study[154] at the time of writing. The reported median survival time for first-time surgery was 92 months (95% CI: 41, 143) and that for re-surgery was 39 months (95% CI: 6, 72).[154] 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).[155] 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 post-operation. The impact on physical pain has also been explored by two other studies;[156,157] both reported the post-operative pain was of

relatively short duration, with the intensity peaking at three to five hours post-operatively and progressively decreasing with time, with no significant influencing factors identified.

CONCLUSIONS

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 economic perspective, conventional root canal treatment is a very cost-effective treatment as a first line intervention to extend the life of the tooth with a pre-operative periapical lesion.[2,3] If root canal treatment subsequently fails, non-surgical and surgical retreatments are also more cost-effective than replacement with a prosthesis.[2,3] 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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Chapter 34

Achieving Long-Term Success with Endodontic Therapy

Fernando Goldberg

This chapter highlights the important role of endodontic treatment in retaining the natural tooth in the dental arch and thereby preserving or restoring oral health.

General Aspects

To achieve a successful endodontic outcome, previous chapters describe procedures to perform adequate endodontic treatment. In addition to other procedures, forms of therapy used in endodontic treatment include pulpectomy, treatment of pulp necrosis, retreatment, and periradicular surgery. Success attributed to these treatments varies from 80% to 95%. This rate changes according to the anatomical and pathological variables of each clinical case as well as the practitioner's skills performing the indicated procedures. Given this success rate, endodontic treatment is always preferable to extraction and implant placement.[1] Saving the tooth means that periodontal tissue can be preserved and therefore ensures that normal periradicular bone is maintained (Figure 34-1).

Because an implant is a foreign structure, devoid of periodontium, the most it can do is maintain surrounding bone but not produce new crestal bone. For this reason, the amount of annual bone loss considered normal is used as one of the criteria for assessing implant success. This premise does not exist in endodontics. Our aim is to remove the entire diseased content of the root canal system so one can perform a three-dimensional obturation and thus return the tooth to periodontal health.

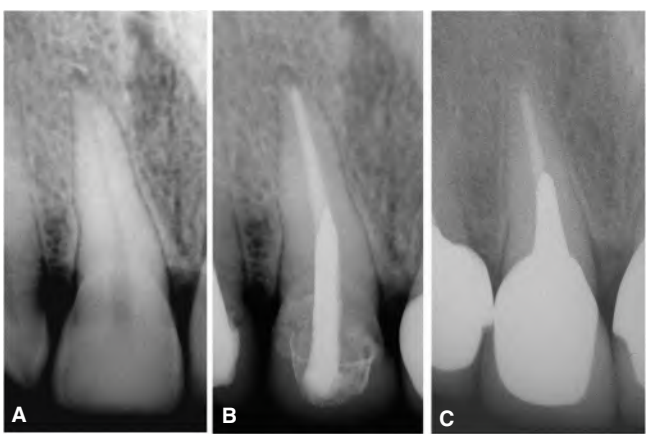

FIGURE 34-1 Long-term follow-up of nonsurgical endodontic treatment in a maxillary central incisor of a 32-year old female. ***A.*** Preoperative radiograph showing evidence of periapical radiolucency. Pulp vitality tests were negative. ***B.*** Postoperative radiograph. ***C.*** 31-year follow-up radiograph showing normal appearance of periapical area and crestal bone.

Efficient Endodontic Treatment → Healthy Periodontium → Normal Periradicular Bone

In general, and except for very specific cases, when endodontic treatment meets all the requirements for successful root canal treatment, the end result will be to maintain or restore the health of the periodontal tissues: cementum, periodontal ligament, compact bone or *lamina dura* and alveolar bone.

It is necessary to re-sensitize the dental profession to the importance of saving the natural tooth. A distressing survey among dentists in the United States in 2011 showed that dentists currently may have less confidence in endodontic treatment versus dental implants.[2]

Concept of Long-Term Success in Endodontics

Success in endodontics is determined by various aspects, for example, clinical, radiographic and patient conditions.

Clinical: Subjective–absence of any type of pain. Objective—absence of local or widespread swelling, mobility, and gingival or cutaneous sinus tract.

Radiographic: Normal thickness of the periodontal ligament space, continuity of the alveolar compact bone, recovery of alveolar cancellous bone, and absence of root resorption.

For calibration purposes, Ørstavik et al.[3] recommended the use of an ordinal scale (PAI, periapical index) of five categories for radiographic assessment of endodontic treatment results. Scores 1 and 2 correspond to a sound or normal periapical status, whereas scores of 3, 4, and 5 reflect changes in this area.

Currently, advanced imaging techniques, such as Cone Beam Computerized Tomography (CBCT), are important diagnostic aids. For more details, see Chapter 9C. In many cases, CBCT allows a more accurate interpretation of periradicular status, than conventional periapical radiography.[4–8]

*Patient Condition*s: The aim is to return the patient's oral health, function, and esthetics. Friedman and Mor[9] consider the terms *success* and *failure* to be ambiguous, and instead suggest using the terms *healed*, *healing* (in progress), or disease. Healed means clinical and radiographic normalcy, whereas ***healing*** means a normal clinical presentation combined with reduced periradicular radiolucency.

The authors identify *disease* as the emergence or persistence of periradicular radiolucency with clinical normalcy, or the presence of clinical signs or symptoms despite normal radiographic appearance. They also define *functional retention* as the presence of clinical normalcy with either the absence or presence of periradicular radiolucency.

According to the American Association of Endodontists' recommendations[10] the long-term results of endodontic treatment may be categorized as:

Healed—Functional, asymptomatic tooth with minimal or no radiographic periradicular pathosis.

Non-healed—Nonfunctional, symptomatic tooth with or without radiographic periradicular pathosis.

Healing—Tooth, asymptomatic and functional, with periradicular radiographic pathosis, or symptomatic tooth with or without radiographic periradicular pathosis but with unaffected function. In this regard, a functional tooth is a treated tooth or root that is serving its intended purpose in the dentition.

Gutmann et al.[11] have suggested the use of three categories when assessing long-term postoperative clinical and radiographic follow-ups: *Acceptable, Uncertain and Unacceptable*, with a fourth category corresponding to *acceptable clinical function.* After carrying out endodontic treatment, a prudent observation period of four years is generally applied for assessing the state of periradicular health, clinically and radiographically, and deciding the best approach.[11]

Conventional radiography offers an incomplete picture, since it represents a two-dimensional image of a three-dimensional reality. This often hinders accurate diagnosis, both preoperatively and postoperatively. This includes superimposition of neighboring anatomical features. Additionally, formation of non-calcified fibrous scar tissue, without an inflammatory reaction, may be associated with the repair mechanism of large periradicular lesions.[12,13]

It must be remembered that radiographic interpretation merely provides supporting data that should always be backed by a thorough clinical assessment. The patient must be made aware of the importance of long-term observation of the endodontic procedure and periodical follow-up appointments should be scheduled.

Suggested Clinical Approaches

Clinical approaches can be implemented in each of the following scenarios:

Healed: When there are no clinical symptoms and/or radiographic signs indicating recovery of periradicular tissues. One is also advised to monitor coronal restorations periodically. (Figure 34-2).

Healing: In such cases, the patient must be made aware of the need for regular clinical and radiographic check-ups (every 6 months to 1 year) (Figure 34-3). During this period, it is imperative to monitor the coronal restoration to ensure coronal seal and prevent possible fractures.

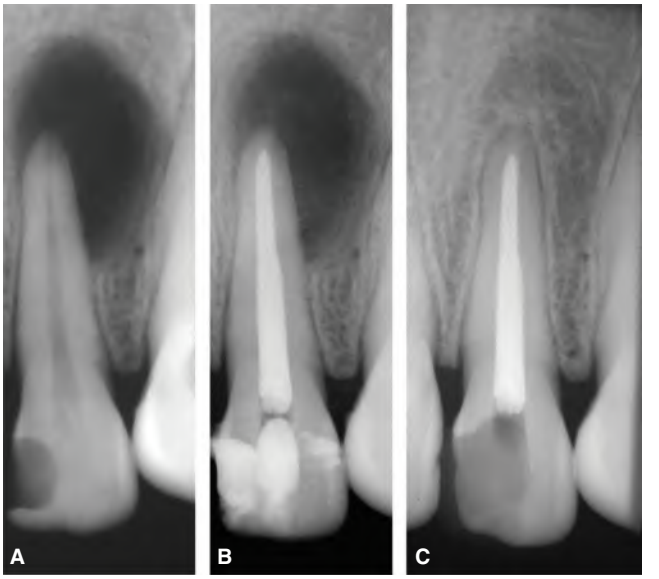

FIGURE 34-2 Healing of large periapical lesion in a maxillary lateral incisor of a 43-year-old female. ***A.*** Preoperative radiograph showing a large periapical radiolucency. Pulp vitality tests were negative. ***B.*** Immediate postoperative radiograph. ***C.*** Four-year follow-up radiograph showing resolution of the periapical radiolucency.

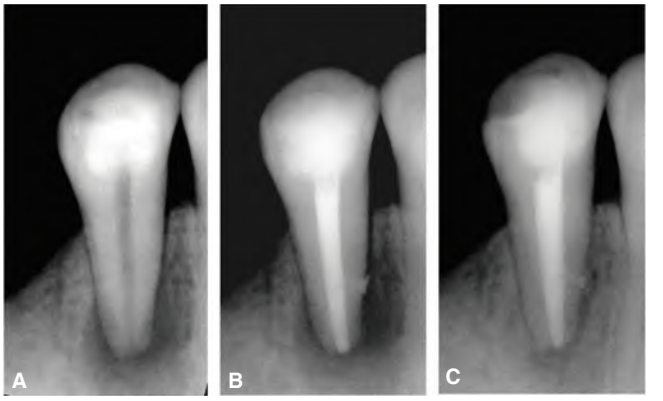

FIGURE 34-3 Healing of periapical lesion in a mandibular premolar of a 55-year-old male. ***A.*** Preoperative radiograph showing a large periapical radiolucency. Pulp vitality tests were negative. ***B.*** Immediate postoperative radiograph. Extrusion of sealer through a lateral portal of exit is evident (arrow). ***C.*** One-year follow-up radiograph showing active resolution of the periapical radiolucency.

Not healed: If a persisting or progressing periradicular radiolucency is diagnosed radiographically, in conjunction with subjective clinical symptoms (induced or spontaneous pain) or objective symptoms (localized or diffuse swelling, gingival or a cutaneous fistula), the patient must be advised to undergo non-surgical or surgical retreatment or extraction. The decision is based on local and general factors (Figure 34-4).

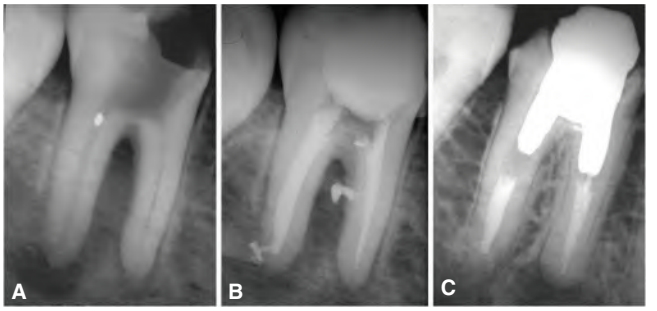

FIGURE 34-4 Treatment of a periradicular pathosis in a mandibular first molar of a 28-year-old male considered a failure. ***A.*** Preoperative radiograph showing periradicular radiolucencies associated with the mesial and distal roots as well as the furcation area. Root canal space exposed to the oral cavity. ***B.*** Immediate postoperative radiograph. Remnants of sealer extruding through a lateral portal of exit toward the furcation area. ***C.*** Five-year follow up radiograph showing marked reduction in size of the radiolucency associated with the furcation and the distal root. However, the periapical radiolucency in the mesial root has not been resolved.

Functional Retention: After a detailed clinical and radiographic examination, the patient should be made aware of the tooth in question, and given proper advice about the desirability of checking the tooth regularly. For example, patients with possible post-extraction complications, elderly patients, patients with anatomical or functional disadvantages for prosthetic and implant replacement or alternatively suggest extraction. When in doubt, and when merited, a computed tomography scan may contribute to a more thorough assessment thereby making it possible to pronosticate the outcome with greater certainty.

In all cases, a differential diagnosis should be done to clarify whether the cause of the detected disease is endodontic (e.g., intracanal infection), non-endodontic (e.g., neighboring conditions of non-endodontic origin) or mixed (e.g., combined endodontic-periodontal lesions). For more details, see Chapters 3, 8, 17, and 36.

Factors Involved in Achieving Endodontic Success

These factors can be divided into those that are derived from the endodontic process itself, general patient-dependent factors, and even factors involving the practitioner. Regarding endodontic treatment factors, all stages of treatment must be considered, beginning with a correct diagnosis and treatment plan, followed by efficient implementation of all procedures—including cosmetic and functional restoration of the treated tooth—and ending with confirmation of the repair by long-term postoperative observation.

During treatment, the practitioner will have to overcome challenges presented by anatomical complexities of the tooth being treated, which may affect a successful outcome (Figure 34-5). For more details, see Chapter 26. Similarly, the severity

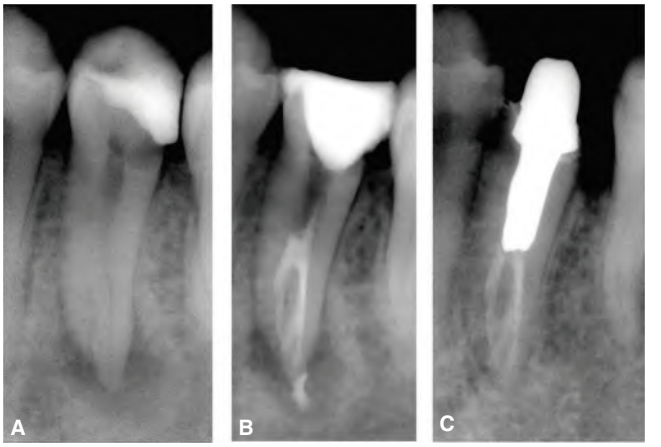

FIGURE 34-5 Endodontic treatment in a symptomatic mandibular premolar with complex radicular anatomy. ***A.*** Preoperative radiograph showing periapical radiolucency associated with necrotic pulp. ***B.*** Immediate postoperative radiograph showing endodontic filling of a trifurcated root canal system. ***C.*** 3-year follow-up radiograph showing resolution of the periapical radiolucency.

of the disease present will be a crucial factor when making a prognosis and deciding on the action to be taken. Among general factors, it is essential to consider those concerning the biological response of the patient (host). With regard to the dentist, the practitioner's experience in endodontic practice is of importance when analyzing the prognosis.

Long-Term Observation

In medical surgery, a successful surgical operation does not always guarantee healing of the treated disease. The same is true of endodontics; proper treatment does not always mean long-term success. For this reason, just taking an immediate postoperative radiograph does not mark the end of treatment. Treatment success is established by long-term postoperative observation.

Radiographs may reveal three different situations: failure of the treatment performed, resolution of the periradicular lesion (bone restored to its original condition), or "scarring" of the periradicular radiolucency (presence of some remnants of periradicular radiolucency) (Figures 34-4, 34-6, & 34-7). Regarding the latter, in some situations, where periradicular radiolucencies are extensive, their radiographic size diminishes after endodontic treatment. However, asymptomatic radiolucent area may persist.[12,13] More prolonged clinical and radiographic observation is needed in such cases.

Relationship Between Immediate Postoperative Pain and Long-Term Success

Once endodontic treatment has been completed, the clinician should monitor the patient during the days following treatment since spontaneous painful symptoms may be present for a few days. Sensitivity to chewing and percussion is common and may last for several days.[14] Administration of analgesics and anti-inflammatory agents is prescribed. When

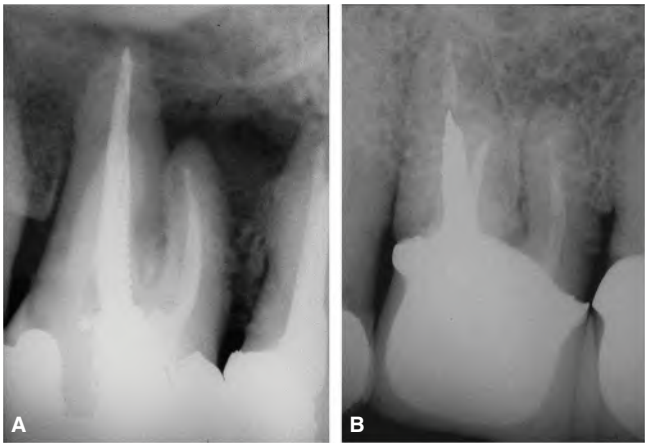

FIGURE 34-6 Long-term follow-up of nonsurgical endodontic treatment in a maxillary first molar of a 28-year-old male. ***A.*** Immediate postoperative radiograph showing a periapical radiolucency associated with the mesiobucal root. ***B.*** 35-year follow-up radiograph showing marked reduction in size of the radiolucency. This may indicate a possible formation of apical scar.

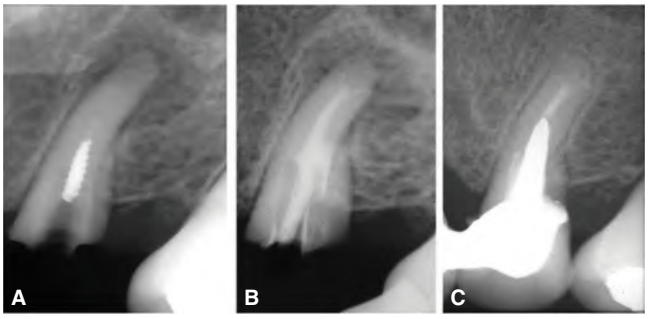

FIGURE 34-7 Resolution of a periradicular lesion. Long-term follow-up of nonsurgical endodontic retreatment in a maxillary second premolar of a 30-year-old female. Patient sought treatment after the prosthetic restoration on the tooth fractured. ***A.*** Preoperative radiograph showing a large periapical radiolucency associated with the tooth. Deficient endodontic treatment and a separated screw post is observed. ***B.*** Immediate postoperative radiograph. Complex apical anatomy is demonstrated by sealer extrusion (arrows). ***C.*** 11-year follow-up radiograph showing resolution of the periapical radiolucency. The tooth is asymptomatic and serves as abutment for fixed prosthesis.

the pulp tissue is necrotic, with or without a periradicular lesion, the painful reaction can be more intense and may be accompanied by localized or diffuse swelling. If systemic signs and symptoms are present (flare-up), it is advisable to prescribe an appropriate antibiotic in addition to analgesics and anti-inflammatory agents.[15,16] For more details, see Chapter 30.

If the endodontic treatment has been properly carried out, the symptoms are bound to clear up in a few days. In very few cases, despite having carried out successful treatment, painful symptoms may persist for a longer period without any visible radiographic changes.[17] In general, it is desirable to avoid an immediate postoperative reaction, since it has no bearing on the long-term prognosis of the treatment. Since endodontic treatment is a surgical procedure by nature, it may give rise to a postoperative inflammatory reaction expressed with a range of symptoms.

Long-Term Assessment of Root Canal Treatment

Teeth With Vital Pulps

When a tooth must undergo pulpectomy, the inflammatory process generally affects only the pulp without radiographic evidence of periradicular involvement. In such cases, endodontic therapy will involve removal of the pulp, cleaning and shaping the root canal system, followed by obturation. Proper endodontic procedure sets out to maintain the health of the periradicular tissues and the long-term radiographic periradicular image should therefore be similar to the immediate postoperative image. Many practitioners carry out this treatment in a single visit. Vital pulpectomy success rate is greater than 94% (Figure 34-8).[18–20]

Teeth With Necrotic Pulps

In endodontic treatments involving necrotic pulps, disinfection is the most important additional step prior to obturation. In such cases, microorganisms are present in the root canal system and may penetrate deeply into the dentin through the dentinal tubules. Therefore the therapeutic process must be more thorough. Endodontic treatment may require more than one visit as well as intracanal dressings with medication between visits.

Radiographically, the periradicular area may or may not be involved. Treatment of this condition can be based on whether the tooth has periradicular periodontitis or not.

a. Teeth with necrotic pulps without periradicular periodontitis

   Several publications indicate that long-term success rates of such cases ranges between 86% and 95%.[18–22] Results depend on various factors: anatomical difficulty, skill of the practitioner (students, graduates and specialists), intraoperative challenges, and long-term observation time. (Figure 34-9).

b. Teeth with necrotic pulps and periradicular periodontitis

   Periradicular periodontitis is considered to be present when a radiograph shows a circumscribed or diffuse radiolucency surrounding, or at the side of, the tooth apex. This means the inflammatory process has developed in the periradicular tissues, connected to the root canal system through the apical foramen or lateral foramina. In such cases, the success rate of endodontic treatment ranges between 80% to 86% (Figure 34-10).[18,20–23]

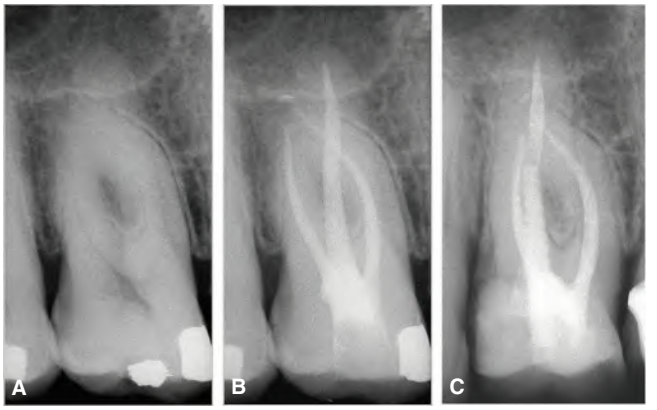

FIGURE 34-8 Nonsurgical endodontic treatment in a maxillary first molar. Patient presented with acute clinical symptoms of pulpitis. ***A.*** Preoperative radiograph. ***B.*** Immediate postoperative radiograph. ***C.*** Five-year follow-up radiograph. The tooth is asymptomatic and has been restored to function.

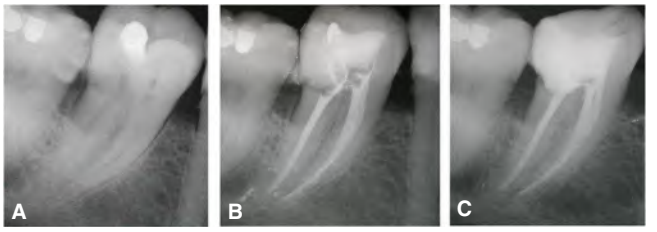

FIGURE 34-9 Nonsurgical endodontic treatment following pulp necrosis in a mandibular second molar of a 32-year-old male. ***A.*** Preoperative radiograph showing occlusal and distal coronal restorations. ***B.*** Immediate postoperative radiograph. ***C.*** Five-year follow-up radiograph showing normal appearance of the periradicular tissues.

There is some disagreement over the relationship between long-term success rate and the number of treatment sessions.[24–27] In general, it may be assumed that if the treatment was properly done, there is no significant difference between both procedures.[28–34] Practitioners who perform endodontic treatment in more than one visit do so to increase disinfection by using an intracanal antimicrobial dressing between visits. In specific cases such as intracanal bleeding, persistent canal exudate, severe preoperative pain and acute clinical symptoms, the use of an intracanal dressing is indicated and multiple-visit treatment is preferred.

When patients of different age and sex were compared, the success rates were similar.[23,35] However, in cases of very young patients presenting with immature apices with pulp necrosis, with or without periradicular involvement, current therapeutic procedures can vary. These range from apexification to endodontic regeneration. Long-term outcome assessment of such cases requires frequent monitoring and follow-up over longer periods of time.

To summarize, the prognosis of endodontic therapy in teeth with vital pulps is similar to that of teeth with necrotic pulps without periradicular periodontitis. Both conditions offer a better prognosis than treatment of teeth with necrotic pulps and periradicular periodontitis.

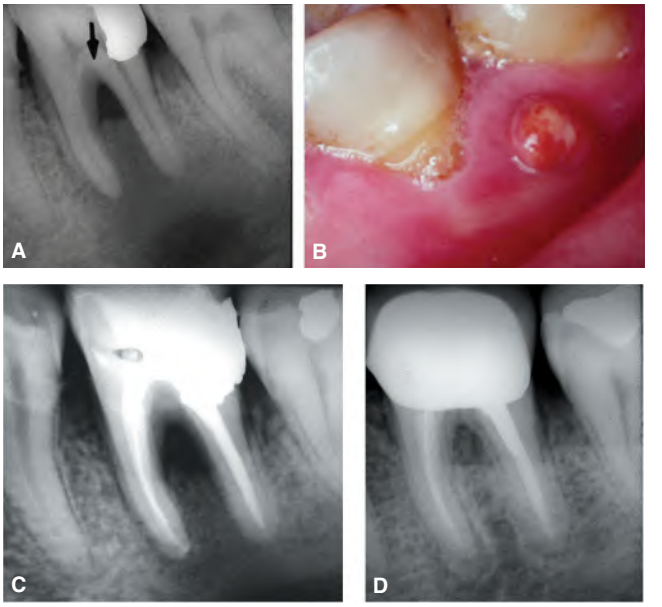

FIGURE 34-10 Nonsurgical endodontic treatment in a mandibular first molar of a 56-year-old male. ***A.*** Preoperative radiograph showing a large periradicular radiolucency extending to the furcation region (arrow). Pulp vitality tests were negative. ***B.*** Clinical photograph showing a sinus tract associated with the affected tooth. ***C.*** Immediate postoperative radiograph. ***D.*** 10-year follow-up radiograph showing resolution of the periradicular radiolucency. The patient was asymptomatic and the tooth restored to function.

Non-Surgical Retreatment (Reintervention)

During orthograde endodontic retreatment, complete removal of previous filling material is crucial, in order to enable new cleaning, shaping, disinfection, and filling of the root canal system.

Several studies indicated a rise in retreatments in endodontic practice.[36,37] Unfortunately, many general practitioners now prefer extraction and implant placement to endodontic retreatment.[2] Numerous publications on procedures performed in different countries indicate that the reason for failure of endodontic therapy is often the low quality of endodontic treatments rendered by general practitioners.[38–43]

The reported success rate obtained with endodontic retreatment varied from 62% to 95% (Figure 34-11).[9,18,20,23,44–48] This wide range is generally due to factors related to the anatomical difficulty of each clinical case, size and type of periradicular lesions, therapeutic procedure performed during primary treatment, postoperative observation periods used in each case, skill of the operator, and the lack of calibration in the studies.

Retreatment in teeth with periradicular radiolucency showed a 13% to 20% lower success rate than those teeth without periradicular pathosis.[18,23,47,48] Gorni and Gagliani[49] reported a success rate of 86.8% when the morphology of treated teeth was not altered as a result of previous treatment, and 47% in cases where the tooth anatomy had been changed. This clearly shows that the prognosis of

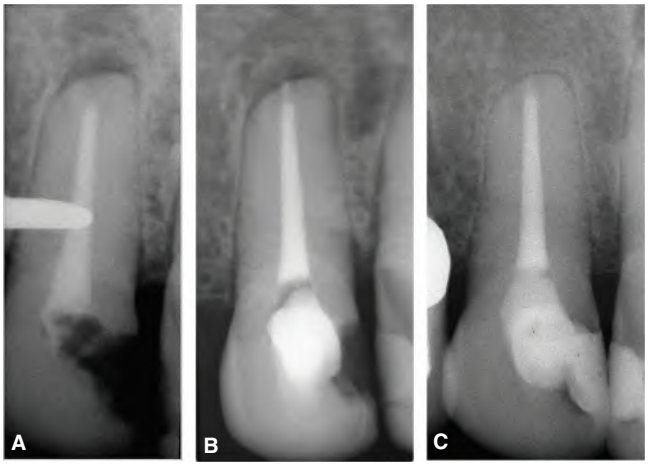

FIGURE 34-11 Nonsurgical endodontic retreatment in a maxillary lateral incisor of a 28-year-old female. The tooth was symptomatic on percussion. ***A.*** Preoperative radiograph showing periradicular radiolucency associated with the affected tooth. The coronal obturation is missing and the pulp chamber is open to the oral cavity. ***B.*** Immediate postoperative radiograph. ***C.*** 17-year follow-up radiograph showing resolution of the periradicular radiolucency. The tooth was asymptomatic throughout the entire follow-up period.

retreatment is linked not only to the presence of disease but also to changes induced in the root canal due to the previous practitioner's actions (blockages, broken instruments, perforations, transportations and other mishaps).

In general, if it is possible, in retreatment, to reach the apical foramen and remove the primary obturation so that proper cleaning, shaping and refilling of the root canal can be achieved, the outcome is no different from that achieved in cases of initial endodontic treatment of teeth with pulp necrosis.

Root Integrity

Prior to endodontic treatment, it is important to observe root integrity of teeth to be treated following radiographic diagnosis. This step is particularly important in teeth that have suffered traumatic injuries. The existence of internal resorptions communicating with the periodontal tissue as well as external resorptions can have an impact on the prognosis (Figure 34-12). In some cases, root resorptions are not radiographically visible, either because they are too small or because they are located on either the buccal, lingual, or palatal root surfaces superimposed over the root on the radiographic image.[50] In both situations, depending on the location, extent and rate of resorption, prognosis is uncertain, and occasionally additional surgery is required. For more details, see Chapter 15.

When considering root integrity, root perforation is another clinical challenge. The use of enhanced illumination and magnification, as well as appropriate materials with efficient sealing capacity such as Mineral Trioxide Aggregate (MTA), are positive factors that considerably increase the likelihood of a favorable prognosis (Figure 34-13).[51,52]

Perforation location, size, origin, and the time between occurrence and treatment must be considered when evaluating

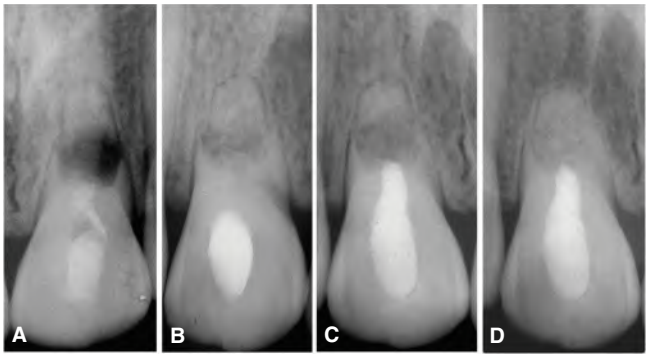

FIGURE 34-12 Nonsurgical endodontic retreatment in a traumatized maxillary central incisor of an 18-year-old male. On examination, the tooth was mobile and symptomatic on biting. ***A.*** Preoperative radiograph showing a large middle root radiolucency. Extensive root defect associated with the affected tooth is observed. ***B.*** Radiograph showing calcium hydroxide intracanal medication in place. ***C.*** Immediate postoperative radiograph. The root canal was obturated with gutta-percha and sealer. ***D.*** Three-year follow-up radiograph showing resolution of the radiolucency. The tooth is asymptomatic and mobility restored to normal.

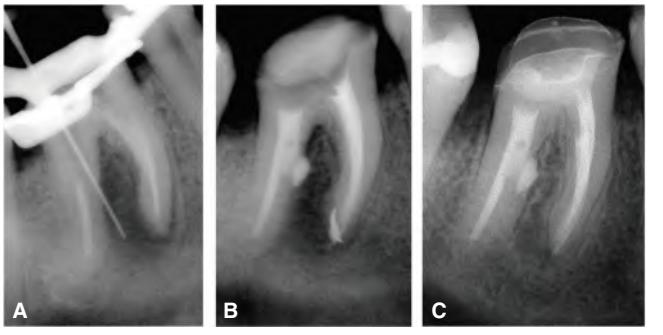

FIGURE 34-13 Nonsurgical endodontic retreatment in a perforated mandibular first molar of a 38-year-old female. ***A.*** Preoperative radiograph showing a large interradicular radiolucency. An endodontic instrument demonstrates the area of perforation in the distal root caused by inadequate post placement. ***B.*** Immediate postoperative radiograph. Endodontic retreatment was done and the perforation sealed with Mineral Trioxide Aggregate (MTA). ***C.*** Three-year follow-up radiograph showing resolution of the radiolucency. The tooth is asymptomatic.

prognosis. Large perforations with a high degree of contamination will yield unfavorable prognosis and often surgical treatment is required.

Endodontic Surgery

Endodontic surgery is not oral surgery in the traditional sense. Rather, it is endodontic treatment through a surgical flap.[53] In some clinical situations, periradicular surgery is the treatment of choice. Possible indications include failed retreatments, cystic lesions, perforations in the middle or apical third of the root, separated instruments that are difficult or unfeasible to remove, and canals that are inaccessible due to calcifications or obstructions (Figure 34-14).

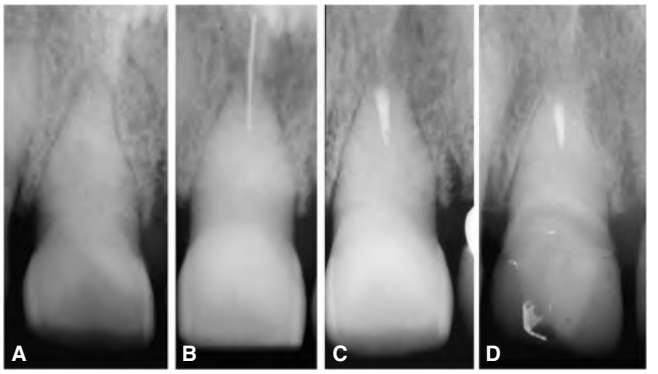

FIGURE 34-14 Surgical endodontic treatment in a maxillary central incisor of a 42-year-old female. The patient reported spontaneous pain in this tooth. ***A.*** Preoperative radiograph showing evidence of a calcified root canal system. ***B.*** Intraoperative radiograph during retro-instrumentation of the accessible portion of the root canal. ***C.*** Immediate postoperative radiograph showing the retro-obturation of the root canal with thermoplasticized gutta-percha and sealer. ***D.*** 18-year follow-up. Periradicular normalcy can be observed.

In recent years, advances in materials and technology contributed to the high success rate of endodontic surgery.[54–58] For more details, see Chapter 24. In this sense, retrograde instrumentation and obturation of the entire untreated root canal increases the chance of success insofar as it meets requirements for cleaning and complete filling of the contaminated endodontic space and closing off communications between the root canal and the periodontal ligament[59,60] (Figure 34-15).

FUNCTIONAL RETENTION

As mentioned earlier, the percentage success rate of nonsurgical and surgical endodontic treatment and retreatment is around 85%. If we now consider the concept of success in terms of functional retention or survival, this average rises to more than 90%.[35,42,48,60–63]

TIME FACTOR IN LONG-TERM OBSERVATION

There is considerable disagreement over the long-term postoperative observation time necessary to consider the outcome of endodontic treatment. The minimum acceptable time is generally considered to be at least 4 to 5 years.[22,28,45,60,64]

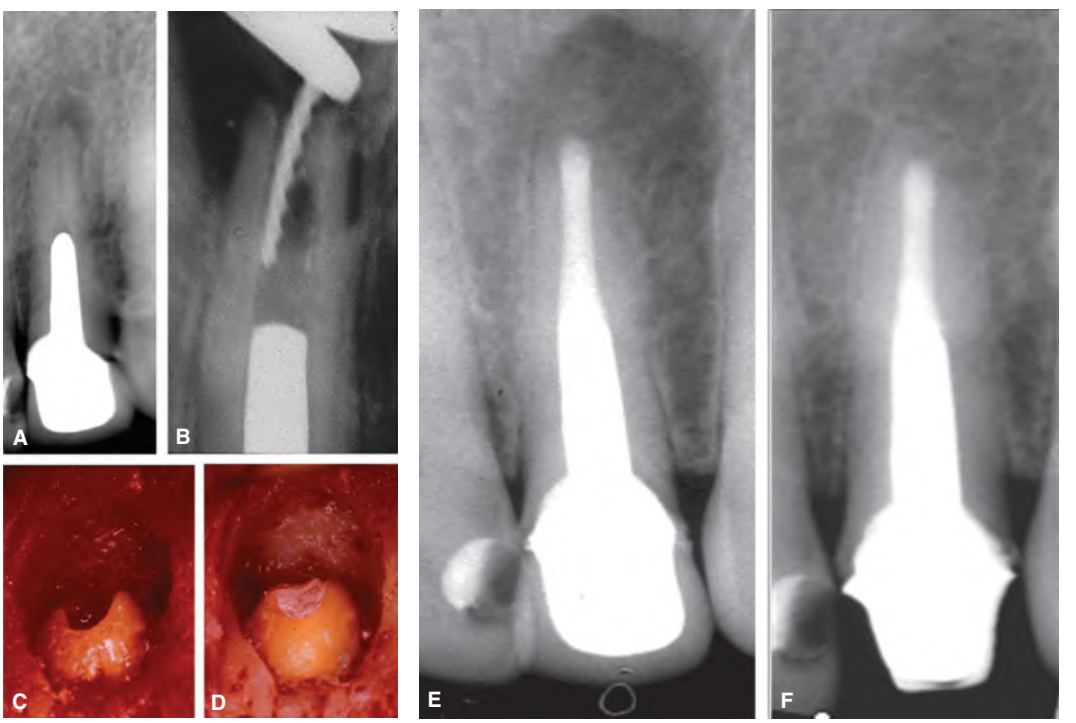

FIGURE 34-15 Surgical endodontic retreatment in a maxillary lateral incisor of a 38-year-old female. ***A.*** Preoperative radiograph showing a periapical radiolucency associated with the tooth. A large post and a crown are present. There is no evidence that the remaining root canal space was treated. The patient reported intermittent symptoms associated with this tooth. A decision was made to keep the post in place, reflect a buccal flap, and approach the remainder of the root canal space from the apical end. ***B.*** Radiograph showing endodontic instrument inserted from the apex. ***C.*** Clinical photograph following instrumentation of the root canal space. ***D.*** Clinical photograph following retro-obturation of the root canal space with thermoplasticized gutta-percha and sealer. ***E.*** Immediate postoperative radiograph. The root canal space was retro-obturated to the post level. ***F.*** 17-year follow-up radiograph. The periapical radiolucency has resolved and the patient is asymptomatic.

Increasing postoperative monitoring time increased the success rate observed in cases of initial endodontic treatment as well as in cases of non-surgical endodontic retreatment (Figure 34-16).[46,60,65] If, during the observation period following endodontic treatment, the tooth is asymptomatic and periradicular radiolucency decreases, it is appropriate to continue monitoring it for a longer period.

Effect of Overfills on Long-Term Prognosis

Overfilling may occur during obturation of non-surgical endodontic treatment or retreatment. In such cases, sealer may extrude laterally (via a lateral portal of exit) or apically (via the apical foramina) (Figures 34-17 & 34-18). The overfill may also result in tiny particles of sealer difficult to identify on the radiograph, that can be mistaken with normal periradicular bone.[23,66–68]

During instrumentation and obturation, contaminated dentin particles may sometimes be projected through the apical foramen together with necrotic organic debris. These are not visible on the radiograph and compose part of the overfilling along with the extruded sealer. Many reactions attributed to overfilled sealer may actually be caused by extruded contaminated material not detectable on the radiograph.

The relationship between overfilling and long-term prognosis of endodontic treatment is controversial. While some believe it reduces the long-term success rate, others claim that it has no effect.[18,21,23,46,47,65,67–70]

The quantity of overfilled material, the type of material, its consistency, and the host's reaction to the material are important considerations. In situations where the overfills are small and involve conventional sealers, the healing may be delayed but not impaired (Figures 34-19).[46,65,70] Under certain circumstances, the overfilled material is fully reabsorbed over time, while in other situations it remains embedded in the tissues without compromising the long-term treatment success. Likewise, during retreatment, fragments of gutta-percha can extrude to periapical tissues via the apical foramen. In some cases, the material may provoke a foreign body reaction, while in others, the material doesn't cause any adverse reaction and is reabsorbed after a period of time (Figure 34-20). In these cases, clinical and radiographic

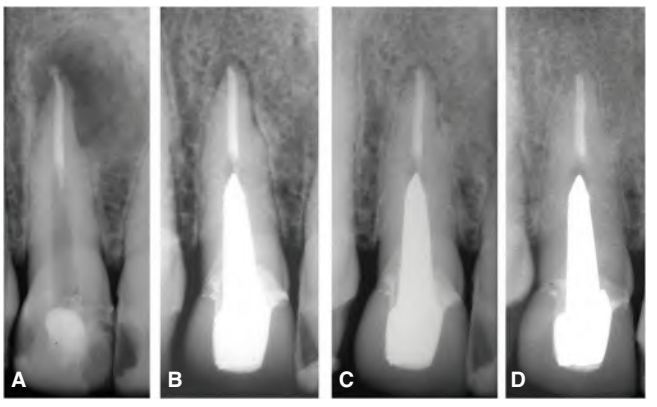

FIGURE 34-16 Endodontic treatment in a maxillary lateral incisor of a 32-year-old female. The patient presented with severe irradiating pain. ***A.*** Immediate postoperative radiograph showing periapical radiolucency associated with the affected tooth. ***B.*** Six-year follow-up radiograph showing marked reduction of periapical radiolucency, however, without complete resolution. The patient is asymptomatic. ***C.*** 10-year follow-up radiograph showing further reduction in size of the periapical radiolucency. The patient is still asymptomatic. ***D.*** 25-year follow-up radiograph. The periapical radiolucency has completely resolved.

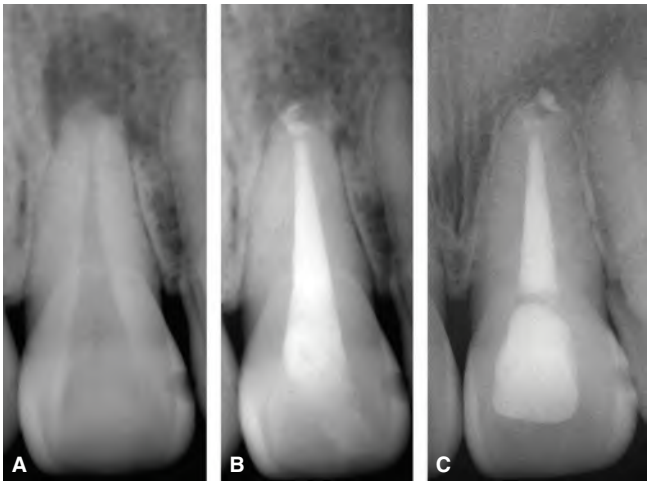

FIGURE 34-17 Endodontic treatment in a maxillary central incisor of a 25-year-old female. The patient reported to have suffered a traumatic injury to the tooth several years earlier. ***A.*** Preoperative radiograph showing periapical radiolucency and apical root resorption. ***B.*** Immediate postoperative radiograph. ***C.*** 17-year follow-up radiograph. Evidence of sealer remnants can still be observed. The periapical radiolucency has resolved and the tooth restored to function.

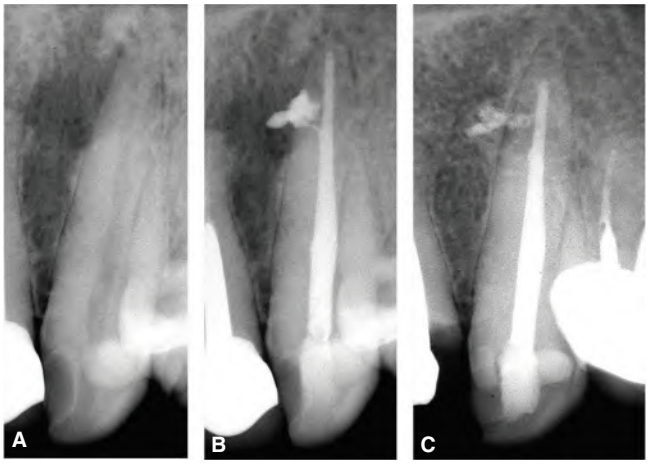

FIGURE 34-18 Endodontic treatment in a maxillary canine of a 42-year-old male. ***A.*** Preoperative radiograph showing a lateral radiolucency between the middle and apical third of the root. Vitality tests were negative. ***B.*** Immediate postoperative radiograph. Sealer extruding through a lateral portal of exit is evident. ***C.*** 12-year follow-up radiograph. The lateral radiolucency has resolved. Evidence of sealer remnants is still observed. The patient is asymptomatic and the tooth restored to function.

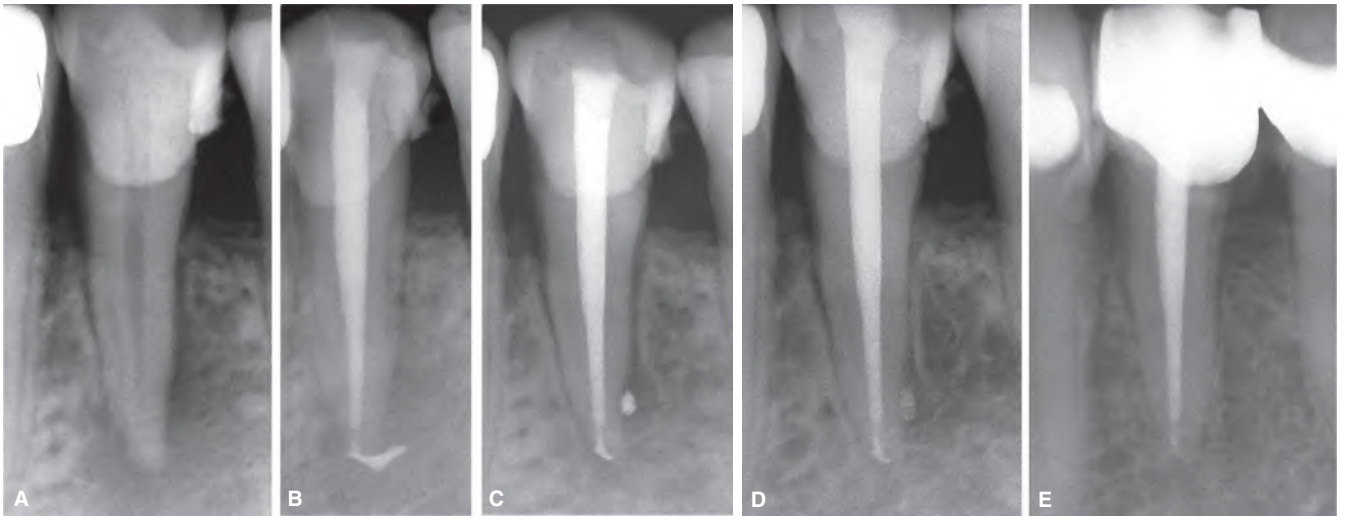

FIGURE 34-19 Endodontic treatment in a mandibular first premolar of a 41-year-old male. The patient reported severe pain associated with the tooth. ***A.*** Preoperative radiograph showing large periradicular radiolucency associated with the affected tooth. Vitality tests were negative. ***B.*** Immediate postoperative radiograph. ***C.*** Six-month follow-up showing marked reduction in size of periapical radiolucency. Persistent radiolucency surrounding sealer remnants still noticeable. ***D.*** Two-year follow-up radiograph. Further reduction in size of the periapical radiolucency is evident. The extruded sealer is less apparent. ***E.*** 11-year follow-up radiograph. The periapical radiolucency has resolved and the extruded sealer has disintegrated. The patient is asymptomatic and the tooth restored to function.

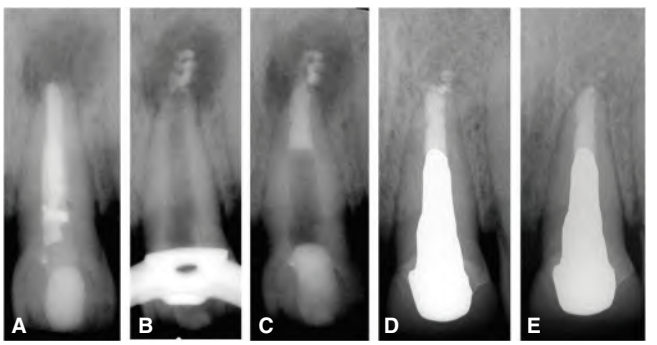

FIGURE 34-20 Endodontic retreatment in a maxillary central incisor of a 28-year-old male. The patient reported to have suffered traumatic injury to the tooth in childhood. ***A.*** Preoperative radiograph showing periapical radiolucency associated with the affected tooth. The patient was asymptomatic. ***B.*** Radiograph taken after removal of the previous root canal filling. Remnants of old filling material can be detected in the periapical area. ***C.*** Immediate postoperative radiograph. The apical part of the root canal was filled with MTA. ***D.*** Six-year follow-up radiograph showing marked reduction in size of the periapical radiolucency and in the amount of the extruded material. ***E.*** 10-year follow-up radiograph. Periapical radiolucency has resolved and extruded material almost completely reabsorbed.

observation must be extended for a longer period of time to assess of the final clinical approach to be taken. For gross overfills, the final outcome is less certain and surgical removal of the extruded material may be necessary.

Reasons for Failure of Endodontic Treatment

In some cases, in spite of correct diagnosis and treatment procedures, a non-surgical endodontic treatment or re-treatment may still fail. Persistence of periradicular periodontitis can be due to microbial or non-microbial reasons. The former includes inadequate removal of intraradicular infection during operating procedures, extracanal endodontic infection, and possible coronal leakage from an inadequate restoration.[13,21,40,41,43,68,70–79] The latter includes deposits of cholesterol crystals in periradicular lesions, true cysts, and reactions to foreign bodies due to the extrusion of various materials into the periradicular area.[13,70,79–82] Likewise, the presence of radicular cracks or fractures not visible on the radiograph, extensive root resorption, and severe endodontic-periodontic disease can also be the cause of failure.

Deficient elimination of the septic contents of the root canal system is generally due to the presence of anatomical complexities not readily accessible, inadequate canal cleaning, disinfection and filling procedures, intraoperative accidents, and missed canals. In such situations, non-surgical or surgical retreatment will be the preferred treatment once the cause of failure has been identified.

Deciding on tooth extraction after endodontic treatment is more likely to occur for periodontal or prosthetic causes rather than failure of root canal treatment.[83]

Differential diagnosis is important since some anatomical structures and pathologic conditions may appear on the radiograph to be associated with the radicular area and therefore be improperly diagnosed as endodontic in origin.[84] Under such circumstances, computed tomography is a useful diagnostic tool. For more details, see Chapters 9C & 10.

During endodontic therapy, unnecessary removal of tooth structure must be avoided to prevent potential fracture of the treated tooth. Endodontic success would then become a dental failure. An important factor to consider when assessing the prognosis of endodontic treatment is coronal reconstruction. Coronal leakage, over time, constitutes a reason for endodontic failure. Although the role of coronal filtration is debatable, a satisfactory restoration is undoubtedly necessary to guarantee functional and esthetic success.[21,23,41,85,86] A permanent restoration must therefore be placed as soon as possible in order to reinforce the tooth and prevent microbial invasion.

Lastly, when considering reasons for endodontic treatment failure, one cannot ignore the role of systemic conditions of the patient. The prognosis must be considered doubtful in patients with uncontrolled diabetes or severe immuno-compromised diseases.[60]

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CHAPTER 35

Contemporary Restoration of Endodontically Treated Teeth

NADIM Z. BABA, CHARLES J. GOODACRE

Various methods of restoring pulpless teeth have been reported for more than 200 years. In 1747 Pierre Fauchard[1] described the process by which roots of maxillary anterior teeth were used for the restoration of single teeth and the replacement of multiple teeth (Figure 35-1). Posts were fabricated of gold or silver and held in the root canal space with a heat-softened adhesive called "mastic."[1,2] The longevity of restorations made using this technique was attested to by Fauchard: "Teeth and artificial dentures, fastened with posts and gold wire, hold better than all others. They sometimes last fifteen to twenty years and even more without displacement. Common thread and silk, used ordinarily to attach all kinds of teeth or artificial pieces, do not last long."[1]

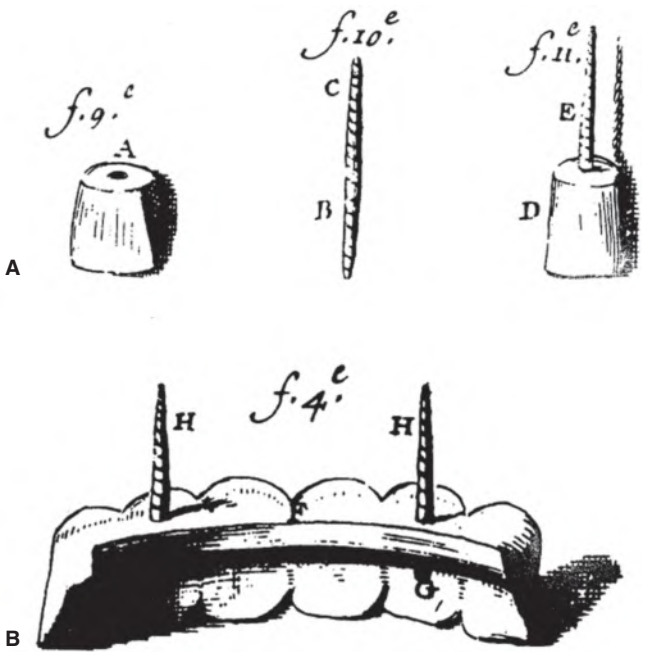


FIGURE 35-1 Early attempts to restore single or multiple units. ***A.*** "Pivottooth" consisting of crown, post, and assembled unit. ***B.*** Six-unit anterior fixed partial dental prosthesis "pivoted" in lateral incisors with canines cantilevered. Crowns were fashioned from a diversity of materials. Human, hippopotamus, sea horse, and ox teeth were used, as well as ivory and oxen leg bones. Posts were usually made from precious metals and fastened to the crown and root by using a heated sticky "mastic" prepared by gum, lac, turpentine, and white coral powder.

The replacement crowns were made from bone, ivory, animal teeth, and sound natural tooth crowns. Gradually the use of these natural substances declined, to be slowly replaced by porcelain. A pivot (what is today termed a post) was used to retain the artificial porcelain crown into a root canal and the crown-post combination was termed a "pivot crown." Porcelain pivot crowns were described in the early 1800's by a well-known dentist of Paris, Dubois de Chemant.[2] Pivoting (posting) of artificial crowns to natural roots became the most common method of replacing artificial teeth, and was reported as the "best that can be employed" by Chapin Harris in *The Dental Art* in 1839.[3]

Early pivot crowns in the United States used seasoned wood (white hickory) pivots.[4] The pivot was adapted to the inside of an all-ceramic crown and also into the root canal space. Moisture would swell the wood and retain the pivot in place.[2] Surprisingly, Prothero[2] reported removing two central incisor crowns with wooden pivots that had been successfully used for 18 years. Subsequently, pivot crowns were fabricated using wood/metal combinations and then more durable all-metal pivots were used. Metal pivot retention was achieved by various means such as threads, pins, surface roughening, and split designs that provided mechanical spring retention.[2]

Unfortunately, adequate cements were not available to these early practitioners, cements that would have enhanced post retention and decreased abrasion of the root caused by movement of metal posts within the canal. One of the best representations of a pivoted tooth appears in *Dental Physiology and Surgery*, written by Sir John Tomes in 1849 (Figure 35-2).[5] Tomes's post length and diameter conformed closely to today's principles in fabricating posts.

Endodontic therapy by these dental pioneers embraced only minimal efforts to clean, shape, and obturate the canal. Frequent use of the wood posts in empty canals led to repeated episodes of swelling and pain. Wood posts, however, did allow the escape of the so-called "morbid humors." A groove in the post or root canal provided a pathway for continual suppuration from the periradicular tissues.[1]

Although many of the restorative techniques used today had their inception in the 1800's and early 1900's, **proper**

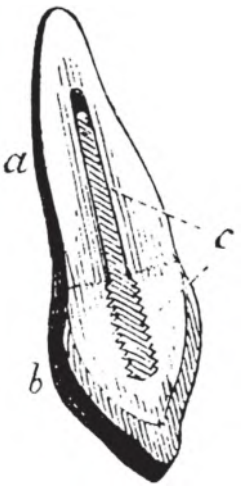


FIGURE 35-2 Principles used today in selecting post length and diameter were understood and taught by early practitioners during mid-1800s.

endodontic treatment was neglected until years later. Today, both the endodontic and prosthodontic aspects of treatment have advanced significantly, new materials and techniques have been developed, and a substantial body of scientific knowledge is available upon which to base clinical treatment decisions.

This chapter describes the techniques commonly employed when restoring endodontically treated teeth and answers frequently associated questions. Whenever possible, the answers and discussion will be supported by scientific evidence. Conflicting results will be presented to provide a comprehensive understanding of the available evidence.

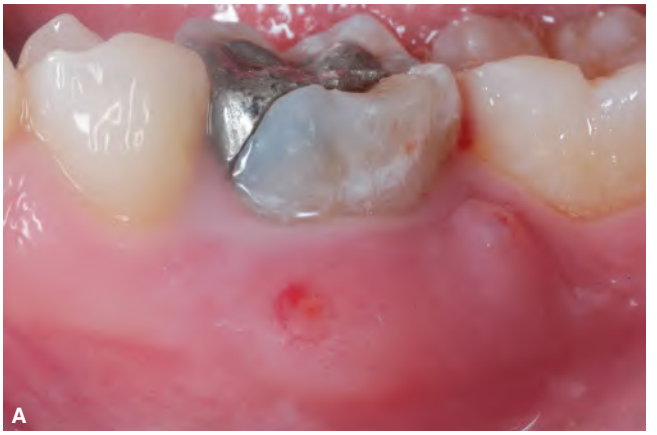
SHOULD CROWNS BE PLACED ON ENDODONTICALLY TREATED TEETH?

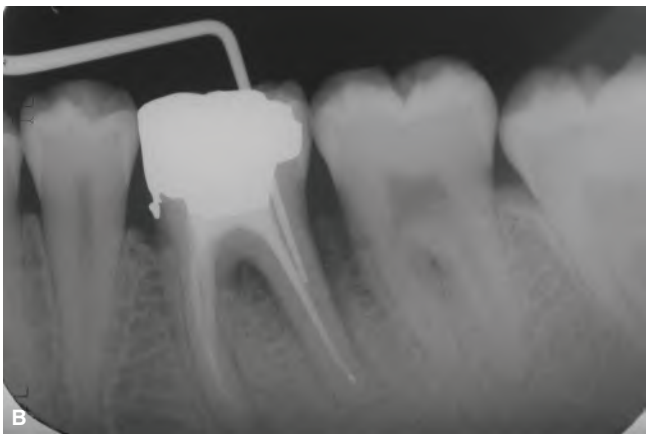
A retrospective study[6] of 1,273 teeth endodontically treated 1 to 25 years previously compared the clinical success of anterior and posterior teeth. Endodontically treated teeth with restorations that encompassed the tooth (onlays, partial or complete coverage metal crowns, and metal ceramic crowns) were compared with endodontically treated teeth with no coronal coverage restorations. It was determined that in **anterior teeth**, **coronal coverage crowns did not significantly improve the success of endodontically treated teeth**. This finding supports the use of a conservative restoration such as an etched resin restoration in the access opening of otherwise intact or minimally restored anterior teeth. Crowns are only indicated on endodontically treated anterior teeth when they are structurally weakened by the presence of large and/or multiple coronal restorations or they require significant form/color changes that cannot be effected by bleaching, resin bonding, or porcelain laminate veneers. Scurria and colleagues[7] collected data from 30 insurance carriers in 45 states regarding the procedures 654 general dentists performed on endodontically treated teeth. The data indicated that 67% of endodontically treated anterior teeth were restored without a crown, supporting the concept that many anterior teeth are being satisfactorily restored without the use of a crown.

When endodontically treated **posterior teeth** (with and without coronal coverage restorations) were compared, **a significant increase in the clinical success** was noted when **cuspal coverage crowns** were placed on maxillary and mandibular molars and premolars.[6] In a study of 116 failed and extracted endodontically treated teeth, Vire[8] reported that teeth restored with crowns had greater longevity than uncrowned teeth. A strong association was found between crown placement and the survival of endodontically treated teeth. Endodontically treated teeth (ETT) with crowns were lost after an average of 87 months whereas ETT without crowns were lost after an average of 50 months. If long-term tooth survival is the primary goal, placing a crown on an endodontically treated posterior tooth enhances survival (Figure 35-3).[9] Aquilino and colleagues[9] reported that ETT with cuspal coverage had six times greater rate of survival than those without cuspal coverage. Therefore, restorations that encompass the cusps should be used on posterior teeth that have intercuspation with opposing teeth and thereby receive occlusal forces that push the cusps apart.

Salehrabi and colleagues[10] performed an epidemiological study in a large patient population and reported that 85% of the teeth that were extracted (3%) after initial non-surgical endodontic treatment, had no full coronal coverage. A prospective study by Ng and colleagues[11] evaluated the factors affecting outcomes of non-surgical RCT. They found that ETT that were not restored with a crown had a worse survival rate than those that were. In a systematic review, Stavropolou and colleagues[12] concluded that ETT restored with crowns had a higher long-term survival rate compared to those not restored with crowns. The previously discussed insurance data[7] indicated that 37% to 40% of posterior endodontically treated teeth were restored by practitioners without a crown, a method of treatment not supported by the long-term clinical prognosis of posterior endodontically treated teeth that do not have cusp-encompassing crowns.

There are, however, certain posterior teeth (not as high as 40%) that do not have substantive occlusal intercuspation or have an occlusal form that precludes intercuspation of a nature that attempts to separate the cusps (such as mandibular first premolars with small, poorly developed lingual cusps). When these teeth are intact or minimally restored (small MO or DO restorations), they would be reasonable candidates for restoration of only the access opening without use of a coronal coverage crown.[13] However, the survival rate of ETT will likely be lower when ETT have lost excessive amounts of tooth structure. In a recent systematic review, Federowicz and colleagues[14] looked at the effects of the restoration of ETT by crowns versus conventional filling materials. They concluded that there is no evidence to support or refute the effectiveness of crowns over filling materials for the restoration of ETT. However, more clinical data are required to determine the long-term survival of these teeth when large composite resin restorations are present in conjunction of heavy wear, heavy forces, or parafunctional habits. ETT that have been previously restored and weakened by





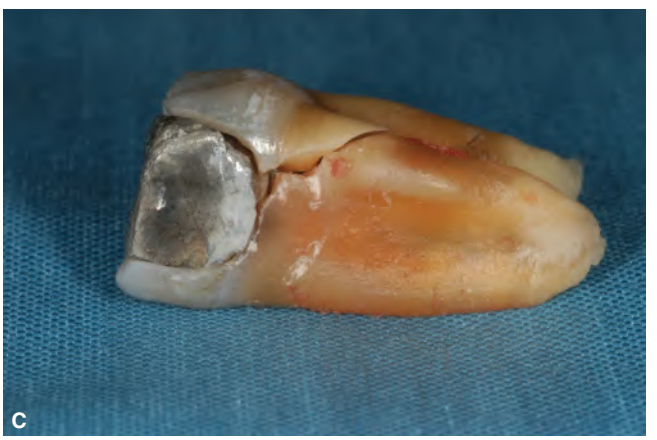


FIGURE 35-3 ***A.*** Buccal view of an endodontically treated mandibular left first molar without cuspal coverage reveals a dental fistula. ***B.*** Pre-operative radiograph showing a deep, isolated, and narrow periodontal pocket. ***C.*** Extracted tooth showing the fracture extending down both roots. (Reprinted with permission from Baba NZ.[331])

prior removal of tooth structure should be restored with a crown that encompasses the cusps.

In contrast to the above recommendations, a 3-year clinical study by Manocci and colleagues[15] evaluated the clinical success rate of endodontically treated premolars restored with a post and direct composite resin restorations with and without complete crown coverage. They found that both had a similar success rate. Nagasiri and colleagues,[16] in a retrospective cohort study, indicated that when endodontically treated molars are completely intact except for a conservative access opening, they could be restored successfully by using composite resin restorations. They found that the 5-year survival rate of teeth without cuspal coverage was 36%.

Multiple clinical studies of fixed partial dentures, many with long spans and cantilevers, have determined that endodontically treated abutments failed more often than vital teeth due to tooth fracture,[17–21] supporting the greater fragility of endodontically treated teeth and the need to design restorations that reduce the potential for both crown and root fractures when extensive fixed prosthodontic treatment is required.

The high demand for esthetic restorations, the desire for conservative treatment and the benefit of covering the cusps caused some clinicians to use endocrowns.[22–24] This restoration consist of an onlay or crown with core material in a single unit. The core material engages in the cavity prepared into the pulp chamber. Endocrown fabricated of nanoceramic was found to have a favorable mode of failure when ETT, with no Build-up material, are used.[25,26] The authors found the endocrowns to be more resistant than teeth restored with a post, composite resin core, and a ceramic crown.

Gutmann[27] reviewed the literature and presented an overview of several articles that identify what happens when teeth are endodontically treated. These articles provide background information important to an understanding of why **coronal coverage crowns help prevent fractures of posterior teeth**. Endodontically treated dog teeth were found to have 9% less moisture than vital teeth.[28] In addition, it was found that dehydration increases stiffness and decreases the flexibility in teeth. However, dehydration by itself does not account for the physical property changes in dentin.[29] Yet in another investigation of 23 matched pairs of human ETT and their vital contralateral teeth, the moisture levels were not statistically significantly different.[30] In some teeth that had undergone root canal therapy as many as 15 to 20 years earlier, the moisture content was not necessarily reduced, even after extended periods of time. It has been stated that dehydration alone does not account for changes in physical properties of dentin.[29] Also, with aging, greater amounts of peritubular dentin are formed, which decreases the amount of organic materials that may contain moisture.

It has been shown that endodontic procedures reduce tooth stiffness by 5%, attributed primarily to the access opening.[31] Tidmarsh[32] described the structure of an intact tooth that permits deformation when loaded occlusally and elastic recovery after removal of the load. The direct relationship between tooth structure removed during tooth preparation and tooth deformation under load has been described.[33] Dentin from endodontically treated teeth has been shown to exhibit significantly lower shear strength and toughness than vital dentin.[34] Rivera and colleagues[35] stated that the effort required to fracture dentin may be less when teeth are endodontically treated because of more immature (potentially weaker) collagen intermolecular cross-links. In a recent study, it was found that collagen fibrils degraded over time

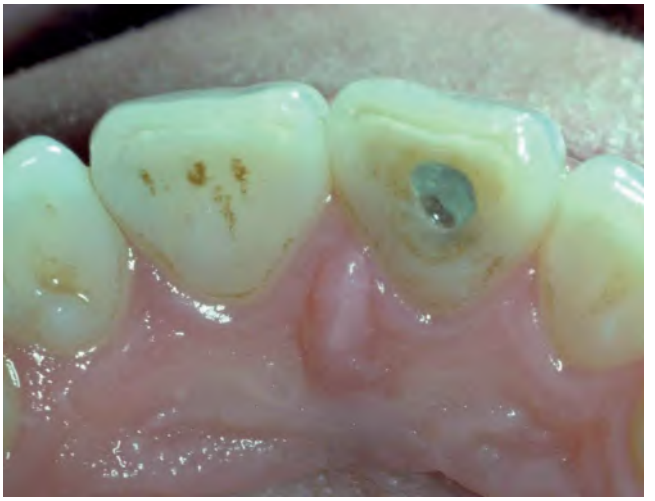


FIGURE 35-4 Traumatized tooth before endodontic procedure. Placement of a bonded resin restoration in the access opening after root canal treatment and whitening is the only treatment required since crowns do not enhance the longevity of anterior endodontically treated teeth. A crown will be indicated only when esthetic and functional needs cannot be achieved through more conservative treatments.

in teeth with zinc phosphate-cemented posts. Acid demineralization can also occur from bacteria and acid etching.[36]

CONCLUSIONS

Restorations that encompass the cusps of endodontically treated posterior teeth have been found to increase the clinical longevity of these teeth. Therefore, **crowns should be placed on endodontically treated posterior teeth that have occlusal intercuspation with opposing teeth** of the nature that places expansive forces on the cusps. Since **crowns do not enhance the clinical success of anterior endodontically treated teeth**, their use on relatively sound teeth should be limited to situations where esthetic and functional requirements cannot be adequately achieved by other more conservative restorations (Figure 35-4).

WITH PULPLESS TEETH, DO POSTS IMPROVE LONG-TERM CLINICAL PROGNOSIS OR ENHANCE STRENGTH?

Historically, the use of posts was based on the concept that a post reinforces the tooth.

Laboratory Data

Virtually all laboratory studies[37–47] have shown that placement of a post and core either fails to increase the fracture resistance of endodontically treated teeth or decreases the fracture resistance of the tooth when a force is applied via a mechanical testing machine. Lovdahl and Nicholls[37] found that endodontically treated maxillary central incisors were stronger when the natural crown was intact, except for the access opening, than when they were restored with cast posts and cores or pin-retained amalgams. Lu[38] found that posts placed in intact endodontically treated central incisors did not lead to an increase in the force required to fracture the tooth or in the position and angulation of the fracture line. Pontius[39] found that maxillary incisors, without posts, resisted higher failure loads than the other groups with posts and crowns. Gluskin[40] found that mandibular incisors with intact natural crowns exhibited greater resistance to transverse loads than teeth with posts and cores. McDonald[41] found no difference in the impact fracture resistance of mandibular incisors with or without posts. Eshelman and Sayegh[42] reported similar results when posts were placed in extracted dog lateral incisors. Guzy and Nicholls[43] determined that there was no significant reinforcement achieved by cementing a post into an endodontically treated tooth that was intact except for the access opening. Leary and colleagues[44] measured the root deflection of endodontically treated teeth before and after posts of various lengths were cemented into prepared root canals. They found no significant differences in strength between the teeth with or without a post. Trope and colleagues[45] determined that preparing a post space weakened endodontically treated teeth compared with ones in which only an access opening was made, but no post space.

A potential situation where a post and core could strengthen a tooth was identified by Hunter and colleagues[46] using photoelastic stress analysis. They determined that removal of internal tooth structure during endodontic therapy is accompanied by a proportional increase in stress. They also determined that minimal root canal enlargement for a post does not substantially weaken a tooth but when excessive root canal enlargement has occurred, a post strengthens the tooth. Therefore, **if the walls of a root canal are thin** due to removal of internal root caries or overinstrumentation during post preparation, then **a post may potentially strengthen the tooth**.

Two-dimensional finite element analysis was used in one study[47] to determine the effect of posts on dentin stress in pulpless teeth. When loaded vertically along the long axis, a post reduced maximal dentin stress by as much as 20%. However, only a small (3% to 8%) decrease in dentin stress was found when a tooth with a post was subjected to masticatory and traumatic loadings at 45° to the incisal edge. The authors proposed that the reinforcement effect of posts is doubtful for anterior teeth because they are subjected to angular forces.

Clinical Data

Sorenson and Martinoff[48] evaluated endodontically treated teeth with and without posts and cores. Some of the teeth were restored with single crowns while others served as fixed partial denture abutments or removable partial denture abutments. Posts and cores significantly decreased the clinical success rate of teeth with single crowns, improved

the clinical success of removable partial denture abutment teeth, and had little influence on the clinical success of fixed partial denture abutments. Eckerbom and colleagues[49] examined the radiographs of 200 consecutive patients and reexamined the patients radiographically 5 to 7 years later to determine the prevalence of apical periodontitis. Of the 636 endodontically treated teeth evaluated, 378 had posts and 258 did not have posts. At both examinations, **apical periodontitis was significantly more common in teeth with posts** than in endodontically treated teeth without posts. Morfis[50] evaluated the incidence of vertical root fracture in 460 endodontically treated teeth, 266 of which had posts. There were 17 teeth with root fracture after a time period of at least 3 years. Nine of the 17 fractured teeth had posts and eight root fractures occurred in teeth without posts. Morfis[50] concluded that the endodontic technique may cause vertical root fracture. In an analysis of data from multiple clinical studies, Goodacre[51] found that 3% of teeth with posts fractured. **None of these clinical data provides definitive support for the concept that post and cores strengthen endodontically treated teeth or improve their long-term prognosis**.

The Purpose of Posts

Since clinical and laboratory data indicate teeth are not strengthened by posts, their purpose is for retention of a core that will provide appropriate support for the definitive crown or prosthesis. Unfortunately, this primary purpose has not been completely recognized. Hussey[52] noted that 24% of general dental practitioners felt that a post strengthens teeth. A 1994 survey (with responses from 1066 practitioners and educators) revealed some interesting facts. Ten percent of the dentist respondents felt that every endodontically treated tooth should receive a post. It was determined that 62% of dentists over age 50 believed a post reinforces the tooth whereas only 41% of the dentists under age 41 believed in that concept. Thirty-nine percent of part-time faculty, 41% of full-time faculty, and 56% of non-faculty practitioners felt that posts reinforce teeth.[53]

CONCLUSIONS

Both laboratory and clinical data fail to provide definitive support for the concept that posts strengthen endodontically treated teeth. Therefore, **the purpose of a post is to provide retention for a core**.

WHAT IS THE CLINICAL FAILURE RATE OF POSTS AND CORES?

Several studies provide clinical data regarding the number of posts and cores that failed over certain time periods[54–66] (Table 35-1). When this number is divided by the total number of posts and cores placed, a failure percentage is determined. A 9% overall average for failure was calculated by averaging the failure percentages from 10 studies (an average study length of six years). In these studies, the percent failure ranged from 7% to 14%.

A review of more specific details from the 11 studies provides insight into the length of each study and the number of posts and cores evaluated. The findings of a 5-year retrospective study of 66 posts and cores indicate there were six failures and a 9% failure rate.[54] Another study found that 17 of 154 posts failed after 3 years for an 11% failure rate.[45] A failure rate of 9% was found in three studies.[56–58] Two studies reported a 7% post and core failure rate after 9 years or more.[59,65] An 8% failure rate (39 of 516 posts and cores) was published when 516 posts and cores placed by senior dental students were retrospectively evaluated,[60] whereas another study recorded a 14% failure rate (eight failures in 56 posts and cores) from posts and cores placed by dental students.[61] A study of 397 posts followed for 25 years reported a 10% post and core failure rate after 25 years (40 of 397 posts failed).[64] A more recent study recorded a 6% failure rate (19 of 308 posts) for posts placed in a specialist practice setting.[66]

Kaplan-Meier survival statistics (percentage of survival over certain time periods) were presented or could be calculated from the data in nine studies[62] (Table 35-2). The survival rates ranged from a high of 99% after 10 years or more

TABLE 35-1 Clinical Failure Rate of Posts and cores

Lead Author	Study Length	% Clinical failure
Turner, 1982*	5 yrs	9 (6 of 66)
Sorenson, 1984	1–25 yrs	9 (36 of 420)
Bergman, 1989*	6 yrs	9 (9 of 96)
Weine, 1991*	10 yrs or more	7 (9 of 138)
Hatzikyriakos, 1992*	3 yrs	11 (17 of 154)
Mentink, 1993*	1–10 yrs (mean 4.8)	8 (39 of 516)
Wallerstedt, 1984*	4–10 yrs (mean 7.8)	14 (8 of 56)
Torbjörner, 1995	4–5 yrs	9 (72 of 788)
Balkenhol, 2006	1–9 yrs (mean 2.1)	7 (50 of 802)
Valderhaug, 1997	1–25 yrs (mean 2.1)	10 (40 of 397)
Salvi, 2007	2–11 yrs (mean 5.3)	6 (19 of 308)
Mean values§	**8 yrs**	**9 (292 of 3433)**

*Studies used to calculate mean study length.
§Calculation made by averaging numeric data from all studies.

TABLE 35-2 Kaplain-Meier Survival Data (%) of Posts and Cores

Lead Author	Study Length	% Survival
Robert, 1970	5.2 yrs mean	78
Wallerstedt, 1984	4–10 yrs range	86
Sorenson, 1985	1–25 yrs range	90
Weine, 1991	10 yrs or more	99
Hatzikyriakos, 1992	3 yrs	92
Mentink, 1993	1–10 yrs (mean 4.8)	82
Creugers, 1993 (meta-analysis)	6 yrs	81 (threaded posts), 91 (cast posts)
Balkenhol, 2006	1–9 yrs range (mean 2.1)	89
Valderhaug, 1997	1–25 yrs range	80

of follow-up to a 78% survival rate after a mean time of 5.2 years. The failure percentage per year has also been calculated and ranged from 1.56% per year[47] to 4.3% per year.[63]

CONCLUSIONS

Posts and cores had an average clinical failure rate of 9% (6% to 14% range) when the data from 11 studies were combined (average study length of 8 years).

WHAT ARE THE MOST COMMON TYPES OF POST AND CORE FAILURES?

Eight studies[54,55,57,58,60,65–68] indicate that **post loosening is the most common cause of post and core failure** (Figure 35-5). Turner[54] reported on 100 failures of post-retained crowns and indicated that post loosening was the most common type of failure. Of the 100 failures, 59 were caused by post loosening. The next most common occurrences were 42 apical abscesses followed by 19 carious lesions. There were 10 root fractures and six post fractures. In another paper by Turner,[67] he reported the findings of a 5-year retrospective study of 52 post-retained crowns. Six posts had come loose which was the most common failure. Lewis and Smith[68] presented data regarding 67 post and core failures after 4 years. Forty-seven of the failures resulted from posts loosening, eight from root fractures, seven from caries, and four from bent or fractured posts. Bergman and colleagues[57] found eight failures in 96 posts after 5 years. Six posts had come loose and two roots fractured. Hatzikyriakos and colleagues[55] reported on 154 posts and cores after 3 years. Five posts had come loose, five crowns had come loose, four roots fractured, and caries caused three failures. Mentink[60] identified 30 post loosenings and 9 tooth fractures when evaluating 516 posts and cores over a 1- to 10-year time period (4.8 years mean study length). Torbjörner and colleagues[58] reported on the frequency of three technical failures (loss of retention, root fracture, and post fracture). They did not report biologic failures. Loss of retention was the most frequent post failure, accounting for 45 of the 72 post and core failures. Root fracture (Figure 35-6) was the second most common failure cause followed by post fracture. Balkenhol and colleagues[65] reported on 802 posts and cores over a 10-year period. Thirty-nine posts had loosened, eight had longitudinal root fracture, and six had transverse root fracture.

In two studies, factors other than loss of retention were listed as the most common cause of failure.[56,59] Sorenson and Martinoff[56] evaluated 420 posts and cores and recorded 36 failures. Of the 36 failures, eight were related to restorable tooth fractures, 12 to nonrestorable tooth fractures, 13 to loss of retention, and three were caused by root perforations. Weine and colleagues[59] found nine failures in 138 cast posts and cores after 10 years or more. Three failures were caused by restorative procedures, two by endodontic treatment, two by periodontal problems, and two by root fractures. No posts failed due to loss of retention.

Four studies have provided data on the incidence of tooth fracture but no information regarding post loosening. Linde[69] reported that three of 42 teeth fractured, Ross[70] found no fractures with 86 posts, Morfis[50] found that 10 of 266 teeth fractured, and Wallerstedt[61] identified two fractures with 56 posts.

Loss of retention and tooth fracture (in that order of occurrence) are the two most common causes of failure

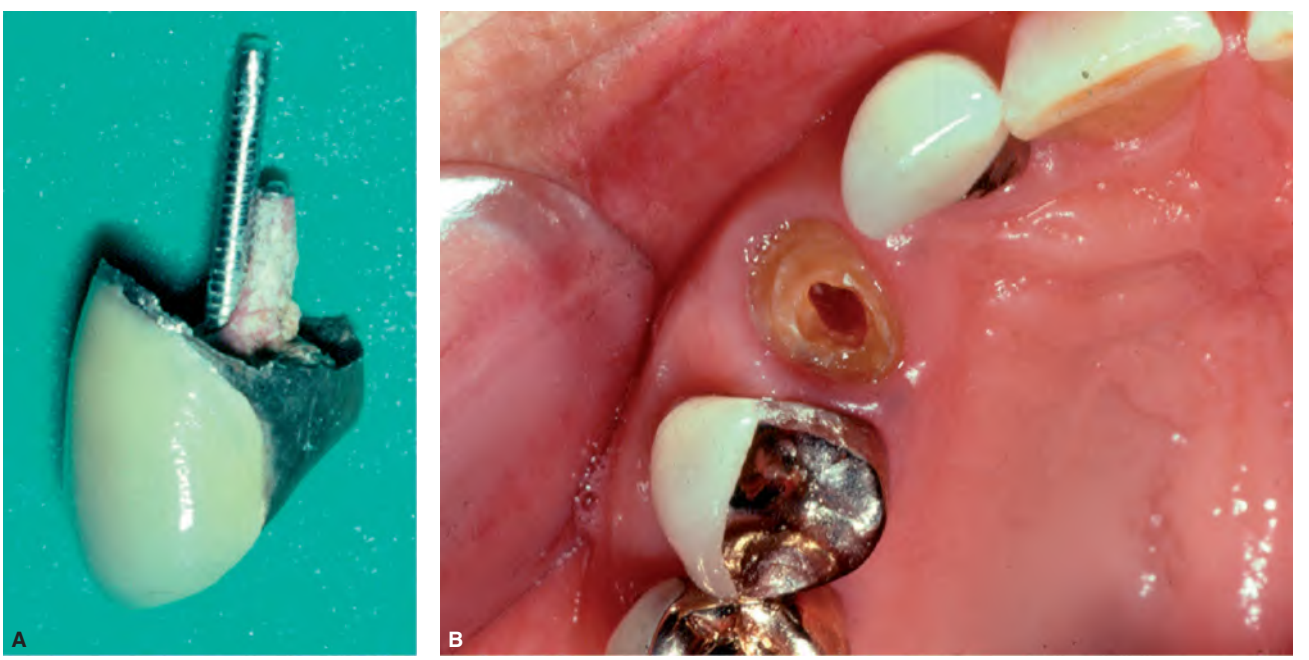


FIGURE 35-5 Post and crown loosened from maxillary canine a few years after placement. ***A.*** Both the post/core and the crown came off. ***B.*** Clinical photo shows very little cervical tooth structure for retention of the crown.

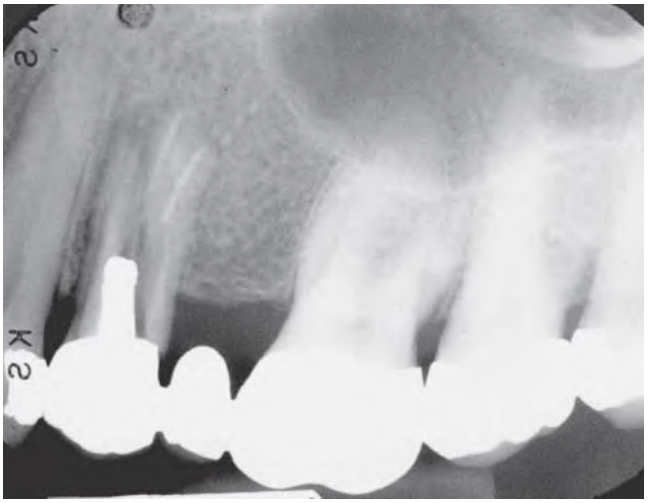


FIGURE 35-6 Radiograph of a fractured maxillary first premolar with a post with excessive diameter and insufficient length, two problems frequently seen in conjunction with fractured roots.

when these studies are collectively analyzed by averaging the numerical data from all the studies. Five percent of the posts placed (144 of 2,980 posts) experienced loss of retention (Table 35-3). Two percent of the posts placed (82 of 3,827 posts) failed via tooth fracture (Table 35-4).

CONCLUSIONS

Loss of retention and tooth fracture are the two most common causes of post and core failure.

WHICH POST DESIGN PRODUCES THE GREATEST RETENTION?

Laboratory Data

There have been many laboratory studies comparing the retention of various post designs. Threaded posts provide the greatest retention, followed by cemented, parallel-sided posts. Tapered cemented posts are the least retentive. Cemented, parallel-sided posts with serrations are more retentive than cemented, smooth-sided parallel posts. These laboratory data are discussed below.

Clinical Data

There is clinical support for these laboratory studies. Torbjörner and colleagues[58] reported significantly greater loss of retention with tapered posts (7%) compared to parallel posts (4%). Sorenson and Martinoff[56] determined that 4% of tapered posts failed by loss of retention whereas 1% of parallel posts failed in that manner. Turner[67] indicated that tapered posts loosened clinically more frequently than parallel-sided posts. Lewis and Smith[68] also found a higher loss of retention with smooth-walled tapered posts than parallel posts. Bergman and colleagues[57] and Mentink and colleagues[60] evaluated only tapered posts, and both studies reported that 6% of tapered posts failed via loss of retention, values higher than those recorded by Torbjörner and colleagues[58] and Sorenson and Martinoff[56] for parallel posts.

Contrasting results were reported by Weine and colleagues.[59] They found no clinical failures from loss of retention with cast tapered posts. Hatzikyriakos and colleagues[55] studied tapered threaded posts, parallel cemented posts, and tapered cemented posts. The only posts that loosened from the root were parallel cemented posts.

CONCLUSIONS

Tapered posts are the least retentive and threaded posts the most retentive in laboratory studies. Most of the clinical data supports the laboratory findings.

IS THERE A RELATIONSHIP BETWEEN POST FORM AND THE POTENTIAL FOR ROOT FRACTURE?

Laboratory Data

Using photoelastic stress analysis, Henry[71] determined that threaded posts produced undesirable levels of stress. Another study used strain gauges attached to the root and compared four parallel-sided threaded posts with one parallel-sided nonthreaded post.[70] Two of the threaded posts produced the highest strains, whereas two other threaded posts caused strains comparable to the nonthreaded post. Standlee and coleagues,[72] using photoelastic methods, indicated that tapered, threaded posts were the worst stress producers. When three types of threaded posts were compared in extracted teeth, Deutsch and colleagues[73] found that **tapered, threaded posts increased root fracture by 20 times that of the parallel threaded posts** (Figure 35-7).

Laboratory testing of **split threaded posts** has provided varying results, but more research groups have concluded

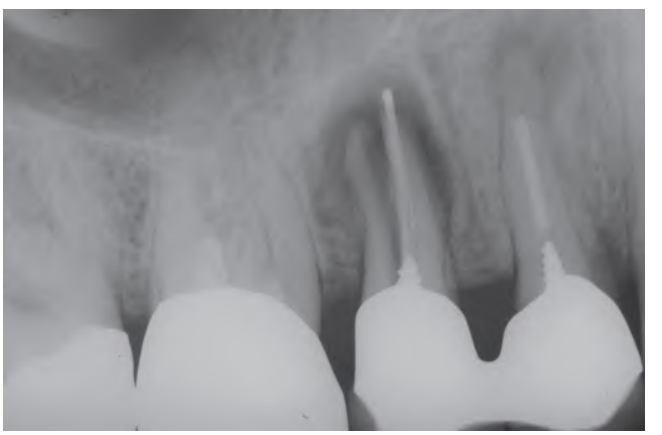


FIGURE 35-7 Radiograph showing a threaded post that may have caused root fracture in the second premolar.

TABLE 35-3 Clinical Loss of Retention Associated with Posts and Cores

Lead Author	Study Length	% of Posts Placed that Loosened	Post Form	% of Failures Owing to Loosening
Turner, 1982	5 yrs	9 (6 of 66)	Appeared to be tapered	*
Turner, 1982	1–5 yrs or more	*	Tapered	59 (59 of 100)
Sorensen, 1984	1–25 yrs	3 (13 of 420)	Tapered and parallel	36 (13 of 36)
Lewis, 1988	4 yrs	*	Threaded, tapered, and parallel	70 (47 of 67)
Bergman, 1989	6 yrs	6 (6 of 96)	Tapered	67 (6 of 9)
Weine, 1991	10 yrs or more	0 (0 of 138)	Tapered	0 (0 of 9)
Hatzikyriakos, 1992	3 yrs	3 (5 of 154)	Threaded, tapered, and parallel	29 (5 of 17)
Mentink, 1993	1–10 yrs (mean 4.8)	6 (30 of 516)	Tapered	77 (30 of 39)
Torbjörner, 1995	4–5 yrs	6 (45 of 788)	Tapered and parallel	63 (45 of 72)
Balkenhol, 2006	1–9 yrs (mean 2.1)	5 (39 of 802)	Tapered	43 (39 of 90)
Mean values§		**5 (144 of 2980)**		**56 (244 post loosenings of 439 total failures)**

*Data not available in publication.
§Calculation made by averaging numeric data from all studies.

TABLE 35-4 Clinical Tooth Fractured Associated with Posts and Cores

Lead Author	Study Length	% of Teeth Restored with Posts that Fractured	Post Form (s) studied	% of Failures Owing to Fracture
Turner, 1982	5 yrs	0 (0 of 66)	Appeared to be tapered	*
Turner, 1982	1–5 yrs or more	*	Tapered, parallel, and threaded	10 (10 of 100)
Sorensen, 1984	1–25 yrs	3 (12 of 420)	Tapered and parallel	33 (12 of 36)
Linde, 1984	2–10 yrs (5 yrs, 8 months mean)	7 (3 of 42)	Threaded	38 (3 of 8)
Lewis, 1988	4 yrs	*	Threaded, tapered, and parallel	12 (8 of 67)
Bergman, 1989	6 yrs	3 (3 of 96)	Tapered	33 (3 of 9)
Ross, 1980	5 yrs or more	0 (0 of 86)	Tapered, parallel, and threaded	0 (0 of 86)
Morfis, 1990	3 yrs at least	4 (10 of 266)	Threaded and parallel	*
Weine, 1991	10 yrs or more	1 (2 of 138)	Tapered	50 (2 of 4)
Hatzikyriakos, 1992	3 yrs	3 (4 of 154)	Threaded, tapered, and parallel	3 (4 of 17)
Mentink, 1993	1–10 yrs (mean 4.8)	2 (9 of 516)	Tapered	23 (9 of 39)
Wallerstedt, 1984	4 – 10 yrs (mean 7.8)	4 (2 of 56)	Threaded	25 (2 of 8)
Torbjörner, 1995	4–5 yrs	3 (21 of 788)	Tapered and parallel	29 (21 of 72)
Balkenhol, 2006	1–9 yrs (mean 2.1)	2 (14 of 802)	Tapered	15 (14 of 90)
Valderhaug, 1997	1–25 yrs (mean 2.1)	1 (2 of 397)	Tapered	5 (2 of 40)
Mean values§		**2 (82 of 3827)**		**16 (90 tooth fractures of 576 total failures)**

*Data not available in publication.
§Calculation made by averaging numeric data from all studies.

they **do not reduce the stress associated with threaded posts.** Thorsteinsson and colleagues[74] determined that split threaded posts did not reduce stress concentration during loading. In another study, split threaded posts were found to produce installation stresses comparable to other threaded posts.[75] Greater stress concentrations than those of some other threaded posts were reported under simulated functional loading.[76–78] Rolf and colleagues[79] found that a split threaded post produced comparable stress to one type of threaded post and less stress than a third threaded post design. Ross and colleagues[70] determined a split threaded post produced less root strain than two other threaded posts and comparable strain to a third threaded post and a nonthreaded post. Another research group[80] concluded the split threaded design reduced the stresses caused during cementation compared to a rigid threaded post design. Multiple photoelastic stress studies have concluded that **posts designed for cementation produced less stress than threaded posts.**[71,72,79]

When parallel-sided cemented posts were compared with tapered cemented posts, photoelastic stress testing results have generally favored parallel-sided posts. Using this methodology, Henry[71] found that parallel-sided posts distribute stress more evenly to the root. Finite element analysis studies produced similar results.[81,82] Two additional photoelastic studies[74,77] concluded that parallel posts concentrate stress apically and tapered posts concentrate stress at the post-core junction. Also using photoelastic testing, Assif and colleagues[83] found that tapered posts showed equal stress distribution between the cementoenamel junction and the

apex compared with parallel posts that concentrated the stress apically.

When fracture patterns in extracted teeth were used to compare parallel and tapered posts, the evidence favoring parallel posts is less favorable. Sorenson and Engelman[84] determined that tapered posts caused more extensive fractures than parallel-sided posts did, but the load required to create fracture was significantly higher with tapered posts. Lu,[38] also using extracted teeth, found no difference in the fracture location between prefabricated parallel posts and cast posts and cores. Assif and colleagues[85] tested the resistance of extracted teeth to fracture when the teeth were restored with either parallel or tapered posts and complete crowns. No significant differences were noted, and post design did not influence fracture resistance.

In analyzing the stress distribution of posts, it was noted that tapered posts generate the least cementation stress and should be considered for teeth that have thin root walls, are nearly perforated or have perforation repairs.[77]

Clinical Data

There are several clinical studies that provide data related to the incidence of root fracture associated with different post forms. Some of these studies provide a comparison of multiple post forms, whereas other studies evaluated only one type of post. Combining all the root fracture data for each post form from both types of studies reveals some interesting trends (Table 35-5). Five studies present data regarding root fractures and threaded posts,[50,56,61,69,86] four regarding fractures associated with parallel-sided cemented posts[50,56,58,86] and nine related to tapered cemented posts.[50,56–59,60,64,65,86] If the total number of threaded posts evaluated in the five studies is divided into the total number of fractures found with threaded posts, a percent value can be determined that represents the average incidence of tooth fracture associated with threaded posts in the five studies. The same data can be calculated for parallel cemented and tapered cemented posts, permitting a comparison of the root fracture incidences associated with these three post forms.

Combining the five studies that reported data relative to **threaded posts** produced a mean **fracture rate of 7%** (11 fractures from 169 posts). The four clinical studies that contain fracture data from **parallel-sided cemented posts** produced a mean **fracture incidence of 1%** (9 fractures from 687 posts). From the seven studies reporting root fracture with **tapered posts**, there is a mean **fracture rate of 2%** (66 root fractures from 2,752 posts). This combined study data support the previously cited photoelastic laboratory stress tests, indicating that the greatest incidence of root fractures occurred with threaded posts and that the lowest incidence of root fracture was associated with parallel cemented posts. In a meta-analysis of selected clinical studies, Creugers and colleagues[62] calculated a 91% tooth survival rate for cemented cast posts and cores and an 81% survival rate for threaded posts with resin cores.

While the combined data from all the studies for each type of post revealed certain trends, analysis of individual studies (where multiple post forms were compared in the same study) produced less conclusive results. One study of threaded posts and cemented posts determined that teeth with threaded posts were lost more frequently than teeth with cast posts.[49] In three other clinical comparisons of threaded and cemented posts, no tooth fracture differences were noted.[50,55,86] In addition to the comparisons of threaded and cemented posts, four clinical studies provide data comparing the tooth fracture incidences associated with parallel-sided and tapered posts. In comparing parallel and tapered posts by reviewing dental charting records, a higher failure rate was reported with tapered posts than with parallel posts in two studies, and the failures were judged to be more severe with tapered posts.[56,58] Two other clinical studies determined there were no differences between tapered and parallel-sided posts.[58,86] Hatzikynakos and colleagues[55] found no significant differences between 47 parallel cemented posts and 44 tapered cemented posts after 3 years of service. Ross[86] evaluated 86 teeth with posts and cores that had been restored at least 5 years previously. No fracture differences were found between 38 tapered cemented posts and 39 parallel cemented posts.

Unfortunately, the total number of clinical studies that compared multiple post forms in the same study is limited. Also, several factors may have affected the findings of available studies. Two of the papers that contained a comparison of multiple post forms covered sufficiently long time periods (10 to 25 years) that the tapered cemented posts may have been in place for much longer time periods than the parallel-sided cemented posts (due to the later

TABLE 35-5 Post Form and Tooth Fracture

Clinical Data (% of Post and Cores Studied That Failed via Tooth Fracture)

Threaded Posts (Lead Author)	Parallel-Sided Posts (Lead Author)	Tapered Posts (Lead Author)
40 (2 of 5) (Sorensen)	0 (0 of 170) (Sorensen)	7 (18 of 245) (Sorensen)
0 (0 of 10) (Ross)	2 (5 0f 332) (Torbjörner)	4 (16 of 456) (Torbjörner)
4 (2 of 56) (Wallerstedt)	0 (0 of 39) (Ross)	1 (2 of 138) (Weine)
7 (3 of 42) (Linde)	3 (4 of 146) (Morfis)	2 (9 of 516) (Mentink)
7 (4 of 56) (Morfis)		3 (3 of 96) (Bergman)
		0 (0 of 38) (Ross)
		3 (2 of 64) (Morfis)
		2 (14 of 802) (Balkenhol)
		1 (2 of 397) (Valderhaug)
7% Mean* (11 of 169)	**1% Mean* (9 of 687)**	**2% Mean* (66 of 2752)**

*Calculation made by averaging numeric data from all studies.

introduction of parallel posts into the dental market).[56,58] The mean time since the placement of each post form was not identified in these studies. Also, both of these studies were based on reviews of patient records (rather than clinical examinations) and depended on the accuracy of dental charts in determining if and when posts failed as well as the cause of the failure. Another factor that affected the results of many of the referenced clinical studies was the length of the posts. For instance, in Sorenson and Martinoff's study,[56] 44% of the tapered cemented posts had a length that was half (or less than half) the incisocervical/occlusocervical dimension of the crown whereas only 4% of the parallel cemented posts were that short. Since short posts have been associated with higher root stresses in laboratory studies, the difference in post length may have affected their findings where tooth fractures occurred with 18 of 245 tapered posts compared with no fractures with 170 parallel posts.

CONCLUSIONS

When evaluating the relationship between post form and root fracture, **laboratory tests** generally indicate all types of **threaded posts produce the greatest potential for root fracture**. When comparing tapered and parallel cemented posts using photoelastic stress analysis, the results generally favor the parallel cemented posts. However, the evidence is mixed when the comparison between tapered and parallel posts is based on fracture patterns in extracted teeth created by applying a force via a mechanical testing machine.

When evaluating the combined data from multiple **clinical studies, threaded posts generally produced the highest root fracture incidence** (7%) compared with tapered cemented posts (2%) and parallel cemented posts (1%). Analysis of individual clinical studies as opposed to the combined data produces less conclusive results. Additional comparative clinical studies would be beneficial, including designs that have not yet been evaluated in comparative studies.

WHAT IS THE PROPER LENGTH FOR A POST?

A wide range of recommendations has been made regarding post length, which includes the following:

1. The post length should equal the incisocervical or occlusocervical dimension of the crown.[87–94]
2. The post should be longer than the crown.[95]
3. The post should be one-third of the crown length.[96]
4. The post should be half of the root length.[97,98]
5. The post should be two-thirds of the root length.[99–103]
6. The post should be four-fifths of the root length.[104]
7. The post should be terminated halfway between the crestal bone and root apex.[105–107]
8. The post should be as long as possible without disturbing the apical seal.[71]

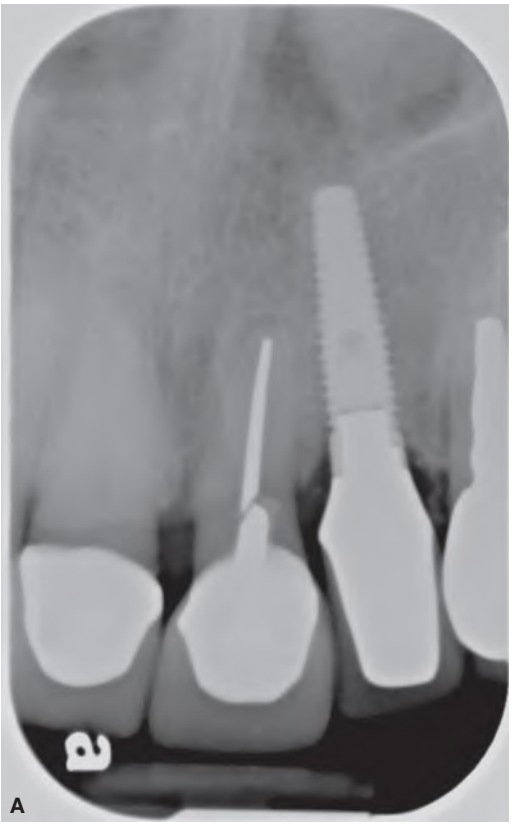
A review of scientific data provides the basis for differentiating between these varied guidelines. While short posts have never been advocated, they have been frequently observed during radiographic examinations (Figures 35-8 and 35-9). Grieve and McAndrew[108] found that only 34% of 327 posts were as long as the incisocervical length of the crown. In a clinical study of 200 endodontically treated teeth, Ross[86] determined that only 14% of posts were two-thirds or more of the root length and 49% of the posts were one-third or less of the root length. A radiographic study of 217 posts determined that only 5% of the posts were two-thirds to three-fourths of the root length.[109] In a retrospective clinical study of 52 posts, Turner[54] radiographically compared the length of the post with the maximal length available if 3 mm of gutta-percha were retained. Posts that came loose used only 59% of the ideal length and only 37% of the posts were longer than the proposed minimal length. Nine millimeters was proposed as the ideal length. Short posts have been associated with higher root stresses[46,75,77,81,82] and a greater tendency for root fracture to occur.

Sorensen and Martinoff[56] determined that the clinical success was markedly improved when the post was equal to or greater than the crown length. According to Johnson and Sakumura[110] posts that were three-fourths or more of the root length were up to 30% more retentive than posts that were half of the root length or equal to the crown length. Leary and colleagues[111] indicated that posts with a length at least three-fourths of the root offered the greatest rigidity and least root bending.

This data indicates post length would appropriately be three-fourths that of root length. However, some interesting results occur when post length guidelines of two-thirds to three-fourths of the root length are applied to teeth with average, long, and short root lengths. It was determined that a post approaching this recommended length range is not possible without compromising the apical seal by retaining less than 5 mm of gutta-percha.[112] When post length was half that of the root, the apical seal was rarely compromised on average length roots. However, when posts were two-thirds of the root length, many of the average and short length roots would have less than the optimal gutta-percha seal. Shillingburg and colleagues[113] also indicated making the post length equal the clinical crown length can cause the post to encroach on the 4-mm "safety zone" required for an apical seal.

Abou-Rass and calleagues[114] proposed a post length guideline for maxillary and mandibular molars based on the incidence of lateral root perforations when post preparations were made in 150 extracted teeth. They determined molar posts should not be extended more than 7 mm apical to the root canal orifice.

When teeth have diminished bone support, stresses increase dramatically and are concentrated in the dentin near the post apex.[115] A recent finite element model study established a relationship between post length and alveolar bone level.[116] To minimize stress in the dentin and in the post, the post should extend more than 4 mm apical to the bone.



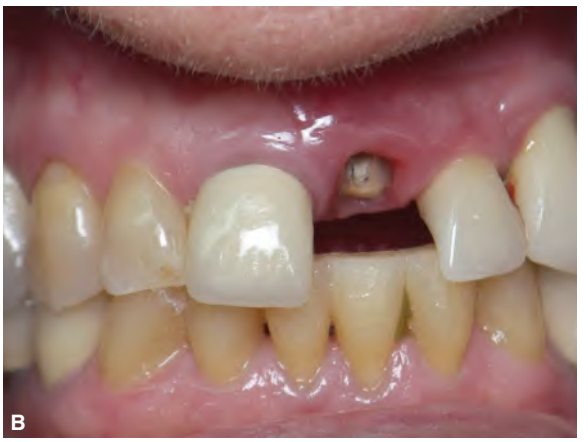
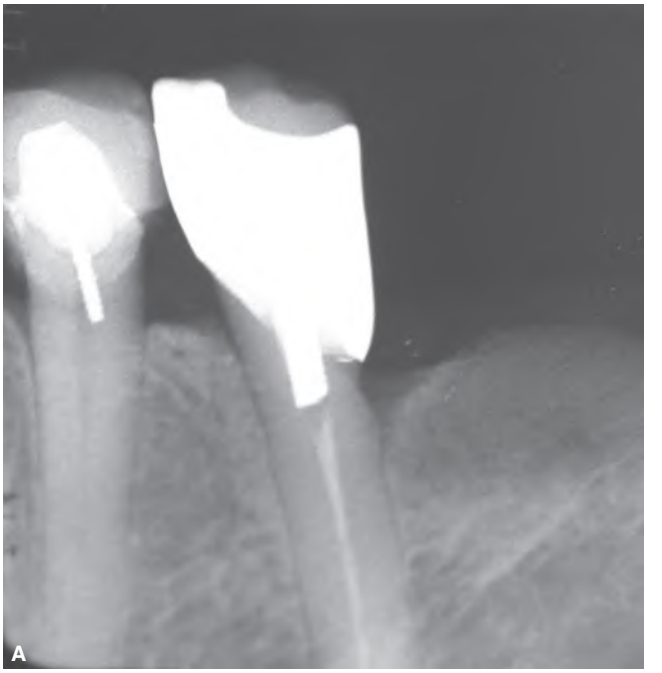


FIGURE 35-8 ***A.*** Diminished bone support of a maxillary central incisor. ***B.*** Root fracture caused by stresses on the tooth. ***C.*** Broken crown showing the fracture line at the apex of the post. (Reprinted with permission from Baba NZ.[331])



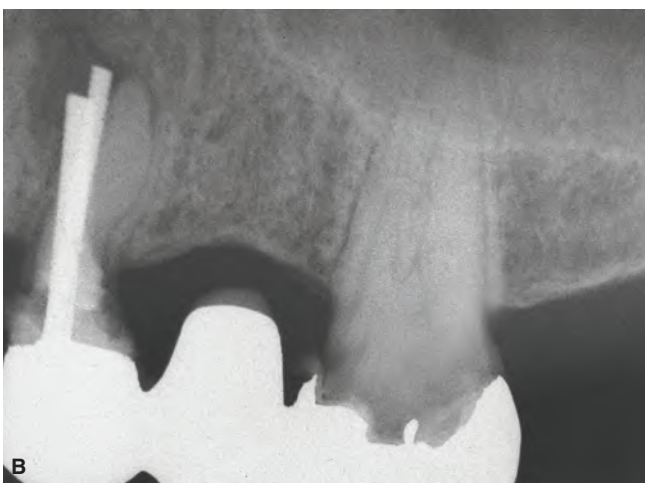


FIGURE 35-9 ***A.*** Radiograph of a second premolar with a very short post; the lack of adequate retention for the core can result in the prosthesis loosening. ***B.*** Radiograph of first maxillary left premolar with a very long post; the inadequate size of the post caused the perforation of the roots. (Figure 9B reprinted with permission from Baba NZ.[331])

CONCLUSIONS

Reasonable clinical guidelines for length include the following:

1. Make the post approximately three-fourths of the length of the root when treating long rooted teeth.
2. When average root length is encountered, post length is dictated by **retaining 5 mm of apical gutta-percha** and extending the post to the gutta-percha (Figure 35-10).
3. Whenever possible, posts should extend at least 4 mm apical to the bone crest to decrease dentin stress.

4. Molar posts should not be extended more than 7 mm into the root canal apical to the base of the pulp chamber (Figure 35-11).

HOW MUCH GUTTA-PERCHA SHOULD BE RETAINED TO PRESERVE THE APICAL SEAL?

It has been determined that when 4 mm of gutta-percha is retained, only one of 89 specimens showed leakage, whereas 32 of 88 specimens leaked when only 2 mm of gutta-percha was retained.[117] Two studies found no leakage at 4 mm,[118,119] and two additional studies found little leakage at 4 mm.[117,120] Portell and colleagues[121] found that most specimens with only 3 mm of apical gutta-percha had some leakage. When the leakage associated with 3, 5, and 7 mm of gutta-percha was compared, Mattison and colleagues[122] found significant leakage differences between each of the dimensions. They proposed that at least 5 mm of gutta-percha is required for an adequate apical seal. Nixon and colleagues[123] compared the sealing capabilities of 3, 4, 5, 6, and 7 mm of apical gutta-percha using dye penetration. The greatest leakage occurred when only 3 mm was retained, and it was significantly different from the other groups. They also noted a significant decrease in leakage occurred when 6 mm of gutta-percha remained. Raiden and Gendelman[124] cemented stainless steel posts with zinc phosphate cement in teeth with residual root canal fillings of 1, 2, 3, and 4 mm. They tested apical leakage using a passive dye system and concluded that 4 mm of apical seal provided a leakage value of zero. Kvist and colleagues[125] examined radiographs from 852 clinical endodontic treatments. Posts were present in 424 of the teeth. Roots with posts in which the remaining root filling material was shorter than 3 mm showed a significantly higher frequency of periapical radiolucencies. Using a pressure-driven tracer assay, Wu and colleagues[126] and Abramovitz and colleagues[127] found that 4 or 5 mm of apical seal were inferior in their ability to prevent leakage, compared with an original full-length root canal filling. Similarly, Metzger and colleagues[128] compared the sealing capabilities of 3, 5, 7, and 9 mm of apical gutta-percha using a pressure-driven radioactive tracer assay. They concluded that the sealing is proportional to the length of the remaining filling and that original full-length root canal fillings have a superior seal compared to 3, 5, and 7 mm of apical gutta-percha.

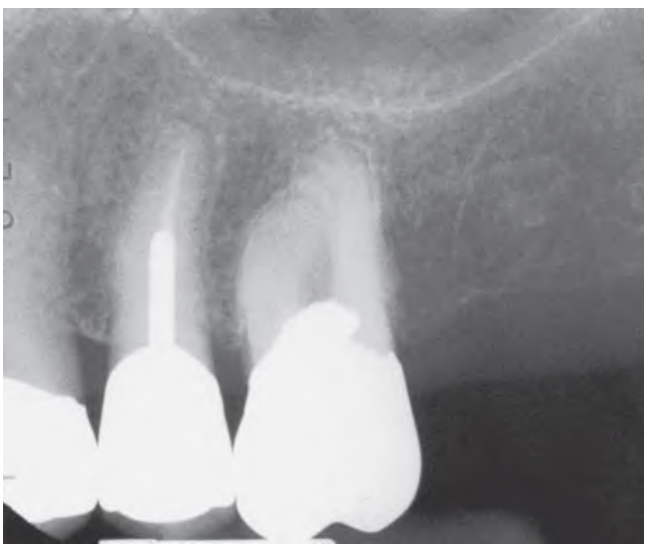


FIGURE 35-10 Five millimeters of gutta-percha was retained in the maxillary premolar and the post extended to that point.

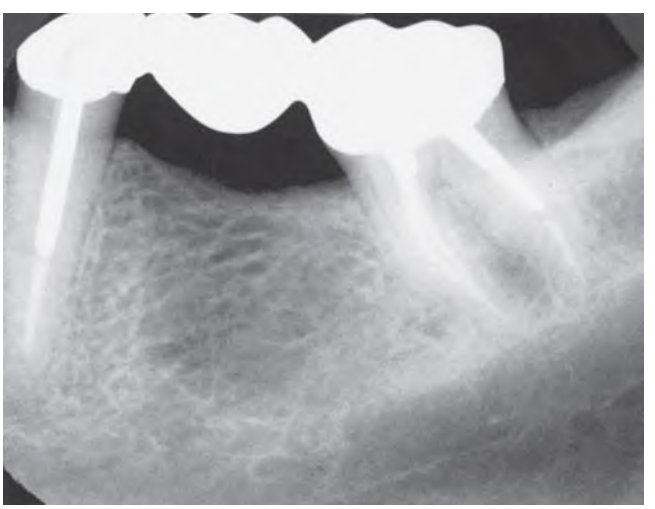


FIGURE 35-11 The post in the distal canal of the mandibular molar extends to a maximal length of 7 mm.

CONCLUSIONS

Since there is greater leakage when only 2 to 3 mm of gutta-percha is present (Figure 35-12), 4 to 5 millimeters should be retained apically to ensure an adequate seal. Although studies indicate 4 mm produces an adequate seal, stopping at 4 mm is difficult and radiographic angulation errors could lead to retention of less than 4 mm. **Therefore, at least 5 mm of gutta-percha should be retained apically. This seal**

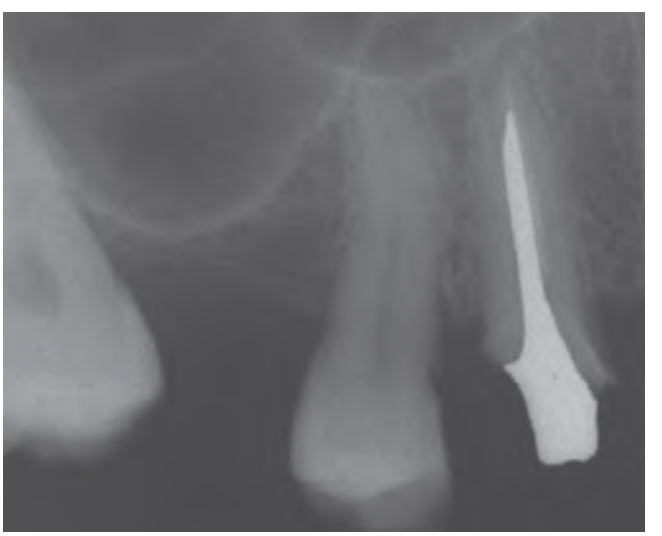


FIGURE 35-12 Less than 2 mm of gutta-percha remains in the maxillary first premola rapical to the cast post and core, increasing the risk of failure.

is complemented by a seal provided by the post and core, and the overlying crown. (Figure 35-11).

DOES POST DIAMETER AFFECT RETENTION AND THE POTENTIAL FOR TOOTH FRACTURE?

Studies relating post diameter to post retention have failed to establish a definitive relationship. Two studies determined there was an increase in post retention as the diameter increased,[113,129] whereas three studies found no significant retention changes with diameter variations.[130,121,131] Krupp and colleagues[132] indicated that post length was the most important factor affecting retention and post diameter was a secondary factor.

A more definitive relationship has been established between post diameter and stress in the tooth. Mattison[133] found that as the post diameter increased, **stress increased in the tooth**. Trabert and colleagues[134] measured the impact resistance of extracted maxillary central incisors as post diameter increased and found that increasing the post diameter decreased the tooth's resistance to fracture. Deutsch and colleagues[73] determined there was a six-fold increase in the potential for root fracture with every millimeter decrease in tooth diameter. However, two finite element studies failed to find higher tooth stresses with larger-diameter posts.[80,82]

CONCLUSIONS

Laboratory studies relating retention to post diameter have produced mixed results, whereas a more definitive relationship has been established between root fracture and large-diameter posts (Figure 35-13).

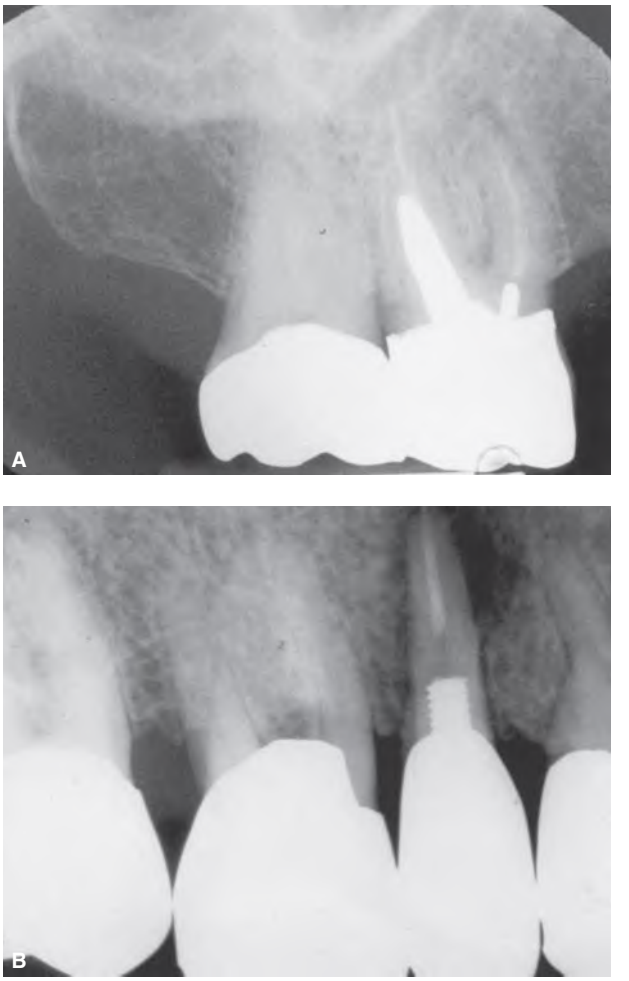


FIGURE 35-13 Excessive post diameters. ***A.*** Large-diameter post placed in the palatal root of the maxillary molar. ***B.*** Large-diameter threaded post caused fracture of the maxillary second premolar. The radiographic appearance of the bone is typical of a fractured root—a teardrop-shaped lesion with diffuse border.

WHAT IS THE RELATIONSHIP BETWEEN POST DIAMETER AND THE POTENTIAL FOR ROOT PERFORATIONS?

In a literature review of guidelines associated with post diameter, Lloyd and Palik[135] indicate there are three distinct philosophies of post space preparation. One group advocates the narrowest diameter for fabrication of a certain post length (the conservationists). Another group proposes a space with a diameter that does not exceed one-third the root diameter (the proportionists). The third group advised leaving at least 1 mm of sound dentin surrounding the entire post (the preservationists).

Based on the proportional concept of one-third the root diameter, three studies measured the root diameters of extracted teeth and proposed post diameters that would not exceed that proportion.[113,114,136] Tilk and colleagues[136] examined 1,500 roots. They measured the narrowest mesiodistal dimension at the apical, middle, and cervical one-thirds of the teeth except the palatal root of the maxillary first molar that was measured faciolingually. Based on the 95% confidence level that post width would not exceed one-third of the apical width of the root, they proposed the following post widths: small teeth such as mandibular incisors (0.6 to 0.7 mm); large-diameter roots such as maxillary central incisors and the palatal root of the maxillary first molar (1.0 mm); and for the remaining teeth (0.8 to 0.9 mm).

Shillingburg and colleagues[113] measured 700 root dimensions to determine the post diameters that would minimize the risk of perforation. Also based on not exceeding one-third the mesiodistal root width, they recommended the following post diameters: mandibular incisors (0.7 mm); maxillary central incisors or other large roots (1.7 mm - which was the maximal recommended dimension); post tip diameter (at least 1.5 mm less than the root diameter at that point); and post diameter at the middle of the root length (2.0 mm less than the root diameter).

Post spaces were prepared in 150 extracted maxillary and mandibular molars by using different instrument diameters and the resulting incidences of perforations were recorded.[114] The authors determined that the mesial roots of mandibular

molars and the buccal roots of maxillary molars should not be used for posts due to the higher risk of perforation on the furcation side of the root. For the principal roots (mandibular distal and maxillary palatal), they determined that posts should not be extended more than 7 mm into the root canal (apical to the pulp chamber) due to the risk of perforation. Regarding instrument size, they concluded that post preparations can be safely completed using a No. 2 Peeso instrument, but perforations are more likely when the larger No. 3 and 4 Peeso instruments were used.

Raiden and colleagues[137] evaluated several instrument diameters (0.7, 0.9, 1.1, 1.3, 1.5 and 1.7 millimeters) to determine which one(s) would preserve at least 1 mm of root wall thickness following post preparation in maxillary first premolars. They determined that instrument diameter must be small (0.7 mm or less) for maxillary first premolars with single canals because the mesial and distal developmental root depressions restrict the amount of available tooth structure in the centrally located single root canal. However, when there are dual canals, the instrument can be as large as 1.1 millimeters because the canals are located buccally and lingually into thicker areas of the roots.

CONCLUSIONS

Instruments used to prepare posts should be related in size to root dimensions to avoid excessive post diameters that lead to root perforation (Figure 35-14). Safe instrument diameters to use are 0.6 to 0.7 mm for small teeth such as mandibular incisors and 1 to 1.2 mm for large-diameter roots such as the maxillary central incisor. Molar posts longer than 7 mm have an increased chance of perforations and therefore should be avoided even when using instruments of an appropriate diameter.

CAN GUTTA-PERCHA BE REMOVED IMMEDIATELY AFTER ENDODONTIC TREATMENT AND A POST SPACE PREPARED?

Several studies indicate there is no difference in the leakage of the root canal filling material when the post space is prepared immediately after completing endodontic therapy.[118,138,120,139] Bourgeois and Lemon[138] found no difference between immediate preparation of a post space and preparation 1 week later when 4 mm of gutta-percha was retained. Zmener[120] found no difference in dye penetration between gutta-percha removal after 5 minutes and 48 hours. Two sealers were tested and 4 mm of gutta-percha was retained apically. When lateral condensation of gutta-percha was used, Madison and Zakariasen[118] found no difference in the dye penetration between immediate removal and 48-hour removal. Using the chloropercha filling technique, Schnell[139] found no difference between immediate removal of gutta-percha and no removal of gutta-percha. By contrast, Dickey and colleagues[140] found significantly greater leakage with immediate gutta-percha removal.

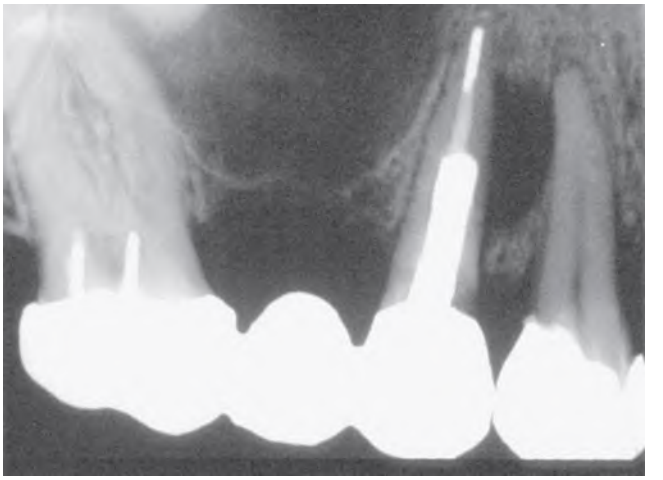


FIGURE 35-14 Excessive post diameter in the maxillary second premolar created a perforation in the mesial root concavity. Note the distinct border and round form of the radiolucent lesion, characteristics indicative of a root perforation.

Kwan and Harrington[141] tested the effect of immediate gutta-percha removal using both warm instruments and rotary instruments. There was no significant difference between the controls and immediate removal using warm pluggers and files.

Karapanou and colleagues[142] compared immediate and delayed removal of two sealers (a zinc oxide-eugenol-based sealer and a resin-based sealer). No difference between immediate and delayed removed was noted with the resin-based sealer, but delayed removal of the zinc oxide-eugenol-based sealer produced significantly greater leakage. Abramovitz and colleagues[127] compared immediate gutta-percha removal using hot pluggers and delayed gutta-percha removal (after two weeks) using Gates Glidden drills. They found no difference between the two methods.

Portell and colleagues[143] found that delayed gutta-percha removal (after two weeks) caused significantly more leakage than immediate removal when only 3 mm of gutta-percha was retained apically. Fan and colleagues[144] found more leakage from delayed removal of gutta-percha. Solano and colleagues[145] found a less significant difference in apical leakage between teeth whose post spaces were prepared at the time of the obturation and 1 week later using warm gutta-percha condensation and AH Plus sealer.

CONCLUSIONS

Adequately condensed gutta-percha can be safely removed immediately after endodontic treatment.

WHAT INSTRUMENTS REMOVE GUTTA-PERCHA WITHOUT DISTURBING THE APICAL SEAL?

Three methods have been advocated for the removal of gutta-percha during preparation of a post space: chemical (oil of eucalyptus, oil of turpentine and chloroform),

thermal (electrical or heated instruments), and mechanical (Gates Glidden drills, Peeso reamers, etc.). The chemical removal of gutta-percha for post space preparation is not utilized for specific reasons (microleakage, inability to control removal).[122,138] However, thermal and mechanical techniques or a combination of both are routinely used.

Multiple studies have determined there is no difference in leakage between removing gutta-percha with hot instruments and removing it with rotary instruments.[117,122,146] Suchina and Ludington[146] and Mattison and colleagues[122] found no difference between hot instrument removal and removal with Gates Glidden burs. Camp and Todd[117] found no difference between Peeso reamers, Gates Glidden burs, and hot instruments. Hiltner and colleagues[147] compared warm plugger removal with two types of rotary instruments (GPX burs and Peeso reamers). There were no significant differences in dye leakage between any of the groups. Contrasting results were found by Haddix and colleagues.[148] They measured significantly less leakage when the gutta-percha was removed with a heated plugger than when either a GPX instrument or Gates Glidden drills were used. DeCleen[149] found that it is desirable to remove gutta-percha using a heated instrument first, and then a small Gates Glidden drill. Using a pressure-driven radioactive tracer assay, Abramovitz and colleagues[127] found no difference in apical leakage between hot pluggers and Gates Glidden drills. Balto and colleagues[150] compared two methods of gutta-percha removal and their impact on apical leakage. They found that removing gutta-percha with Peeso reamers showed less leakage compared with using a hot plugger.

CONCLUSIONS

Both rotary instruments and hot hand instruments can be safely used to remove adequately condensed gutta-percha when 5 mm is retained apically.

CAN A SEPERATED INSTRUMENT BE REMOVED DURING POST SPACE PREPARATION AND STILL MAINTAIN THE APICAL SEAL?

Following root canal therapy, the endodontically treated tooth could present with a separated instrument (files, rotary instruments, etc.) in any part of the canal. Attempts to remove these fragments prior to post space preparation could lead to loss of apical seal, perforations, ledge formation, and/or over enlargement of the canal. A decision to bypass or remove the fragment will depend on the type and position of the broken instrument, the canal anatomy, the position of the fractured instrument, and should be determined by an endodontist with the use of an operating microscope. For more details, see Chapter 23.

Bypassing fractured instruments seems to be the best approach. Hülsmann and Schinkel[151] reported an overall success rate of 68% when bypassing broken instruments from canals in vivo. Ward and colleagues[152] reported an overall success rate of 73%. Suter and colleagues[153] with the use of an operating microscope were able to achieve an overall success rate of 87% when removing broken instruments.

CONCLUSIONS

If a separated fragment of any instrument cannot be removed from the canal during post space preparation, it should be bypassed or left in the canal.

CAN A PORTION OF SILVER POINT BE REMOVED AND STILL MAINTAIN THE APICAL SEAL?

In one study, all the specimens leaked when 1 mm of a 5-mm long silver point was removed by using a round bur.[120] Neagley[119] found that removal of the filling material coronal to the silver point with a Peeso reamer caused no leakage. However, when all the filling material was removed and 1 mm of the silver point removed, complete dye penetration occurred in eight of nine specimens.

CONCLUSIONS

The removal of a portion of a silver point during post preparation causes apical leakage.

HOW SOON SHOULD THE DEFINITIVE RESTORATION BE PLACED AFTER POST SPACE PREPARATION?

One-Piece Provisional Restoration

It is well established that a deficient root canal obturation and a poor coronal restoration will potentially allow saliva and microorganisms to penetrate the root canal causing periapical disease.[154–160]

Provisional restorations are mainly used to provide the patient with a functional and esthetic restoration.[150,161–164] Demarchi and colleagues[161] found that a tooth restored with a temporary post-crown combination had significantly greater leakage than definitively cemented prefabricated posts and separate crowns. Similar results were found by Fox and colleagues[162] when they compared cast post and cores cemented with zinc phosphate cement, prefabricated posts and composite resin cores cemented with resin cement, and provisional post-crowns cemented with zinc oxide-eugenol cement. In a 3-year retrospective clinical study, Lynch and colleagues[163] evaluated 176 endodontically treated teeth. They found that the loss of endodontically treated teeth occurred more often with those teeth restored with provisional restorations. Following these results, several studies[150,162] suggested that the definitive post and restoration

should be cemented as soon as possible in order to prevent recontamination of the root canal. However, to minimize leakage and enhance long-term success, the definitive coronal restoration should be of superior quality.[158,165–168]

Temporary Coronal Access Filling

Balto and colleagues[150] compared the leakage of different provisional materials used in post-prepared root canals. They found that none of the provisional restorations tested (Cavit, IRM, and Temp Bond) prevented coronal leakage when left for a long period of time (30 days). Safavi and colleagues[164] evaluated the prognosis of endodontically treated teeth following delayed placement of the definitive coronal restoration. A total of 464 endodontically treated teeth were followed radiographically. They found a higher success rate when the definitive restorations (silver amalgam, composite resin, or definitive restoration with or without post and cores) were placed as compared with teeth restored with provisional coronal access restorations (IRM or Cavit).

Torabinejad and colleagues[158] found that defective restorations could cause root canal system reinfection within 19 days. Ray and Trope[165] found that a combination of a poor coronal restoration and poor endodontic treatment resulted in a high failure rate of endodontically treated teeth. In a retrospective study, Iqbal and colleagues[166] found that a combination of good quality endodontic treatment and coronal restoration leads to a high success rate of endodontically treated teeth. Tronstad and colleagues[167] indicated that the quality of the endodontic treatment was more important than that of the coronal restoration.

CONCLUSIONS

Following endodontic treatment, post-space preparation should be performed and a post definitively cemented as soon as possible: the same day for a prefabricated post, and as soon as possible for a custom-fabricated post and core. The prepared tooth should then be restored with a well-fitting provisional restoration (good marginal seal and occlusion) followed by cementation of the definitive crown in as short a time as possible.

DOES THE USE OF A CERVICAL FERRULE (CIRCUMFERENTIAL BAND OF METAL) THAT ENGAGES TOOTH STRUCTURE HELP PREVENT TOOTH FRACTURE?

Survey data have been published that indicates the percentage of respondents who felt a ferrule (circumferential band of metal) increased a tooth's resistance to fracture.[53] Fifty-six percent of general dentists, 67% of prosthodontists, and 73% of board-certified prosthodontists felt that core ferrules increased a tooth's fracture resistance. To investigate this concept, several research studies have been performed. Some of the articles indicate ferrules are beneficial whereas others found no increase in fracture resistance.

The results appear indecisive until differences between study designs are analyzed. First, some of the studies tested ferrules that were part of a cast metal core (core ferrules),[169–173] whereas other studies evaluated the effectiveness of ferrules created by the overlying crown engaging tooth structure.[174–187] One study evaluated both core ferrules and crown ferrules.[188] Second, there were differences in the form of the ferrule, and therefore, the manner by which the metal engaged tooth structure (beveled sloping surface versus extension over relatively parallel prepared tooth structure). Third, there were variations in the amount of tooth structure encompassed by the ferrules.

The data generally indicates that ferrules formed as part of the core are less effective than ferrules created when the overlying crown engages tooth structure. In four of the six core ferrule studies, they were found to be ineffective.[170,171,173,188] Also, in one of the two studies where the core ferrule was effective, the ferrule form was a 2-mm parallel extension of the core over tooth structure as opposed to a bevel.[169] In the other study where core ferrules were found to be effective, a torsional force was used as opposed to an angular lingual force.[172] In the crown ferrule studies, most of the ferrules effectively increased a tooth's resistance to fracture. Only when the crown ferrule was of minimal dimension or a sloping form, it was found to be ineffective.[174,188] In support of these studies, Rosen and Partida-Rivera[189] found that a 2-mm cast gold collar (not part of the post and core) was very effective in preventing root fracture when a tapered screw post was intentionally threaded into roots so as to induce fracture. Assif and colleagues[190] found no difference in the tooth fracture patterns of parallel posts, tapered posts and parallel posts with a tapered end when they were covered by a crown that grasped 2 mm of tooth structure. Akkayan and colleagues[181] found no significant difference between fiber-reinforced and zirconia dowels when the ferrule length was 2 mm.

The data also supports the concept that ferrules that grasp larger amounts of tooth structure are more effective than those engaging only a small amount of tooth structure. In both the core and crown ferrule studies the tooth's resistance to fracture was increased when a substantive amount of tooth structure was engaged (2 mm in the core ferrule studies and 1 to 2 mm in the crown ferrule studies). Libman and Nicholls[174] found the 0.5- to 1.0-mm crown ferrule to be ineffective whereas a 1.5- to 2.0-mm crown ferrule to be effective. Isidor and colleagues[176] determined that increasing crown ferrule length significantly increased the number of cyclic cycles required to cause specimen failure. They compared no ferrule with 1.25 and 2.55 mm crown ferrules. They concluded that ferrule length was more important than post length in increasing a tooth's resistance to fracture under cyclic loading. Zhi-Yue and Yu-Xing[180] found that a 2-mm crown ferrule effectively enhanced the fracture strength of endodontically treated teeth when a cast post and core was used. Pereira and colleagues[187] compared the effect of no crown ferrule with 1-, 2-, and 3-mm crown ferrules. They found that a 3-mm crown ferrule

significantly increased the fracture resistance of endodontically treated teeth compared to a 2-mm crown ferrule.

The form of the prepared ferrule also appears to affect a tooth's fracture resistance in the previously cited studies. Only one beveled/sloping ferrule was effective in enhancing a tooth's fracture resistance and that was when a torsional force was applied to the tooth. Tan and colleagues[183] compared a 2-mm uniform crown ferrule that extended around the entire crown circumference with a 2-mm nonuniform crown ferrule where the ferrule was only 0.5 mm on the proximal surfaces. The uniform ferrule produced significantly greater fracture resistance than a nonuniform ferrule. In an *in vitro* study, Naumann and colleagues[184] evaluated the effect of chewing simulation on the fracture resistance of maxillary endodontically treated teeth with incomplete crown ferrules. The greatest variation of failure load was associated with the absence of portions (facial, palatal, or interproximal) of the crown ferrule. Ng and colleagues[185] investigated the effect of limited residual axial tooth structure on the fracture resistance of maxillary anterior endodontically treated teeth. They found that in the absence of 360° of circumferential coronal tooth structure, the location of the remaining coronal tooth structure may be an important factor for determining the fracture resistance of endodontically treated teeth. The palatal axial wall was as effective as a 360° circumference in providing fracture resistance.

CONCLUSIONS

Differences of opinion exist regarding the effectiveness of ferrules in preventing tooth fracture. Ferrules have been tested when they are part of the core and also when the ferrule is created by the overlying crown engaging tooth structure. Most data indicates **a ferrule created by the crown** encompassing tooth structure **is more effective** than a ferrule that is part of the post and core (Figure 35-15). Ferrule effectiveness is enhanced by grasping larger amounts of tooth structure. The amount of tooth structure engaged by the overlying crown appears to be more important than the length of the post in increasing a tooth's resistance to fracture. **Ferrules are more effective** when the crown encompasses relatively **parallel prepared tooth structure** than when it engages beveled/sloping tooth surfaces. Ferrules that encompass 2 mm of tooth structure around the entire circumference of a tooth are more effective than nonuniform ferrules.

POST AND CORE PLACEMENT TECHNIQUES

Pretreatment Data Review

When it has been determined that a post and core is required to properly retain a definitive single crown or fixed partial denture of an endodontically treated tooth, the following characteristics should be determined prior to beginning the clinical procedures associated with the fabrication of a post and core.

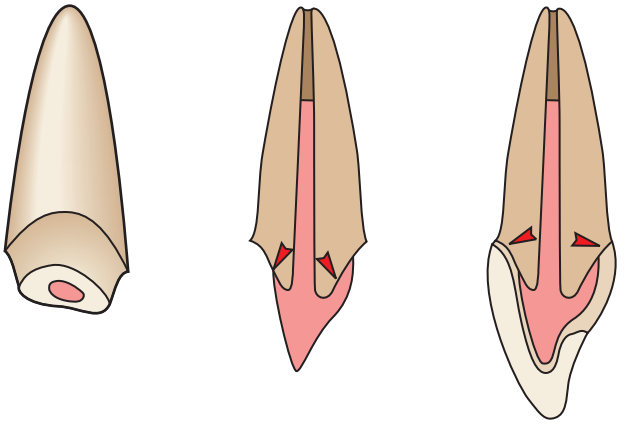


FIGURE 35-15 Types of ferrules. ***A.*** Tooth prepared for a post and core. ***B.*** Post and core has been cemented into the tooth. The arrows note how the core has created a ferrule around the tooth (note the reduction in the width of remaining coronal tooth structure to develop this core ferrule). ***C.*** A metal ceramic crown has been cemented over the core. The arrows show how the crown encompasses the tooth cervically, establishing a crown ferrule.

Post Length

Since 5 mm of gutta-percha should be retained apically to assure a good seal (as measured radiographically), posts should be extended to that length in all teeth except molars. With molars, posts should be placed in the primary roots (palatal root of maxillary molars and distal roots of mandibular molars) and should not be extended more than 7 mm apical to the origin of the root canal in the base of the pulp chamber (Figure 35-11). Extension beyond this length can lead to root perforation or only very thin areas of remaining tooth structure.

Post Diameter

A frequently used and clinically appropriate guideline for post diameter is not to exceed one-third the root canal diameter. It has been determined that when a root canal is prepared for a post and the diameter is increased beyond one-third of the root diameter, the tooth becomes exponentially weaker. Each millimeter of increase (beyond one-third the root diameter) causes a six-fold increase in the potential for root fracture.[73] Based on measuring the root dimensions of 1,500 teeth (125 of each tooth) and using the guideline that the post should be one-third the root diameter, optimal post diameter measurements have been determined to be approximately 0.6 mm for mandibular incisors, 1.0 mm for maxillary central incisors, maxillary and mandibular canines and the palatal root of the maxillary first molar.[135] The recommended post diameter for the other teeth was 0.8 mm.[135] Another study of 700 teeth recommended that post diameter should range from 0.7 mm for mandibular incisors to a maximum of 1.7 mmm for maxillary central incisors.[113]

Anatomic and Structural Limitations

The practitioner who completed the endodontic treatment is ideally suited to identify characteristics of the pulp chamber,

rooted canal(s) anatomy, and completed endodontic filling that should be reviewed before placing a post and core. These characteristics include the presence and extent of dentinal craze lines, identification of teeth where further root preparation (beyond that needed to complete endodontic instrumentation) will result in less than 1 mm of remaining dentin or a post diameter greater than one-third of the root diameter area, information regarding areas where the remaining tooth structure is thin, and the point at which significant root curvature begins.

Craze Lines

Craze lines in dentin are areas of weakness where further crack propagation may result in root fracture and tooth loss. The patient should be informed of their presence with appropriate chart documentation of crack location. It is prudent to avoid post placement, if possible, in favor of a restorative material core. If a post is required, it should passively fit the canal and the definitive restoration should entirely encompass the cracked area, whenever possible, by forming a ferrule.

Dentin Thickness After Endodontic Treatment

Following normal and appropriate endodontic instrumentation, teeth can possess less than 1 mm of dentin, indicating there should be no further root preparation for the post. When these teeth are encountered, it is best to fabricate a post that fits into the existing morphologic form and diameter rather than additionally preparing the root to accept a prefabricated type of post. This characteristic is one of the primary indications for the use of a custom cast post and core. One study determined that canines (maxillary and mandibular), maxillary central and lateral incisors, and the palatal root of maxillary first molars possessed more than 1 mm of dentin after endodontic cleaning and shaping.[192] All other teeth had roots with less than 1 mm of remaining dentin following endodontic treatment. With the goal of preserving 1 mm of remaining dentin lateral to posts, it has been determined that single-canal maxillary first premolars should have posts that are 0.7 mm in diameter or less.[137] Mandibular premolars with oval/ribbon-shaped canals should not be subjected to any preparation of the root canal for a post since it will result in less than 1 mm of dentin.[192] Preparation of the mesial root canals in mandibular molars and the facial root canals in maxillary molars can result in perforation or only thin areas of remaining dentin. Based on the measurements of residual dentin thickness, it is recommended that posts not be placed in these roots if possible.

Root Curvature

When root curvature is present, post length must be limited so as to preserve remaining dentin, thereby helping to prevent root fracture or perforation. Root curvature occurs most frequently in the apical 5 mm of the root. Therefore, if 5 mm of gutta-percha is retained apically, curved portions of the root are usually avoided. As discussed previously under post length, molar posts should not exceed 7 mm in the roots because of the potential for perforation due to root curvature and the presence of developmental root depressions. Molar roots are frequently curved and the post should terminate at the point where substantive curvature begins.

Type of Post and Core

Custom Cast Post

For many years, custom cast post and cores have been considered to be the standard of care when restoring endodontically treated teeth and have historically been made of metal. Gold, silver-palladium, and base metal alloys are the most commonly used metals. For economic reasons, base metal alloys were introduced as an alternative metal to high-noble alloys. The major disadvantages of base metal alloys are their manipulation (laboratory and clinical), their hardness, and their unstable chemical structure.[193] The degradation of base metal alloys releases substances that could be harmful to the patient.[193–195] Silver-palladium alloys were introduced as a replacement for gold and base metal alloys. They are relatively easy to manipulate and have many properties similar to those of gold casting alloys.[196] However, the castability of silver-palladium alloys was shown to be inferior to that of gold-based alloys.[193,197,198]

Cast posts and cores can be fabricated by using either a direct or an indirect technique, and several methods have been described for the intraoral fabrication of an acrylic resin pattern for a direct post and core.[199–203] Prefabricated plastic patterns are commonly used, and they are relined to fit the post space with an autopolymerizing acrylic resin (Figure 35-16). The coronal adaptation of the tooth is usually completed using the same resin, and the core contoured intraorally to the desired form. The only disadvantage of this direct technique is the amount of chairside time required to perform the procedure.

As an alternative, indirect techniques for the fabrication of cast post and cores have been proposed.[204–206] However, this procedure requires meticulous attention to a defined protocol to insure success. One technique uses a lentulo spiral instrument to carry impression material to the apical aspect of the prepared post space. Since a thin projection of polymerized impression material can be distorted or torn upon removal from the mouth, reinforcement of the impression material is required. This reinforcement can be accomplished by using several materials such as a plastic pin or metal wire. Care must be taken when using plastic pins to ensure that they are not slightly flexed by placement into a curved canal or through contact with the impression tray, thereby allowing a return to their original form upon impression removal with some resulting distortion. The use of a portion of a safety pin is an excellent material as long as it is made of spring steel, thereby preventing flexion. A bendable metal pin can be distorted upon impression removal. Complete seating of a section from a safety pin until it contacts the gutta-percha is recommended. Upon removal of the impression from the mouth, the presence of the metal pin at the apical extension of the impression material indicates there was no elongation of the impression material upon removal.

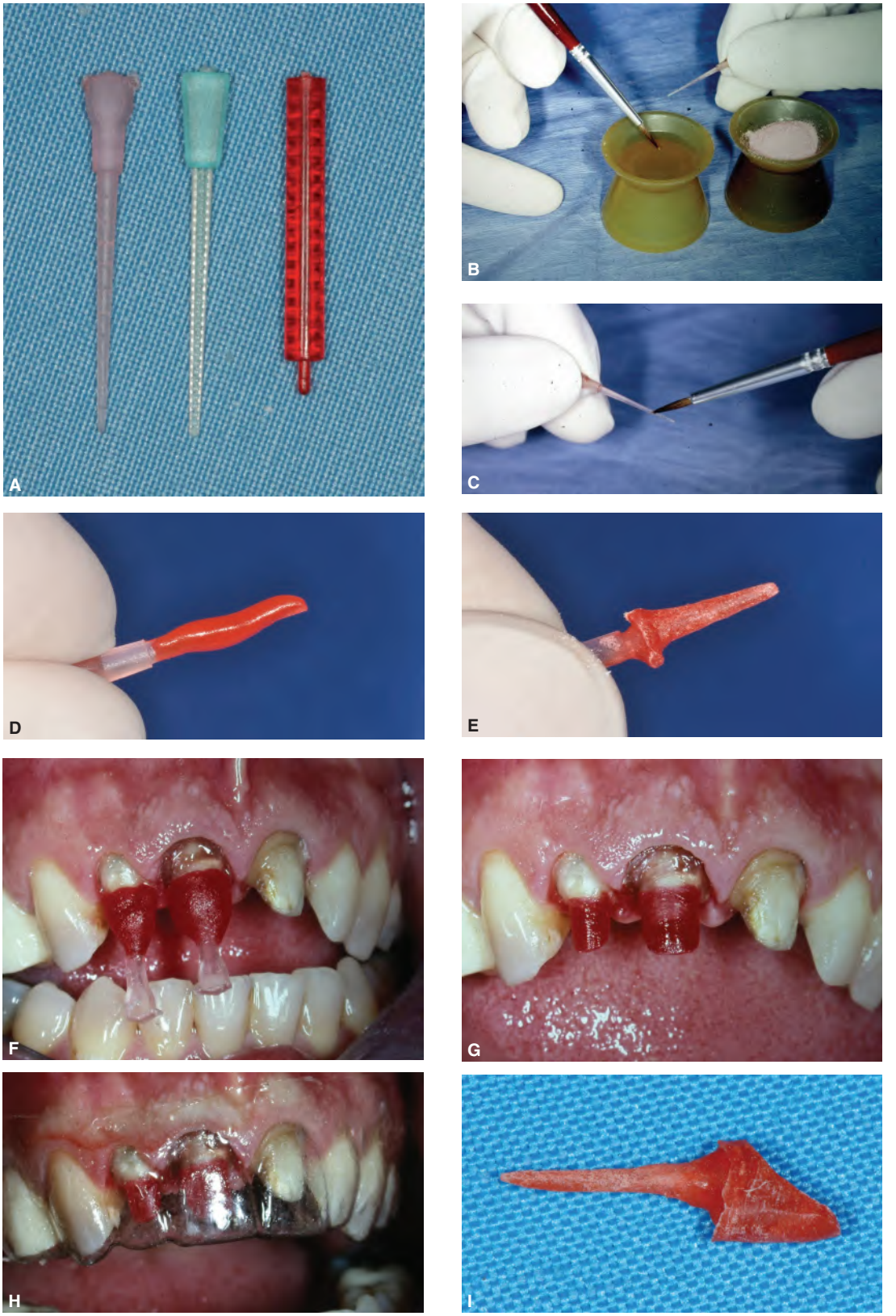


FIGURE 35-16 ***A.*** Prefabricated plastic patterns commonly used for the direct technique method for the intraoral fabrication of an acrylic resin pattern for a cast post and core. ***B, C.*** Monomer is applied to the body of the plastic post. ***D.*** Resin is applied to the body of the post using the bead-brush technique. ***E.*** Polymerized pattern is removes from the post space and inspect for integrity and lack of voids. ***F.*** Coronal resin is added so as to form the desired dimensions of the core. ***G.*** The resin is prepared with a high-speed diamond and water spray to resemble a complete crown tooth preparation. ***H.*** Core reduction is verified using a vacuum formed template fabricated from the diagnostic wax pattern. ***I.*** The resin pattern post and core is removed and sent to the laboratory for investing and casting. (Figure 35-16B to I reprinted with permission from Baba NZ.[331])

To make the impression technique easier, prefabricated precision plastic dowels were introduced.[207,208] After the selection of the desired diameter for the dowel, a corresponding drill is used to prepare the post space at the appropriate length. The dowel is then inserted into the prepared canal, picked up in an impression, and transferred to the dental laboratory for fabrication.

A proposed advantage of cast post and cores is their purported ease of removal in case of an endodontic retreatement.[209–211] In addition, several long-term clinical studies have reported high success rates with cast post and cores.[60,212]

Prefabricated Post

Prefabricated posts have become quite popular and a wide variety of systems are available: parallel-sided or tapered, smooth or serrated, passive (cement/bonded) or active (threaded) or combinations of these.[213–215] Threaded posts depend primarily on engaging the tooth—either through threads formed in the dentin as the post is screwed into the root or through threads previously "tapped" into the dentin. The majority of these posts are metallic. Recently, in response to a need for tooth-colored posts, several nonmetallic posts such as carbon fiber epoxy resin, zirconia, glass fiber-reinforced (GFR) epoxy resin, and ultra high strength polyethelene fiber-reinforced (PFR) posts are available and early data indicates they can be acceptable alternatives to metallic posts.

The carbon fiber-reinforced (CFR) epoxy resin post system was developed in France in 1988 by Duret and Renaud[216–218] and first introduced in Europe in the early 1990s.[219–221] The matrix for this post is an epoxy resin reinforced with unidirectional carbon fibers parallel to the long axis of the post. The fibers are 8 μm in diameter and uniformly embedded in the epoxy resin matrix. By weight, the fibers comprise 64% of the post and are stretched before injection of the resin matrix to maximize the physical properties of the post (Figure 35-17).[216,222,223] The post is reported to absorb applied stresses and distribute these stresses along the entire post channel.[224] The bulk of the carbon fibers is made from polyacrylonitrile by heating it in air at 200°C to 250°C and then in an inert atmosphere at 1200°C. This process removes hydrogen, nitrogen and oxygen, leaving a chain of carbon atoms and forming carbon fibers.[225]

The CFR post has been reported to exhibit high fatigue strength, high tensile strength, and a modulus of elasticity similar to dentin.[219,222,226–229] The post was originally radiolucent; however, a radiopaque post is now available. Radiopacity is produced by placing traces of barium sulfate and/or silicate inside the post. Mannocci and colleagues[230] examined radiographically five different types of fiber posts. They found that only Composiposts and Snowposts had uniform radiopacity. Finger and colleagues[231] examined the radiopacity of seven fiber-reinforced resin posts compared to a titanium post. Compared to other posts, they found CFR posts had an acceptable radiopacity.

The posts are available in different shapes: double cylindrical with conical stabilization ledges or conical shapes (Figure 35-18). The surface texture of the post may be smooth or serrated. Studies have indicated that serrations increased mechanical retention although the smooth post also bonded well to adhesive dental resin.[228,232] The post has a surface roughness of 5 to 10 μm to enhance mechanical adhesion of the autopolymerizing luting material, and the post appears to be biocompatible based on cytotoxicity tests.[227,233]

Several studies indicate that CFR posts exhibit adequate physical properties compared to metal posts.[223,226,234,235] In a retrospective study over 4 years, Ferrari and colleagues[234] indicated that the Cosmopost system was superior to the

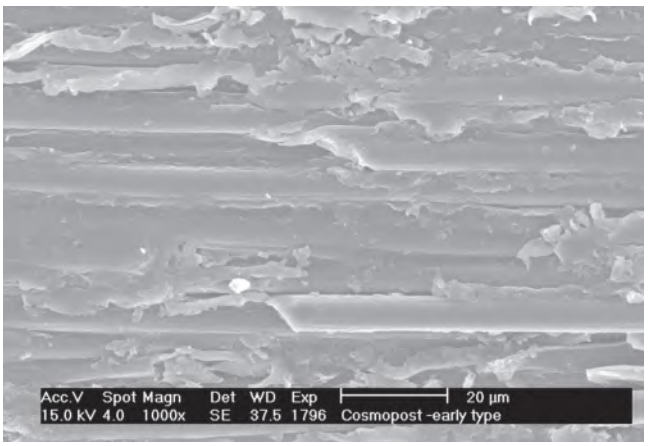


FIGURE 35-17 Surface texture of a carbon fiber-reinforced epoxy resin dowel (SEM magnification: ×1000).

FIGURE 35-18 Available shapes of carbon fiber-reinforced epoxy resin posts.

conventional cast post and core system. King and Setchell [226] and Duret and colleagues[223] evaluated the physical properties (fracture resistance and modulus of elasticity) of CFR posts and both reported that these posts are stronger than prefabricated metal posts.

Contrasting results were reported by Sidoli and colleagues[236] in an *in vitro* study. They found that CFR posts exhibited inferior strength when compared with metallic posts. Similar results were also obtained by Purton and Love[237] and Asmussen and colleagues.[238]

Martinez-Insua and colleagues[239] studied the fracture resistance of teeth restored with CFR posts and cast posts. They reported a significantly higher fracture threshold for cast post and cores. A clinical evaluation of CFR posts suggested that these posts did not perform as well as conventional cast post and cores.[240] However, the results of this study must be interpreted with caution because of the relatively small sample size (27 teeth) used in this study.

Multiple studies indicate a decrease in the strength of CFR posts after thermocycling and cyclic loading.[241–244] In addition, contact of the post with oral fluids reduced their flexural strength values.[233–245] In two studies, results showed no significant difference among a CFR posts, cast post and cores, and metal posts when restoring mandibular incisor teeth. No differences were noted in the mode or state of fracture observed.[246,247]

Multiple *in-vitro* studies have reported that CFR posts are less likely to cause fracture of the root at failure. The mode of failure of teeth restored with CFR posts in these studies was more favorable to the remaining tooth structure.[181,226,236,239,248–252] However, despite all these advantageous properties, one should question certain *in vivo* applications of the CFR post. When the ferrule is small or absent in an endodontically treated tooth restored with a CFR post, loads may cause the post to flex causing a micromovement of the entire core. The cement seal at the margin of the crown can be compromised, accompanied by microleakage of oral bacteria and fluids. As a result, secondary caries may develop in the space and may not be easily detected.[252]

The short-term clinical studies of CFR posts appear promising but some of the long-term studies report higher failure rates. Wennström[210] restored 173 teeth with CFR posts and reported two failures after a period of 3 to 4 years. A short-term retrospective study of 236 teeth treated 2 to 3 years previously reported no failures of the posts.[253] Ferrari and colleagues[234] studied 100 CFR posts and reported a failure rate of 3.2% after a mean time of 3.8 years of clinical service. In a prospective study of 59 CFR posts, Glazer and colleagues[254] indicated that these posts did not fracture but there was a 7.7% absolute failure rate. Another retrospective study[255] of 1,304 CFR posts followed from 1 to 6 years found that a 3.2% failure rate that was attributed to debonding during removal of temporary crowns and periapical lesions. Hedlund and colleagues[256] studied the clinical performance of 65 CFR posts for an average of 2.3 years of clinical service and found a 3% failure rate. In a retrospective study of 64 CFR posts, Segerström and colleagues[257] found that nearly 50% of the CFR posts were lost after a mean time of 6.7 years.

Should fracture of CFR posts occur, a potential advantage is the relative ease of removal from the post space compared to metal posts. A removal kit has been suggested for this procedure and the latter is recommended as a single use only item.[258–262]

Glass fiber-reinforced (GFR) epoxy resin posts The high demand for esthetic restorations and all-ceramic crowns led to the development of a variety of tooth-colored post systems as an alternative to metal and CFR posts. The GFR epoxy resin post is made of glass or silica fibers (white or translucent). Glass fiber posts can be made of different types of glasses: electrical glass, high-strength glass, or quartz fibers (Figure 35-19).[244,263] The commonly used fibers are silica-based (50 to 70% SiO_2), in addition to other oxides.[264]

The GFR post is available in different shapes: cylindrical, cylindroconical, or conical shape (Figure 35-20). An *in vitro* assessment of several GFR post systems indicated that parallel-sided GFR posts are more retentive than tapered GFR posts.[265]

The composition of the glass fibers in the matrix tends to play an important role in the strength of the post. Newman and colleagues[243] compared the fracture resistance of two GFR posts containing different weight percentages of glass fibers. They found that the higher content of glass fibers in

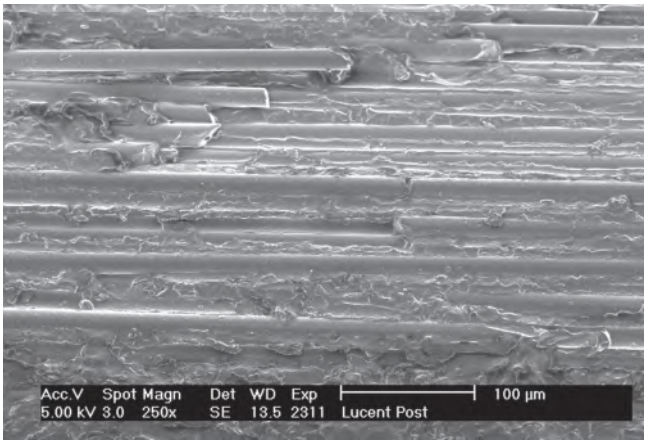


FIGURE 35-19 Surface texture of a glass fiber-reinforced epoxy resin post (SEM magnification: ×250).

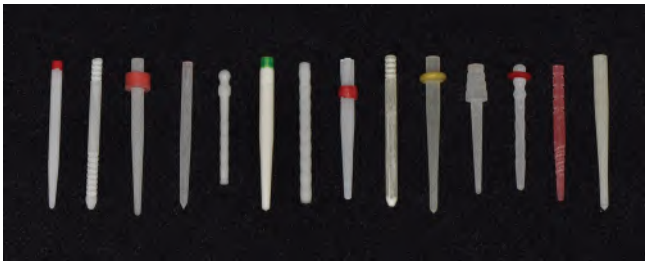


FIGURE 35-20 Available shapes and designs for glass fiber-reinforced epoxy resin posts. (Reprinted with permission from Baba NZ.[331])

the post contributed to the greater strength displayed by the tested post.

The GFR post has been reported to exhibit high fatigue strength, high tensile strength, and a modulus of elasticity closer to dentin than that of CFR posts.[229,238,266] The GFR post is as strong as the CFR post and approximately twice as rigid.[267]

The flexural strength of GFR posts is not related to the type of glass fiber used. Galhano and colleagues[229] evaluated the flexural strength of carbon fiber, quartz fiber, and glass fiber posts. They found that the posts behaved similarly because of the same concentration and type of the epoxy resin used to join the fibers together. Pfeiffer and colleagues[268] evaluated, *in vitro*, the yield strength of GFR post, titanium, and zirconia posts. They found that the yield strength was significantly higher for the zirconia and titanium posts when compared with GFR posts.

Several studies have determined that there is a decrease in the strength (about 40%) of GFR posts after thermocycling and cyclic loading. In addition, contact of the post with oral fluids (short- and long-term) reduced their flexural strength.[241,244,269–271]

Two studies indicate that the tensile bond strength between the composite resin core material and the GFR post is less than that developed with a titanium post.[272,273] Other studies indicated that there was a good adhesive bond between the GFR post and composite resin cements.[272,274,275] The bonding of the core to the post can be improved by treating the post with airborne-particle abrasion.[276] Similar results were obtained by treating the surface of the post with hydrogen peroxide and silane or hydrofluoric acid and silane.[277,278] During fatigue loading, a composite resin core bonded to a GFR post provided significantly stronger crown retention than cast gold posts and cores and titanium posts with composite resin cores.[182]

Similarly to CFR posts, multiple studies have shown that GFR posts are less likely to cause fracture of the root at failure.[243,279–281] The mode of failure of teeth restored with CFR posts in these studies was more favorable to the remaining tooth structure. However, studies have discussed the importance of the presence of a ferrule effect in achieving a high success rate.[181,184,185,282] Malferrari and colleagues[281] restored 180 teeth with GFR posts and reported no post, core, or root fracture after 30 months of service. Naumann and colleagues[283] found that the survival rate of parallel-sided and tapered GFR posts was similar.

Polyethylene fiber-reinforced posts (PFR) are made of ultra high molecular weight polyethylene woven-fiber ribbon (Ribbond, Ribbond Inc, Seattle, Washington). They are not post and cores in the traditional sense. It is a polyethylene-woven fiber ribbon that is coated with a dentin bonding agent and packed into the canal, where it is then light polymerized in position.[284–286] The Ribbond material has a three-dimensional structure due to either a leno weave or a triaxial architectural design (Figure 35-21). These designs are composed of a great number of nodal intersections that prevent crack propagation and provide a mechanical retention for the composite resin cement. When PFR posts were compared with metal posts in the laboratory, the fiber-reinforced posts reduced the incidence of vertical root fracture. The addition of a small-size prefabricated post to the PFR post increased the strength of the post-and-core complex. However, the strength of the PFR post did not approach that of a cast metal post and core.[284]

FIGURE 35-21 Close-up of polyethelene fiber-reinforced post material in its original package. (Reprinted with permission from Baba NZ.[331])

When compared to other fiber-reinforced composite post systems the PFR posts were also found to protect the remaining tooth structure.[243] These results may be attributed to the manufacturer's recommendations not to enlarging the root canals, not to removing undercuts present in the root canal, and forming a 1.5 to 2 mm crown ferrule. The presence of a large volume of core material, and a sufficient dentin bonding area coronally seems to greatly affect the mean load-to-failure value of PFR posts.[243] Eskitascioglu and colleagues[287] evaluated two post and core systems using a fracture strength test and a finite element analysis. They found that stress accumulated along the cervical region of the tooth and along the buccal bone. Minimum stress was recorded within the PFR post system. They suggested that the PFR post could be advantageous for the restoration of teeth with apical resection.

Newman and colleagues[243] compared the effect of three fiber-reinforced composite post systems on the fracture resistance of endodontically treated teeth. They found that when PFR posts were placed in narrow canals, they performed better than GFR posts. They suggested the PFR post be formed to the shape of the canal.

The use of PFR posts to restore endodontically treated teeth appears to be a promising alternative to the stainless steel and zirconia dowel posts with respect to microleakage.[288] Usumez and colleagues[288] compared in vitro the microleakage of three esthetic, adhesively luted dowel systems with a conventional dowel system. They found that the PFR posts and the GFR posts exhibited less microleakage compared to zirconia posts.

Zirconia posts The trend toward the use of all-ceramic crowns has encouraged manufacturers to explore the development of all-ceramic posts.[289–292] A metal-free post avoids the

discoloration of tooth structure that can occur with metal posts and produces optical properties comparable to all-ceramic crowns.[293–296] One type of all-ceramic post is the zirconia post, composed of zirconium oxide (ZrO_2), an inert material used for a range of applications. Its high fracture toughness, high flexural strength and excellent resistance to corrosion encouraged orthopedists to use it at articulation surfaces.[297] Studies have suggested that zirconia specimens transplanted in animals were very stable after long-term aging, and there was no apparent degradation of the specimens.[297–301]

Zirconia (Tetragonal Zirconium Polycrystals, TZP) exhibits phase transformation. Low-temperature degradation of TZP is known to occur as a result of spontaneous phase transformation of tetragonal zirconia to monoclinic phase during ageing at 130 to 300°C possibly within a water environment. It has been reported that this degradation leads to a decrease in strength due to the formation of microcracks accompanying the phase transformation. To inhibit this phase transformation, certain oxides (magnesium, yttrium or calcium oxide) are added to fully or partially stabilize the tetragonal phase of zirconia at room temperature. This mechanism is known as transformation toughening.[292,298,302–304]

The type of zirconia used for dental posts is composed of TZP with 3% mol yttrium oxide (Y_2O_3) and is called YTZP (Yttria-stabilized tetragonal polycrystalline zirconia).[292,305] YTZP is composed of a dense fine-grained structure (0.5 μm average diameter) that provides the post with toughness and a smooth surface.[303,305,306] The zirconia post is extremely radiopaque, biocompatible, possesses high flexural strength and fracture toughness, and may act similar to steel.[298–302,307–315] In addition, the post has a low solubility[312] and is not affected by thermocycling.[241] The post is available in a cylindroconical shape (Figure 35–22).

The Zirconia post has a smooth surface configuration with no grooves, serrations or roughness to enhance mechanical retention (Figure 35-23). As a result the zirconia post does not bond well to composite resins and may not provide the best support for a brittle all-ceramic crown. [272,317–319] Dietshi and colleagues[318] found that these posts also have poor resin-bonding capabilities to dentin after dynamic loading and thermocycling due to the rigidity of the post. In a cyclic loading test performed in a wet environment, Mannoci and colleagues[250] found that the survival rate of zirconia posts compared to fiber posts was significantly lower.

In vitro studies[272,275,319,320] indicated that the smooth surface configuration of untreated zirconia posts leads to failure at the cement/post interface. The vast majority of the cement remained in the root and was not attached to the zirconia dowels. Wegner and Kern[321] evaluated the bond strength of composite resin cement to zirconia posts. They found that the long-term bond strength of the composite resin cement to zirconia posts is weak. Several studies found that acid etching and silanization of zirconia posts does not improve the strength of the resin bond to the zirconia-based material because of the lack of or no silica content in the post.[321–324] However, tribochemical silica coating was found to increase the bond strength of composite resin to the zirconia post.[325,326]

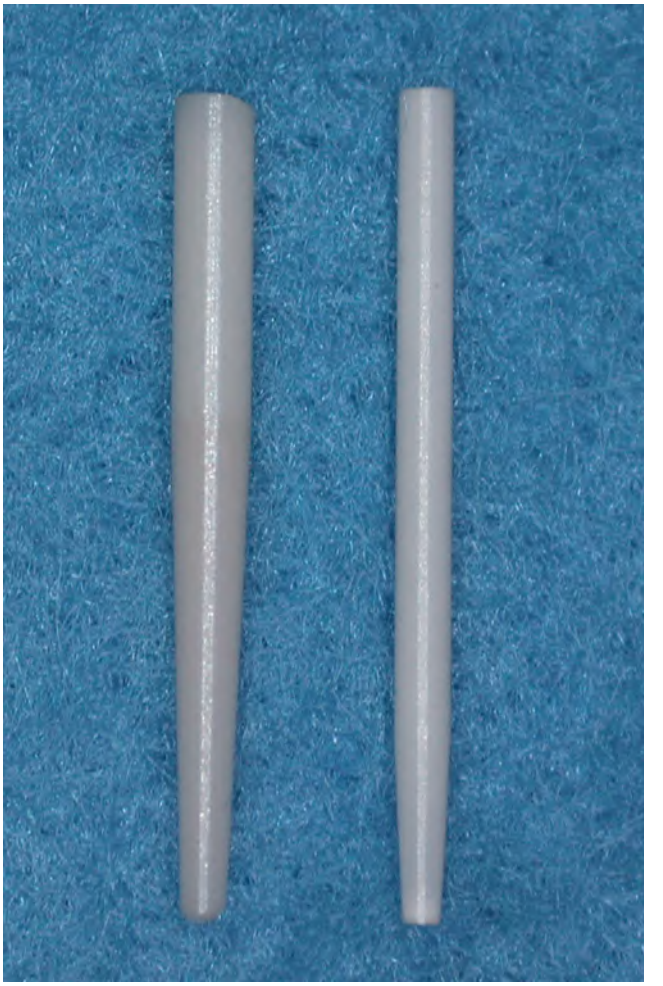


FIGURE 35-22 Available shapes and designs for zirconia posts.

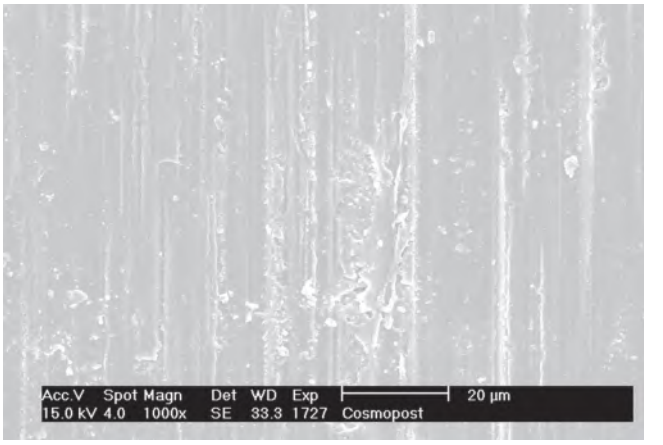


FIGURE 35-23 Surface texture of zirconia post (SEM magnification: ×1000).

Oblak and colleagues[327] compared the fracture resistance of prefabricated zirconia posts after different surface treatments. They found that airborne particle-abraded posts exhibited significantly higher resistance to fracture than posts that have been ground with a diamond instrument.

The use of heat-pressed glass to form the core instead of composite resin has been suggested.[212,310,311] This approach

may improve the physical properties of the all-ceramic post and core.

When the mechanical properties of zirconia posts were evaluated it was reported that these posts are very stiff and strong, with no plastic behavior.[238,309,310] Pfeiffer and colleagues[268] found that the zirconia post had a significantly higher yield strength compared with titanium and GFR posts.

Several studies indicated that many commonly used posts exhibit higher fracture resistance than zirconia posts.[249,308,328,329] In addition, once they fracture, the irretrievable posts will leave unrestorable roots.[181,329] Nothdurft and colleagues[330] evaluated the clinical performance of 30 zirconia posts in a short-term retrospective study. They found no signs of failure of these posts. However, these results should be analyzed with caution because of the small sample size and a short follow-up time.

Root Selection for Multirooted Teeth

Premolars

When post and cores are needed in prelomars, posts are best placed in the palatal root of maxillary premolar and the straightest root for the mandibular canal. The buccal root could be prepared to a depth of 1 to 2 mm and to serve as an antirotational lock, if needed.

Molars

When posts and cores are needed in molars, posts are best placed in roots that have the greatest dentin thickness and the smallest developmental root depressions. The most appropriate roots (the primary roots) in maxillary molars are the palatal roots and in mandibular molars are the distal roots (Figure 35-24). The facial roots of maxillary molars and the mesial root of mandibular molars should be avoided if at all possible. If these roots must be used in addition to the primary roots, then the post length should be short (3 to 4 mm) and a small-diameter instrument should be used (no larger than a No. 2 Peeso instrument that is 1.0 mm in diameter). When 7-mm long posts were placed in the mesial root of mandibular molars, 20 of the 75 tested teeth had only a thin layer of remaining dentin or were perforated.[101]

Type of Definitive Restoration

It is important to know the type of single crown or retainer (all-metal, all-ceramic, metal ceramic) that will be used as the definitive restoration for each endodontically treated tooth that requires a post and core. This knowledge permits the tooth to be reduced in accordance with the reduction depths and form recommended for each type of crown/retainer.

Coronal Tooth Preparation

Restoration of endodontically treated teeth must be a team effort between the endodontist and the dentist responsible for the coronal restoration, requiring two-way communication. If it can be co-coordinated, the first step in the fabrication of a post and core should ideally be preparation of the coronal tooth (Figure 35-25) for the type of definitive restoration that will be placed (all-metal crown, metal ceramic crown, all-ceramic crown). This procedure will help in determining the structure of the post-core fabrication. Each type of restoration requires different amounts of tooth reduction, and the form of the tooth preparation varies considerably. By preparing the coronal tooth structure first, the structural integrity of remaining dentin and enamel can be assessed. When the remaining peripheral tooth structure is very thin and would likely not possess sufficient strength to resist occlusal forces transmitted through the crown to the tooth, the thin structure is removed and replaced as part of the core.

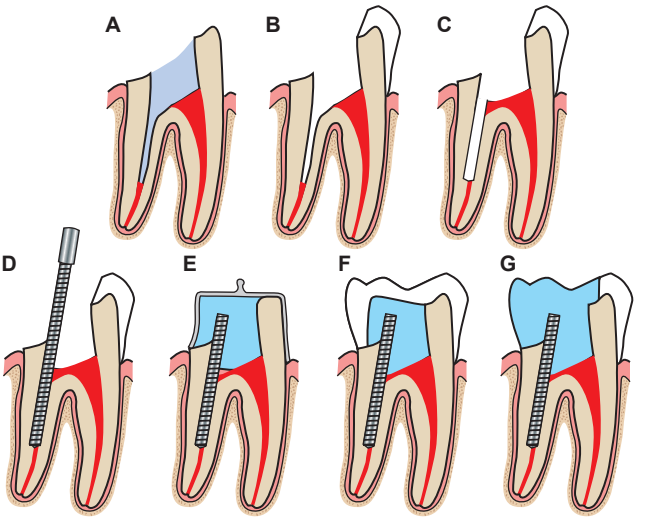


FIGURE 35-24 Placement of Para Post and restorative material core in a molar. ***A.*** Endodontic treatment completed. ***B.*** Provisional restorative material in the pulpal chamber has been removed and gutta-percha removed from the distal root canal. ***C.*** Post space formed with a drill. ***D.*** Trial placement of the post. ***E.*** The post has been shortened and cemented. A restorative material core has been formed. ***F.*** The tooth with core has been prepared, an impression made, and the definitive crown cemented. ***G.*** If there is an extended time delay between placement of the core and preparation of the tooth for a crown, the core can be built to full tooth contour to serve as the interim restoration.

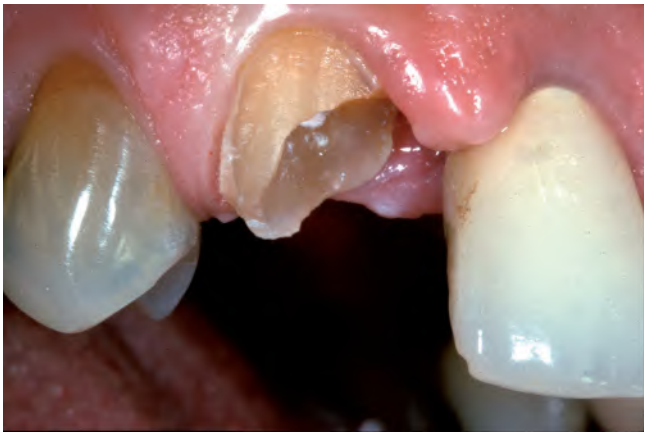


FIGURE 35-25 Coronal tooth preparation. The existing crown has been removed on an endodontically treated tooth. Initial reduction of the tooth has been completed to permit assessment of integrity of remaining coronal tooth structure.

This order of procedure also establishes morphologic borders that can be used to guide core fabrication so it is confluent with surrounding tooth structure and possesses the desired tooth preparation form.

Pulp Chamber Preparation

Treatment materials present in the pulp chamber following endodontic treatment (restorative materials sealing the coronal access and gutta-percha) are removed using rotary instruments (Figure 35-26). If a prefabricated post will be cemented into a root canal and a restorative material core built around the post, morphologic undercuts present in the pulp chamber should be retained for core retention. If a custom cast post and core is fabricated, then pulp chamber undercuts should be either blocked out with a definitive cement or a restorative material that is bonded to the tooth or the undercut eliminated by removing tooth structure. If removing the undercut through tooth preparation would result in substantive tooth structure removal that weakens the tooth, then blocking out the undercut is the treatment of choice.

Root Canal Preparation

The individual who completed the root canal is ideally suited to prepare the root canal, being the one that is most knowledgeable regarding root curvatures and areas where no further root preparation should be performed because it will result in areas of thin residual dentin. For this reason, it may be prudent to prepare the root canal for a post as a continuation of the endodontic treatment.

If the canal has not been prepared for a post as part of the endodontic treatment process, it will be necessary to remove the filling material using either a warm endodontic hand instrument or a slow-speed rotary instrument such as a Gates Glidden drill (Dentsply Maillefer North America, Tulsa, OK) or Peeso instrument (Dentsply Maillefer North America, Tulsa, OK). If a warm hand instrument is used, it is advisable to place a dental dam so as to prevent aspiration or swallowing of the hand instrument should it be dropped.

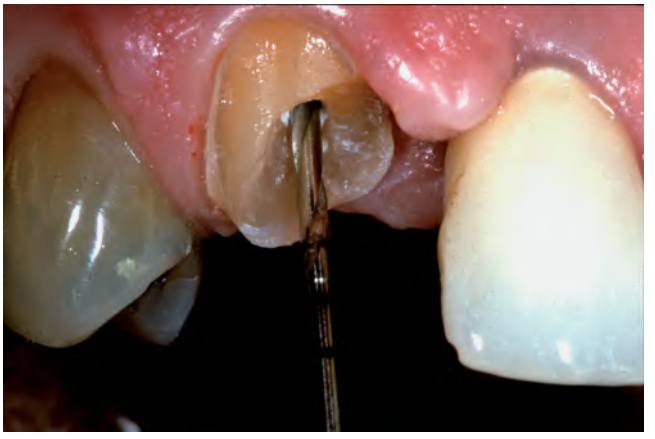


FIGURE 35-26 Pulp chamber preparation. Rotary instrument being used to remove provisional material.

Successful use of rotary instruments is related to initially using a small-diameter instrument (one that only removes the filling material without dentin removal). This small-diameter instrument is used to remove small vertical increments (1 to 2 mm) of the root canal filling material. After each vertical increment is removed, a visual inspection should be made to verify that the endodontic filling material is centered in the post preparation. The incremental root canal filling removal is continued until the appropriate length is established (Figure 35-27). Post preparation length is determined

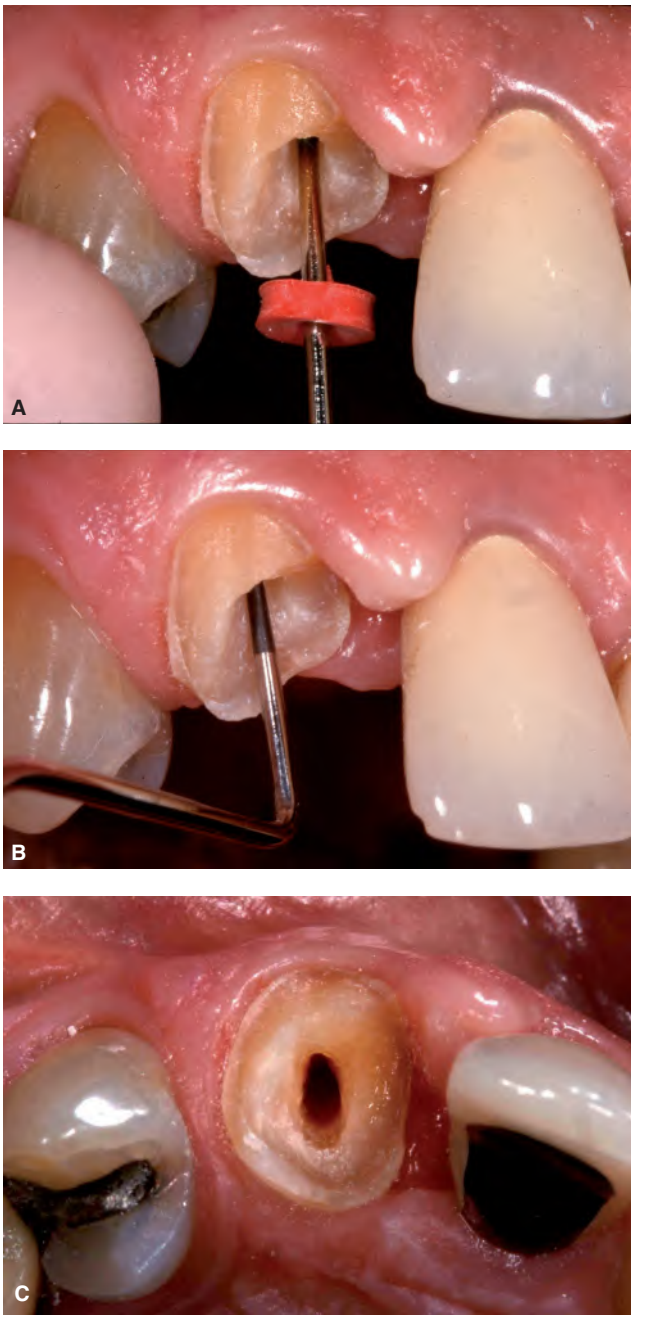


FIGURE 35-27 Root canal preparation. ***A.*** Rotary instrument being used to prepare post space in the root canal. Note the rubber ring around the instrument to identify the appropriate apical extension of the post preparation. ***B.*** Periodontal probe being used to measure post space depth. ***C.*** Post space preparation completed.

using a periodontal probe. Remaining gutta-percha length is evaluated by comparing periodontal probing depths with landmarks on the postendodontic radiograph and by making a radiograph of the prepared post space. After the length is established then any required increases to the post diameter are performed using incrementally larger rotary instruments or hand files.

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Chapter 36

Endodontic–Periodontal Interrelationships

Ilan Rotstein, Borja Zabalegui, Khalid Al-Hezaimi

ENDODONTIC–PERIODONTAL COMMUNICATION

Understanding the interrelationships between endodontic and periodontal diseases is crucial for correct diagnosis, prognosis, and treatment decision making. The dental pulp and periodontium are closely related, and pathways of communications between these structures often determine the progress of disease in these tissues.[1–4] The main pathways for communication between the dental pulp and the periodontium are (1) dentinal tubules, (2) lateral and accessory canals, and (3) the apical foramen.

Dentinal Tubules

Direct communication between the pulp and the periodontium may occur via patent dentinal tubules if the cementum layer is interrupted. This is usually attributed to developmental defects, disease processes, or surgical procedures involving root surfaces. Exposed dentinal tubules, in areas devoid of cementum, may serve as communication pathways between the pulp and the periodontal ligament (Figure 36-1).

Radicular dentin tubules extending from the pulp to the cemento-dentinal junction run a relatively straight course.[4] Their diameter ranges from 1 mm at the periphery to 3 mm near the pulp.[5] The tubular lumen decreases with age or as a response to chronic low grade stimuli causing apposition of highly mineralized peritubular dentin. The density of dentin tubules varies from approximately 15,000/mm^2 in the cervical portion of the root down to 8,000/mm^2 near the apex. At the pulpal end, however, tubular numbers increase to 57,000/mm^2.[5] When the cementum and enamel do not meet at the cemento-enamel junction (CEJ), these tubules remain exposed, thus creating pathways of communication between the pulp and the periodontal ligament. Cervical dentin hypersensitivity is a classic example of such tubular exposure.

Scanning electron microscopic studies have demonstrated that dentin exposure at the CEJ occurs in about 18% of teeth.[6] This incidence is even higher in anterior teeth reaching up to 25%.[6] The same tooth may also have several different CEJ characteristics, presenting dentin covered with cementum on one side whereas the other sides have dentin exposure.[7] Dentin exposure plays an important role in the progression of endodontic or periodontal pathogens, as well as cementum root scaling and planing, trauma, and chemically induced pathosis.[8–10]

Lateral and Accessory Canals

Lateral and accessory canals may be found anywhere along the root[11–16] (Figure 36-2). It is estimated that 30 to 40% of all teeth have lateral or accessory canals, mostly found in the apical third of the root.[14] De Deus[14] found that 17% of the teeth examined presented lateral canals in the apical third of the root, about 9% in the middle third, and less than 2% in the coronal third. Kirkham,[15] however, reported that only 2% of lateral canals were associated with periodontal pockets.

Accessory canals in the furcation of molars may also be pathways of communication between the pulp and periodontium.[12,16] The reported incidence of furcal accessory canals varies from 23% to 76%.[13,14,16] In vital pulps, these accessory canals contain connective tissue and blood vessels that connect the circulatory system of the pulp with the periodontium. However, not all these canals extend the full length from the pulp chamber to the floor of the furcation.[17] Seltzer et al.[18] suggested that pulpal inflammation or necrosis may cause inflammatory reaction in the inter-radicular periodontal tissues. Patent lateral and accessory canals are a potential pathway for the spread of microorganisms and their toxic by-products, as well as other irritants, from the pulp to the periodontal ligament and vice versa, resulting in an inflammatory process in the involved tissues (Figure 36-3).

Clinically, lateral and accessory canals are very difficult to detect. Pineda and Kuttler[19] reported that about 30% of lateral canals could be identified through use of two-view radiographs, whereas standard radiographs identified only about 8%. Moreover, using this method, the identification of any accessory canals in the furcation area of molar teeth was also possible. Clinically, predictable identification of lateral and accessory canals, on the basis of radiographic interpretation alone, may be achieved in a very small number of cases. Several clinical aids, however, may be helpful for their identification: (1) a radiographic image of a discrete lateral lesion associated with a necrotic pulp; (2) radiographic identification of a "notch" on the lateral root surface suggesting the presence of an orifice; (3) demonstration of root canal filling material, or sealer, extruding through the patent orifices; and (4) advanced imaging techniques.

Apical Foramen

The apical foramen is the main pathway of communication between the pulp and periodontium. Irritants from a diseased pulp may permeate readily through the apical foramen resulting in periapical pathosis. The apical foramen may also be a portal of entry of irritants from deep periodontal pockets

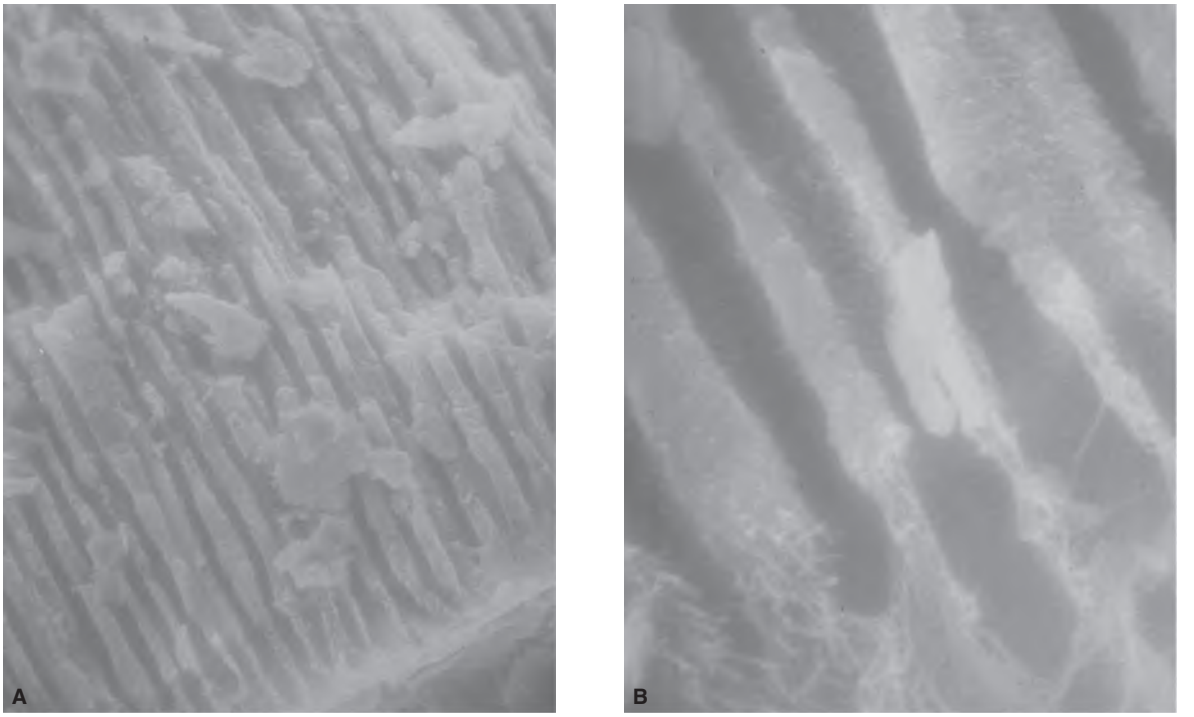

FIGURE 36-1 ***A.*** Scanning electron micrograph of open dentinal tubules. ***B.*** Higher magnification demonstrates absence of odontoblastic processes.

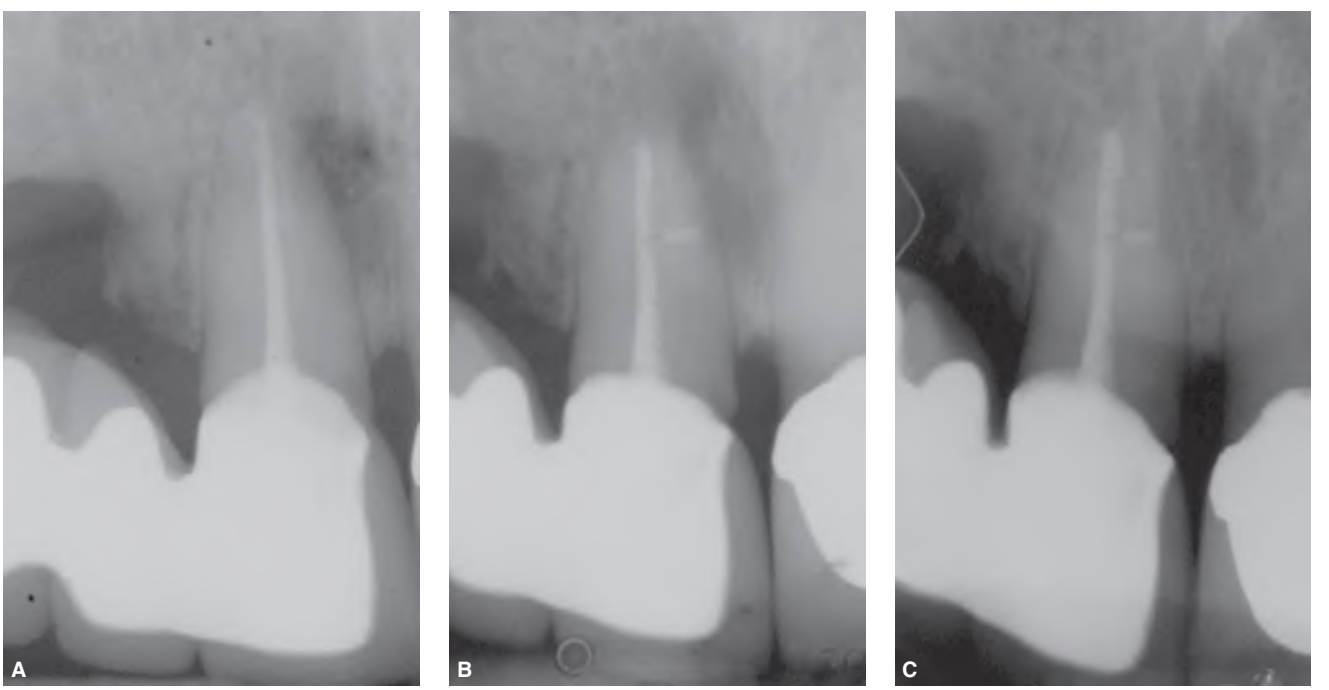

FIGURE 36-2 Non-surgical endodontic treatment of a maxillary central incisor with a lateral radiolucency. ***A.*** Pre-operative radiograph showing previously treated root canal with mesial lateral lesion. ***B.*** Tooth was re-treated and the root canal filled with thermoplasticized gutta-percha and sealer. Note, lateral canal extending toward the lesion. ***C.*** One-year recall shows evidence of active healing.

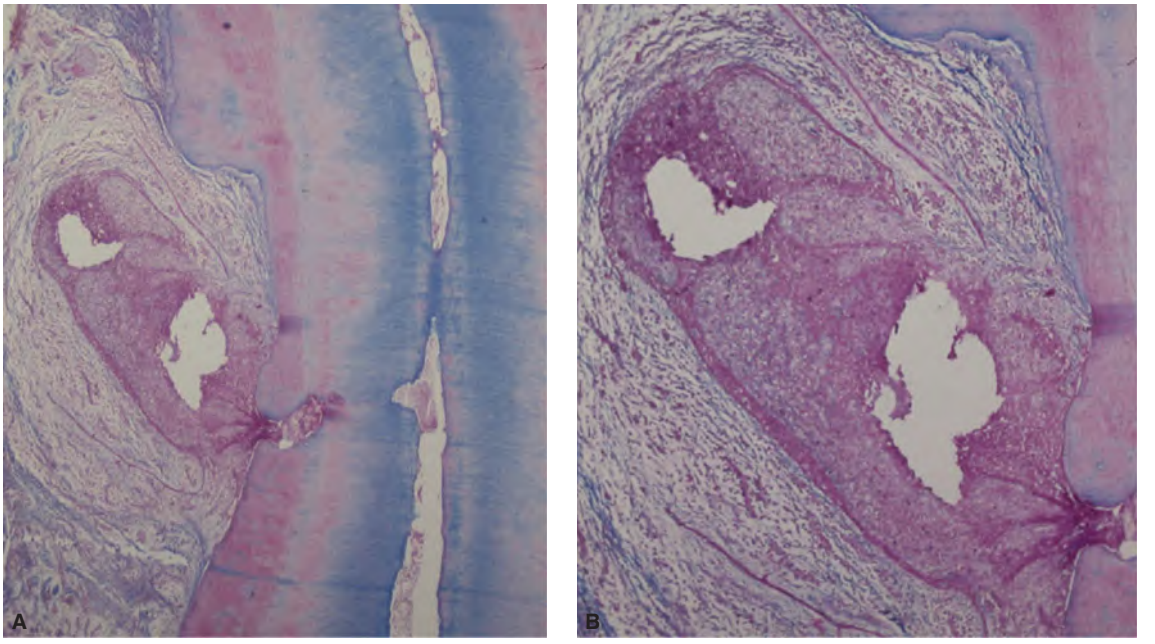

FIGURE 36-3 Micrograph stained with Masson's trichrome of a maxillary lateral incisor with a necrotic pulp associated with a lateral inflammatory process in the periodontal ligament. ***A.*** Main canal, accessory canal, and the resultant inflammatory response in the periodontal ligament are evident. ***B.*** Higher magnification of the area shows chronic inflammation with proliferating epithelium. (Courtesy of Dr. James H.S. Simon, Los Angeles, CA, U.S.A.)

into the pulp. Often, such irritants cause a local inflammatory response associated with bone and root resorption. Elimination of the etiologic irritants from the root canal, as well as from the periodontal tissues, is therefore essential to promote healing.[1,2,20,21]

PULPAL–PERIODONTAL INTERRELATIONSHIP

When the pulp becomes infected, it elicits an inflammatory response of the periodontal ligament at the apical foramen and/or openings of lateral and accessory canals. Inflammatory by-products of pulpal origin may permeate through the apex, lateral and accessory canals, and dentinal tubules to trigger an inflammatory vascular response in the periodontium. Among those products are living pathogens such as certain bacterial strains, fungi, and viruses[22–30] as well as several non-living pathogens.[31–34] In certain cases, pulpal disease may stimulate epithelial growth that will affect the integrity of the periradicular tissues.[35,36]

The result of pulp inflammation can range in extent from minimal inflammation, confined to the periodontal ligament to extensive destruction of the periodontal ligament, tooth socket, and surrounding bone. Such a lesion may result in a localized or diffuse swelling that occasionally involves the gingival attachment. A lesion related to pulpal necrosis may result in a sinus tract that drains through the alveolar mucosa or attached gingiva. Occasionally, it may also drain through the gingival sulcus of the involved tooth or the gingival sulcus of an adjacent tooth. In most instances, after adequate root canal treatment, lesions resulting from pulpal necrosis resolve uneventfully, and the integrity of the periodontal tissues are re-established.[2,20,37]

Certain procedures involved in root canal treatment as well as irrigants, intra-canal medicaments, sealers, and filling materials have the potential to cause an inflammatory response in the periodontium. These inflammatory responses, however, are usually transient in nature and quickly resolved if the materials are confined within the canal space. Periodontal defects, resulting from attachment breakdown, may occur after procedural mishaps, such as perforations of the floor of a pulp chamber or the root surface apical to the gingival attachment, strip perforations, or root perforations from cleaning and shaping procedures. Periodontal defects may also be caused by vertical root fractures associated with excessive force used during canal obturation or restorative procedures.

PERIODONTAL–PULPAL INTERRELATIONSHIP

The effect of periodontal inflammation on the pulp is more controversial.[18,38–52] It appears that, clinically, the pulp is usually not affected by periodontal disease until the defect has exposed a pathway of communication between the root canal and the oral environment.[11] At this stage, pathogens

that pass from the oral cavity through the accessory canal into the pulp may cause a localized inflammatory reaction that could be followed by pulp necrosis. On the other hand, if the microvasculature of the apical foramen remains intact, the pulp may test positive to pulp vitality tests. The effect on the pulp from periodontal treatment is similar during scaling, curettage, or periodontal surgery if accessory canals are severed and/or opened to the oral environment. In such cases, pathogenic invasion and secondary inflammation and necrosis of the pulp may result.[47] It has been suggested that endodontic infection in molars, involved with periodontal disease, might enhance progression of periodontitis by spreading pathogens through accessory canals and dentinal tubules.[50,51]

ETIOLOGICAL FACTORS

Among the living pathogens from a diseased pulp, that can cause lesions in the apical tissues and periodontium, are bacteria (Figure 36-4), fungi (Figure 36-5), and viruses (Figure 36-6). These pathogens and their by-products must be eliminated during endodontic treatment. For more details see Chapter 3.

Bacteria

Bacteria play a critical role in both endodontic and periodontal diseases.[28,53–60] Periapical tissues become involved following bacterial invasion of the pulp, leading to partial or total pulp necrosis. Kakehashi et al.[53] demonstrated the relationship between the presence of microorganisms in the pulp and periapical disease. Pulps of normal rats were exposed and left open to the oral environment. Pulp necrosis ensued followed by periapical inflammation, lesion formation, and furcal inflammation. On the other hand, when the same procedure was performed in germ-free rats, the pulps remained vital and relatively non-inflamed and the exposure sites were repaired by dentin. The study demonstrated that without microorganisms and their products, periapical lesions would not occur from exposed pulps. Möller et al.[55] confirmed these findings in monkeys.

Proteolytic bacteria predominate in the root canal flora, which changes over time to a more anaerobic microbiota.[61,62] Specific profiles of periodontal pathogens in pulpal and periodontal diseases associated with the same tooth were also studied.[63] It appears that periodontal pathogens and endodontic pathogens are similar and that endodontic–periodontal interrelationships are a critical pathway for both diseases.

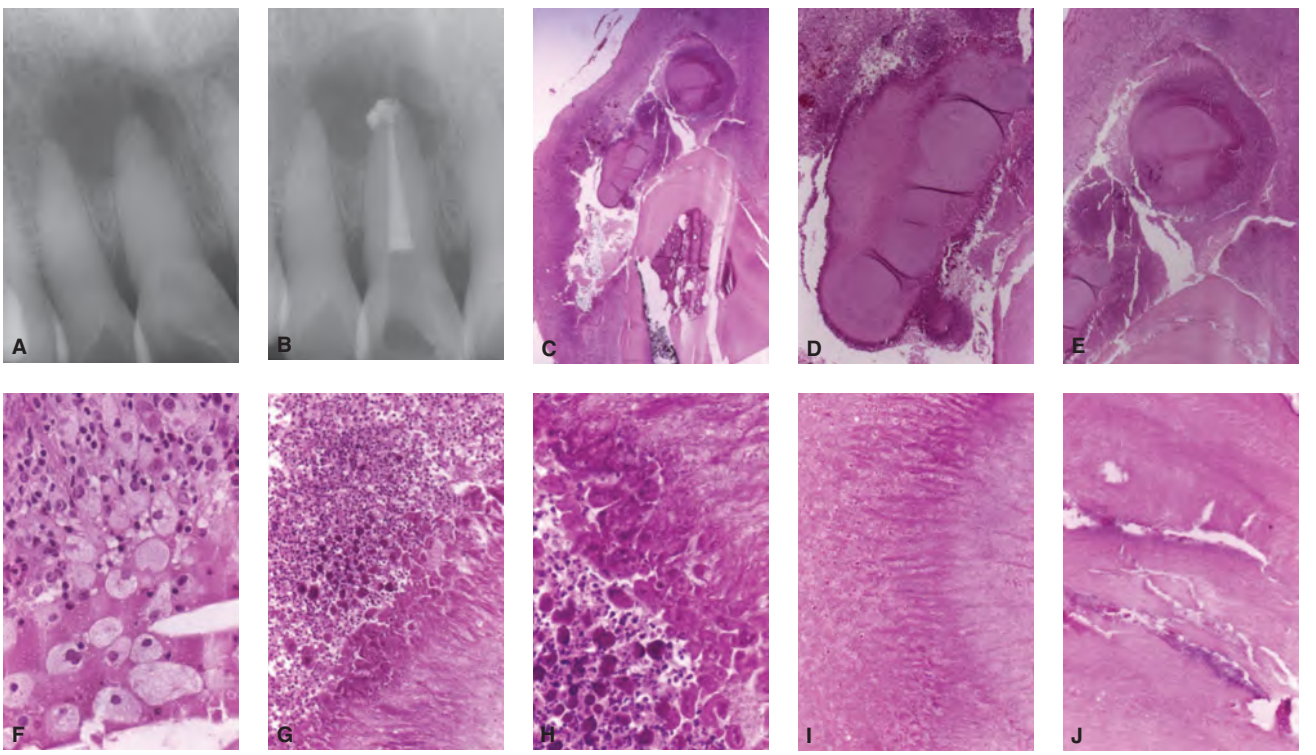

FIGURE 36-4 Periapical *Actinomyces* infection. This case demonstrates the growth of bacteria past the apical foramen and their invasion of apical cementum and periapical tissues. ***A.*** Radiograph of a maxillary central incisor with a necrotic pulp showing a large periapical lesion. ***B.*** Non-surgical endodontic treatment was done but symptoms persisted. ***C.*** Apical surgery was then performed. Photomicrograph shows part of the root with the attached lesion. ***D.*** Colonies of *Actinomyces* in the lumen of the lesion are evident. ***E.*** Higher magnification shows large colony of *Actinomyces*. ***F.*** Foamy macrophages attacking the bacteria. ***G.*** Edge of the bacterial mega-colony showing the absence of inflammatory cells unable to penetrate the colony. ***H.*** Higher magnification of the bacterial colony. ***I***. Center of the colony devoid of inflammatory cells. ***J.*** Viable bacteria within the apical cementum. (Courtesy of Dr. James H.S. Simon, Los Angeles, CA, U.S.A.)

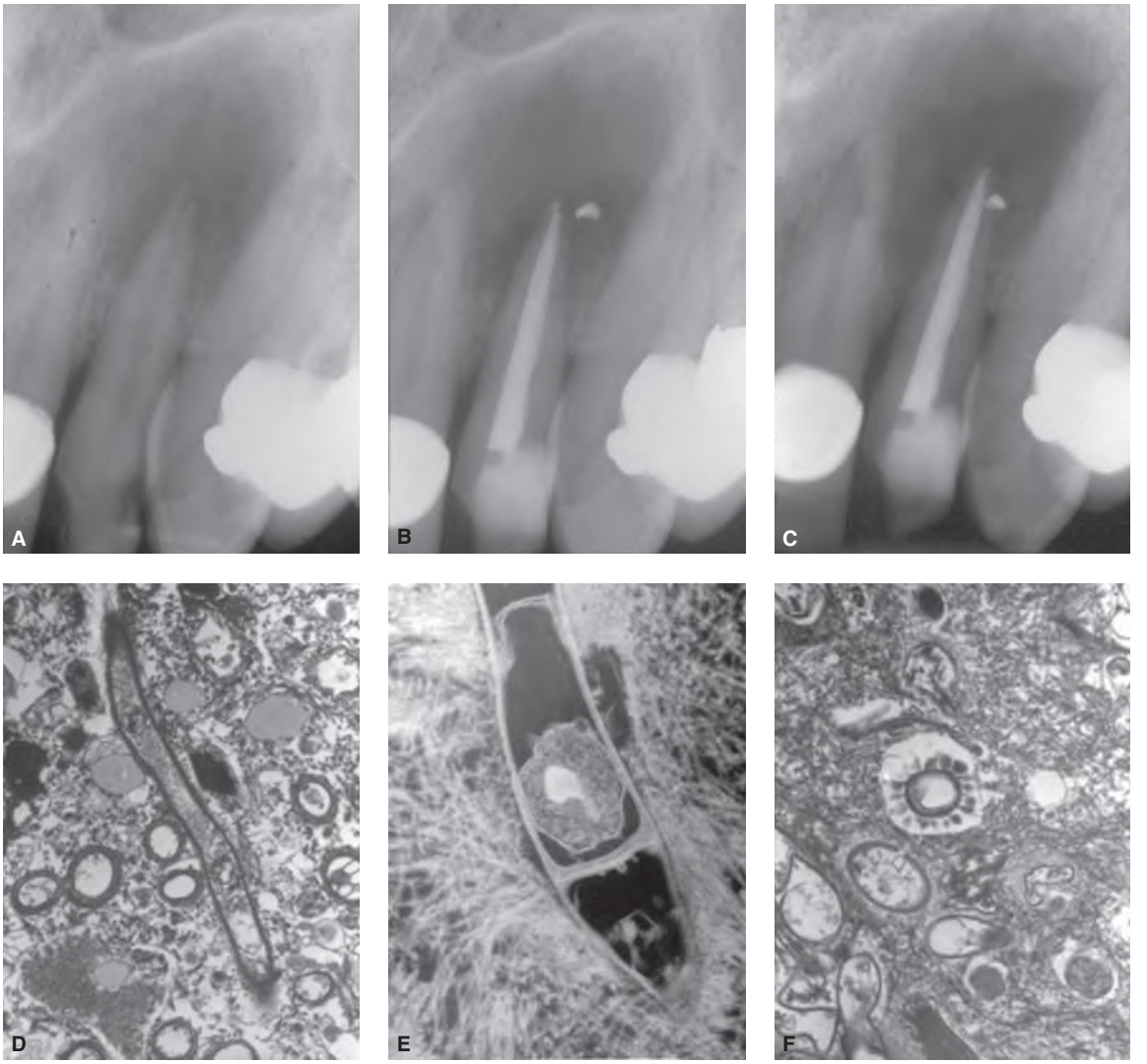

FIGURE 36-5 Fungi in a persistent periapical lesion. ***A.*** Radiograph of maxillary lateral incisor with necrotic pulp and periapical radiolucency. ***B.*** Immediate postoperative radiograph of non-surgical endodontic treatment. ***C.*** At 3-month recall, the patient is still symptomatic and the periapical radiolucency is larger. ***D.*** Transmission electron micrograph shows growing hyphae of a fungus. ***E.*** Higher magnification of the hyphae showing the cell wall. ***F.*** Reproductive fungi spores. (Courtesy of Dr. James H.S. Simon, Los Angeles, CA, U.S.A.)

Spirochetes are associated with both endodontic and periodontal diseases. Spirochetes are found more frequently in subgingival plaque than in root canals. As several studies have shown a large diversity of oral treponemes present in subgingival biofilms of periodontal pockets,[64–66] it has been assumed that the presence or absence of oral spirochetes can be used to differentiate between endodontic and periodontal abscesses.[23] However, today, the presence of spirochetes in the root canal system is well documented and has been demonstrated by different identification techniques.[25,26,67,68] The spirochete species most frequently found in root canals are *Treponema denticola*[69,70] and *Treponema maltophilium*.[71]

L-form bacteria may also have a role in endodontic disease.[72] Some bacteria strains can undergo morphological transition to their L-form after exposure to certain agents, particularly penicillin. L-form bacteria may transform with several intermediate L-form transitional stages. This may occur either spontaneously or by induction in a cyclic manner. Under certain conditions, depending on host resistance factors and bacterial virulence, the L-forms revert to their original pathogenic bacterial form and may then be responsible for acute exacerbation of chronic apical lesions.[72]

Fungi

The presence and prevalence of fungi associated with endodontic infections is well documented.[29,73–83] The majority of these fungi are *Candida albicans*.[82,83] Fungi may also colonize canal walls and invade dentinal tubules. Other species such as *Candida glabrata*, *Candida guillermondii*, *Candida incospicia*, and *Rodotorula mucilaginosa* have also been detected.[82]

Factors affecting the colonization of the root canal by fungi are not fully understood. It has been suggested that the reduction of specific strains of bacteria in the root canal during endodontic treatment may allow fungal overgrowth

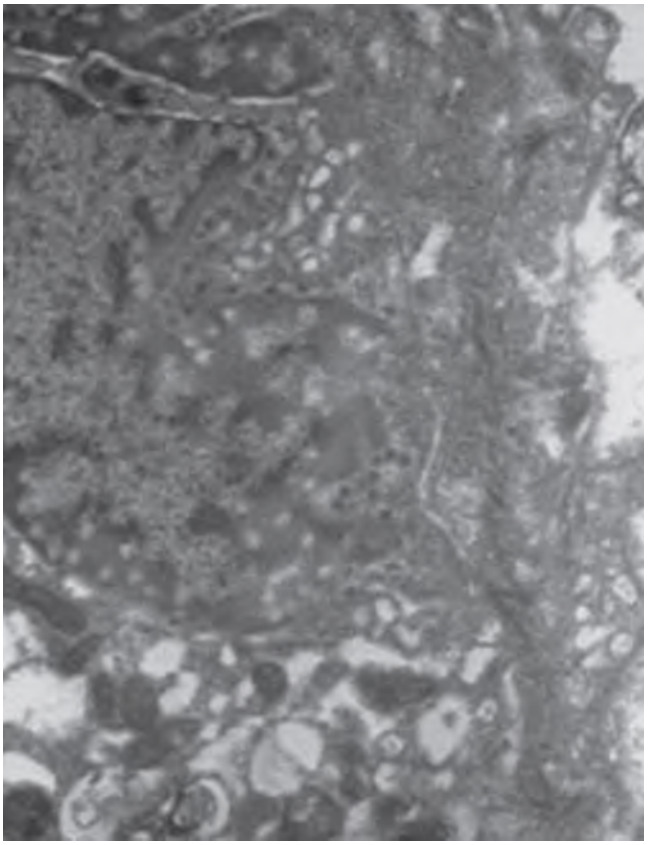

FIGURE 36-6 Transmission electron micrograph of the nucleus of a macrophage in a periapical lesion suggesting a possible viral infection. (Courtesy of Dr. James H.S. Simon, Los Angeles, CA, U.S.A.)

in the remaining low nutrient environment.[78,84] Another possibility is that fungi may gain access to the root canal from the oral cavity as a result of poor asepsis during endodontic treatment or post-preparation procedures. It has been found that approximately 20% of adult periodontitis patients also harbor subgingival fungi.[85,86] As in endodontic infections, *C. albicans* was the most common species isolated.[87]

It has also been demonstrated that the presence of fungi in root canals is directly associated with their presence in saliva.[27] These findings further stress the importance of using aseptic endodontic and periodontal techniques, maintaining the integrity of dental hard tissues, and restoring the tooth crown as soon as practical with a well-sealed permanent restoration in order to prevent re-infection.

Viruses

Viruses may also play a role in the pathogenesis of both endodontic and periodontal diseases. In patients with periodontal disease, herpes simplex virus was frequently detected in gingival crevicular fluid and in gingival biopsies of periodontal lesions.[88] Human cytomegalovirus was observed in about 65% of periodontal pocket samples and in about 85% of gingival tissue samples. Epstein–Barr virus type I was observed in more than 40% of pocket samples and in about 80% of the gingival tissue samples.[88] Gingival herpesviruses were found to be associated with increased occurrence of subgingival *Porphyromonas gingivalis, Bacteroides forsythus, Prevotella intermedia, Prevotella nigrescens, T. denticola*, and *Actinobacillus actinomyce-temcomitans*, thus suggesting a role in overgrowth of periodontal pathogenic bacteria.[89]

Certain viruses may also be involved in pulpal disease and associated periapical pathoses such as human cytomegalovirus and Epstein–Barr virus.[90] It appears that active virus infection may give rise to production of an array of cytokines and chemokines with the potential to induce immunosuppression and tissue destruction.[91] Herpesvirus activation in periapical inflammatory cells may impair the host defense mechanisms and give rise to overgrowth of bacteria, as seen in periodontal lesions. Herpesvirus-mediated immune suppression may also be detrimental in periapical infections due to already compromised host resistant factors and affected connective tissues in situ.[92] Alterations between prolonged periods of herpesvirus latency interrupted by periods of activation may explain some burst-like symptomatic episodes of periapical disease. Absence of herpesvirus infection or viral reactivation may be the reason that some periapical lesions remain clinically stable and asymptomatic for extended periods of time.[90] However, more research is needed to further clarify the relationship between viral infections and pulpal and/or periodontal diseases.

CONTRIBUTING FACTORS

Periapical disease may also be associated with non-microbial factors.[31–34,93–94] For more details see Chapter 3. Clinically, the following factors may play an important roll in the initiation and progression of endodontic disease:

Inadequate Endodontic Treatment

It is essential to clean, shape, and obturate the canal system well in order to enhance successful outcomes.[20,95–97] Poor endodontic treatment often results in treatment failure.[98] Endodontic failures can be treated either by orthograde retreatment or by endodontic surgery with good success rates. In recent years, retreatment techniques have improved dramatically due to use of the operating microscope and development of new armamentarium.

Coronal Leakage

The term "coronal leakage" refers to leakage of microbes and other irritants to and through the root canal filling. Coronal leakage is a major cause of endodontic treatment failure.[99,100] Root canals may also become contaminated by microorganisms due to delay in placement of a coronal restoration and fracture of the coronal

restoration and/or the tooth.[99] Defective restorations and adequate root canal fillings will have a higher incidence of failures than teeth with inadequate root canal fillings and adequate restorations.[98] In an in vitro study, it was found that packing excess gutta-percha and sealer over the floor of the pulp chamber, after completion of root canal filling, did not provide a better seal of the root canals.[100] It is therefore recommended that excess of gutta-percha filling should be removed to the level of the canal orifices and the floor of the pulp chamber be protected with a well-sealed restorative material. An adequate coronal restoration is the primary barrier against coronal leakage and microbial contamination of the root canal treatment.[98] It is essential that the root canal system be protected by good endodontic obturation and a well-sealed coronal restoration.

Traumatic Injuries

Traumatic injuries to teeth may involve the pulp and the surrounding periodontal attachment apparatus. Dental injuries may vary but generally can be classified as enamel fractures, crown fractures without pulp involvement, crown fractures with pulp involvement, crown-root fracture, root fracture, luxation, and avulsion.[101] Treatment and prognosis will depend on the type of injury.[101] Surgical exposure of the remaining tooth structure is one option. For more details see Chapters 12 and 13.

Root Perforations

Root perforations may often cause clinical complications that lead to periodontal lesions. Root perforations may result from extensive carious lesions, resorption, or from operator error during root canal instrumentation or post preparation.[102] 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. It has been recognized that treatment success depends mainly on immediate sealing of the perforation and appropriate infection control. When the root perforation is situated close to the alveolar crest, it may be possible to raise a flap and repair the defect with an appropriate filling material. In deeper perforations, or in a furcation, immediate repair of the perforation has a better prognosis than management of an infected one. Many materials have been used to seal root perforations. Mineral Trioxide Aggregate is widely used to seal root perforations.[102–104] For more details see Chapter 23.

Another treatment modality for perforations, root resorptions, and certain root fractures in the cervical third region is orthodontic root extrusion.[105–108] The procedure has very good prognosis and a low risk of relapse. It can be performed either immediately or over a few weeks period depending on each individual case. The goal of controlled root extrusion is to modify the soft tissues and bone and is therefore used to correct gingival discrepancies and osseous defects of periodontally involved teeth.[106] It is also used in the management of non-restorable teeth and as a non-surgical alternative to crown lengthening.[107]

Developmental Malformations

Radicular invaginations or radicular grooves can lead to an untreatable periodontal condition[108] (Figure 36-7). These grooves usually begin in the central fossa of maxillary central and lateral incisors crossing over the cingulum and continuing apically down the root for varying distances. Such a groove is probably the result of an attempt of the tooth germ to form another root. As long as the epithelial attachment remains intact, the periodontium remains healthy. However, once this attachment is breached and the groove becomes contaminated, a self-sustaining infrabony pocket can be formed along its entire length. This fissure-like defect provides a nidus for accumulation of microorganisms and an avenue for the progression of periodontal

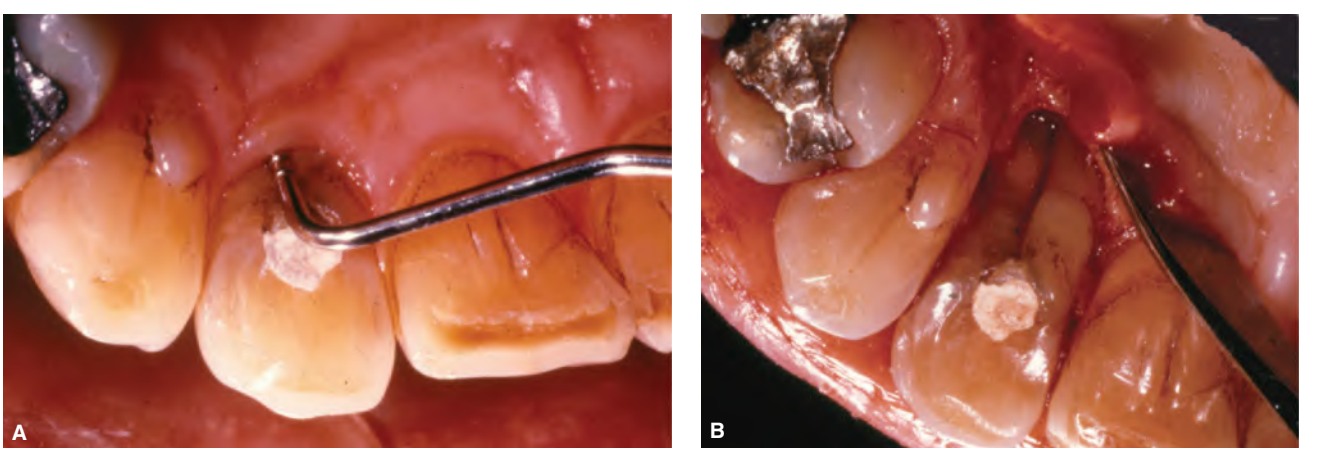

FIGURE 36-7 ***A.*** Probing deep lingual pocket associated with radicular invagination. ***B.*** Raised flap reveals the depth and length of deep lingual groove.

disease that can also affect the pulp. Radiographically, the area of bone destruction follows the course of the groove.

Clinically, the patient may present symptoms of a periodontal abscess or a variety of asymptomatic endodontic conditions. If the condition is purely periodontal, it can be diagnosed by visually following the groove to the gingival margin and by probing the depth of the pocket, which is usually tubular in form and localized to this one area, as opposed to a more generalized periodontal problem. The tooth will also respond to pulp testing procedures. If this condition is also associated with an endodontic disease, the patient may present clinically with any of the spectrum of endodontic symptoms. While performing clinical examination, the clinician must look for the groove as it may have been altered by a previous access opening or restoration placed in the access cavity. The appearance of a teardrop-shaped area on the radiograph should immediately arouse suspicion. The developmental groove may be visible on the radiograph as a dark vertical line. This condition must be differentiated from a vertical fracture, which may give a similar radiographic appearance.

The prognosis of root canal treatment in such cases is guarded, depending on the apical extent of the groove. Radicular grooves are self-sustaining infra-bony pockets and therefore scaling and root planing will not suffice. Although the acute nature of the problem may be alleviated initially, the source of the chronic or acute inflammation must be eradicated by a surgical approach. Treatment consists of burring out the groove, placing bone substitutes, and surgical management of the soft tissues and underlying bone.[109–111] If unsuccessful, the tooth needs to be extracted due to poor prognosis.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis for treatment and prognosis of endodontic-periodontal diseases can be achieved by using the following classification: (1) primary endodontic diseases, (2) primary periodontal diseases, and (3) combined diseases.[1,2] The combined diseases include (1) primary endodontic disease with secondary periodontal involvement, (2) primary periodontal disease with secondary endodontic involvement, and (3) true combined diseases. This classification is based on how these lesions are formed. By understanding the pathogenesis, the clinician can then offer an appropriate course of treatment and better assess the prognosis.

Primary Endodontic Disease

A chronic apical abscess may drain coronally through the periodontal ligament into the gingival sulcus. This condition may clinically mimic the presence of a periodontal abscess draining through a pseudo-pocket. In reality, the pocket is a sinus tract from pulpal origin that opens through the periodontal ligament area. A deep solitary pocket in the absence of periodontal disease may indicate the presence of a lesion of endodontic origin. For diagnostic purposes, a gutta-percha cone, or another tracking instrument, should be inserted into the sinus tract and radiographs taken. This will determine the origin of the lesion.

A sulcular pocket of endodontic origin is typically very narrow compared to a pocket of periodontal origin. A similar condition occurs where drainage from the apex of a molar extends coronally into the furcation area. This may also occur in the presence of accessory canals extending from a necrotic pulp into the furcation area. A vertical root fracture or a radicular crack should always be ruled out as they may give initially similar periodontal clinical findings. Primary endodontic lesions usually heal following root canal treatment. The sinus tract extending into the gingival sulcus or furcation area quickly heals once the affected pulp has been removed and the root canals cleaned, shaped, and obturated (Figures 36-8 to 36-10).

Primary Periodontal Disease

These types of conditions are caused primarily by periodontal pathogens. In this process, chronic marginal periodontitis progresses apically along the root surface. In most cases, pulp tests indicate a clinically normal pulpal reaction (Figures 36-11 & 36-12). There is frequently an accumulation of plaque and calculus and the pockets are wider. The prognosis depends upon the stage of periodontal disease and the efficacy of periodontal treatment. The clinician must also be aware of the radiographic appearance of periodontal disease associated with developmental radicular anomalies (Figure 36-13).

Combined Diseaes

Primary Endodontic Disease with Secondary Priodontal Involvement

Untreated suppurating primary endodontic disease may sometimes become secondarily involved with marginal periodontal breakdown. In such cases, marginal periodontitis is developed as a result of plaque formation at the gingival margin of the sinus tract. When plaque or calculus is present, the treatment and prognosis of the tooth are different than those of teeth involved with only primary endodontic disease. The tooth now requires both endodontic and periodontal treatments. If the endodontic treatment is adequate, the prognosis depends on the severity of the marginal periodontal damage and the efficacy of periodontal treatment. With endodontic treatment alone, only part of the lesion will heal to the level of the secondary periodontal lesion (Figures 36-14).

A similar clinical picture may also occur as a result of root perforation during root canal treatment, or where pins or posts have been misplaced during coronal restoration. Sometimes, symptoms may be acute, with periodontal abscess formation associated with pain, swelling, purulent exudate, and pocket formation and tooth mobility. A more chronic response may also occur without pain and involves the sudden appearance of a pocket with bleeding on probing or exudation of pus.

Root fractures may also mimic the appearance of primary endodontic lesions with secondary periodontal involvement

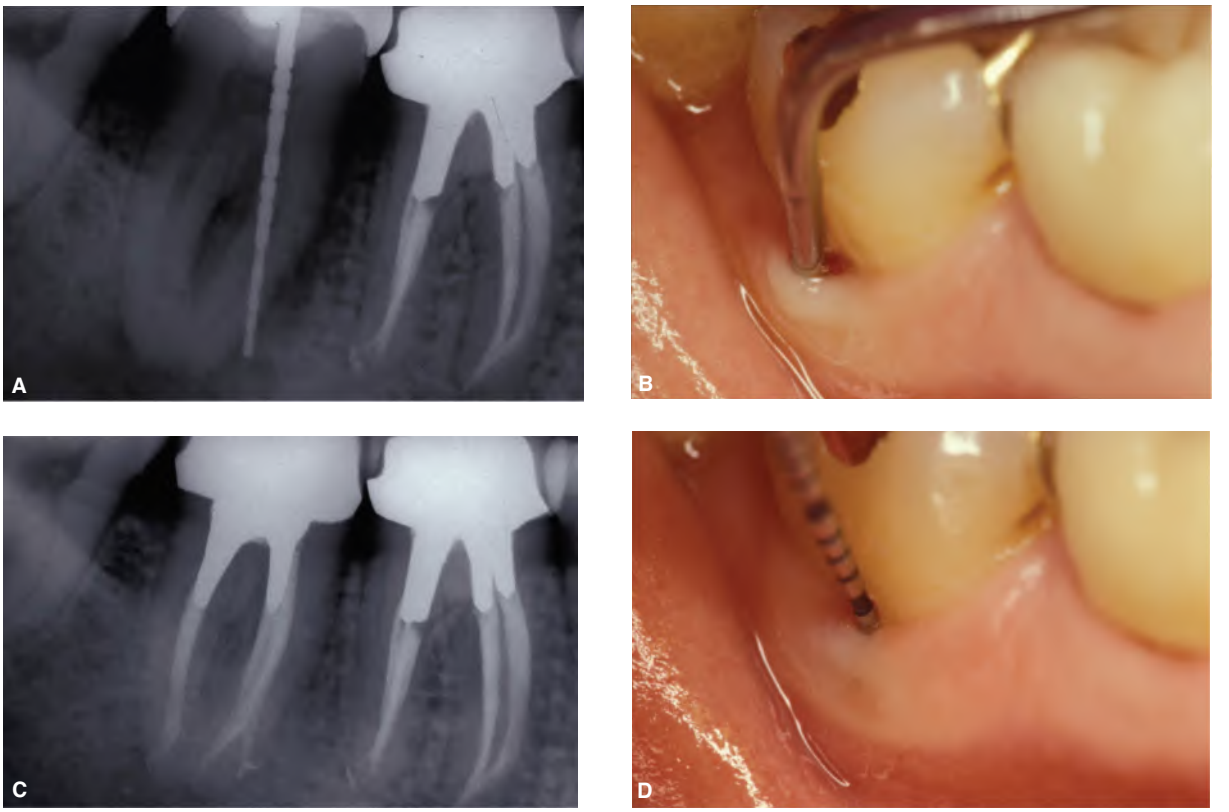

FIGURE 36-8 Primary endodontic disease in a mandibular second molar with necrotic pulp. ***A.*** Preoperative radiograph showing periradicular radiolucency associated with the affected tooth. ***B.*** Clinically, a deep narrow buccal periodontal defect can be probed. ***C.*** One year after root canal treatment, resolution of the periradicular bony radiolucency is evident. ***D.*** Clinically, the buccal defect healed and probing is normal. (Courtesy of Dr. Jean-Yves Cochet, Paris, France.)

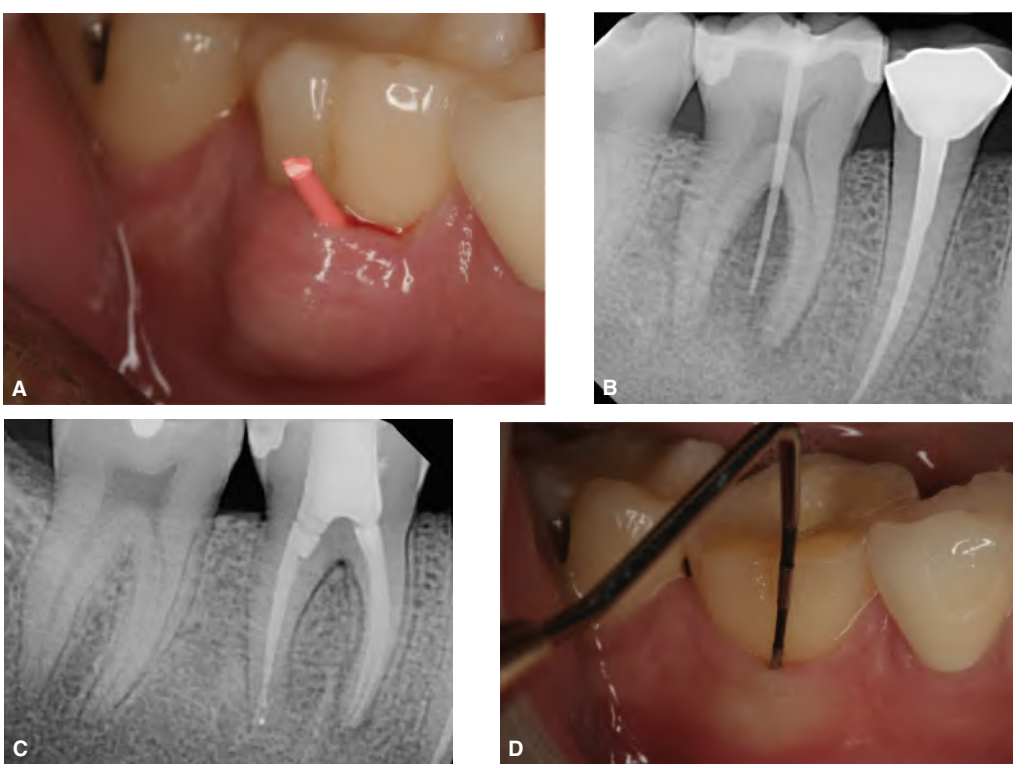

FIGURE 36-9 Primary endodontic disease in a mandibular first molar with a necrotic pulp. ***A.*** Abscess on the buccal aspect of the tooth is traced with a gutta-percha cone. ***B.*** Periapical radiograph showing radiolucency associated with the mesial and distal roots as well as the furcation area. ***C.*** Radiograph taken 4 months after root canal therapy showing active bony healing. ***D.*** Clinically, the buccal periodontal defect healed and probing is normal. (Courtesy of Dr. Ziv Simon, Beverly Hills, CA, U.S.A.)

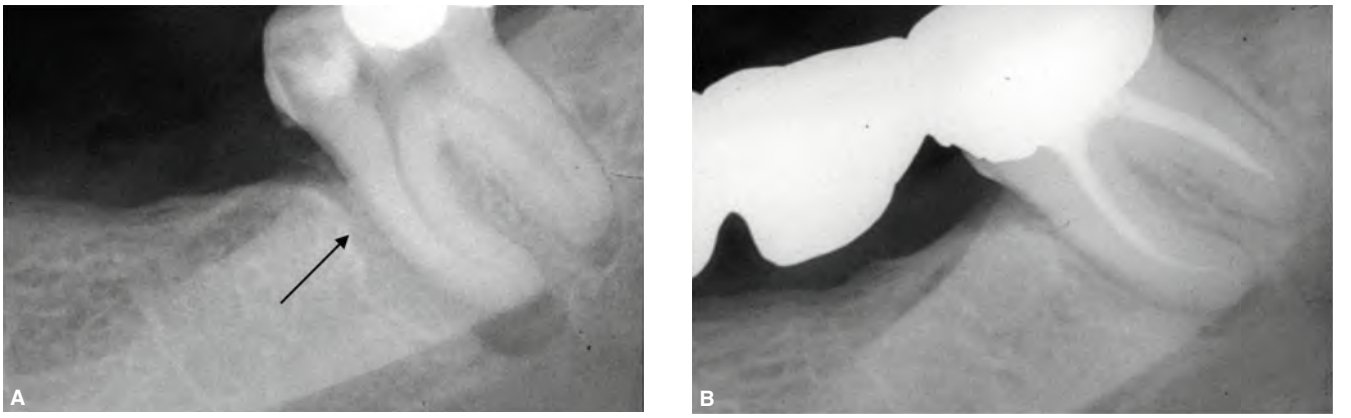

FIGURE 36-10 Primary endodontic disease in a mandibular second molar of a 45-year-old male. ***A.*** Preoperative radiograph showing evidence of coronal caries and periradicular radiolucencies associated with the affected tooth. Note, localized, narrow and elongated radiolucency alongside the mesial root (arrow). The tooth was symptomatic and pulp sensibility tests were negative. Endodontic treatment was done. ***B.*** Two-year follow-up radiograph showing resolution of the periradicular radiolucencies. The patient was comfortable and the tooth has been serving as a distal abutment for a prosthetic reconstruction. (Courtesy of Dr. Borja Zabalegui, Bilbao, Spain.)

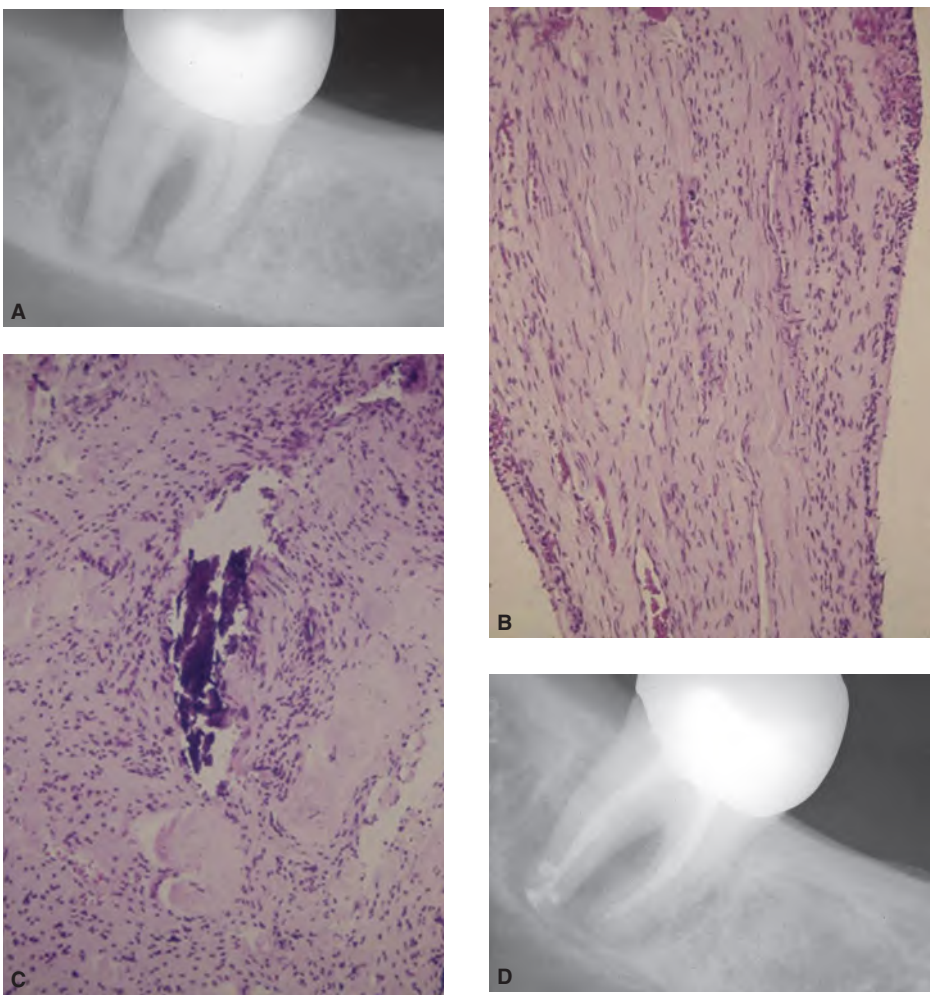

FIGURE 36-11 Primary periodontal disease in a mandibular second molar simulating endodontic lesion. Patient was referred for endodontic treatment. ***A.*** Preoperative radiograph showing periradicular radiolucency; however, the tooth responded to pulp sensibility tests. The referring dentist insisted that endodontic treatment should be done. ***B.*** Photomicrograph of the pulp tissue removed during treatment. Note normal appearance of the pulp. ***C.*** Higher magnification shows normal cellular components as well as blood microvasculature. ***D.*** Post-operative radiograph. The tooth was subsequently lost due to the periodontal disease. (Courtesy of Dr. James H.S. Simon, Los Angeles, CA, U.S.A.)

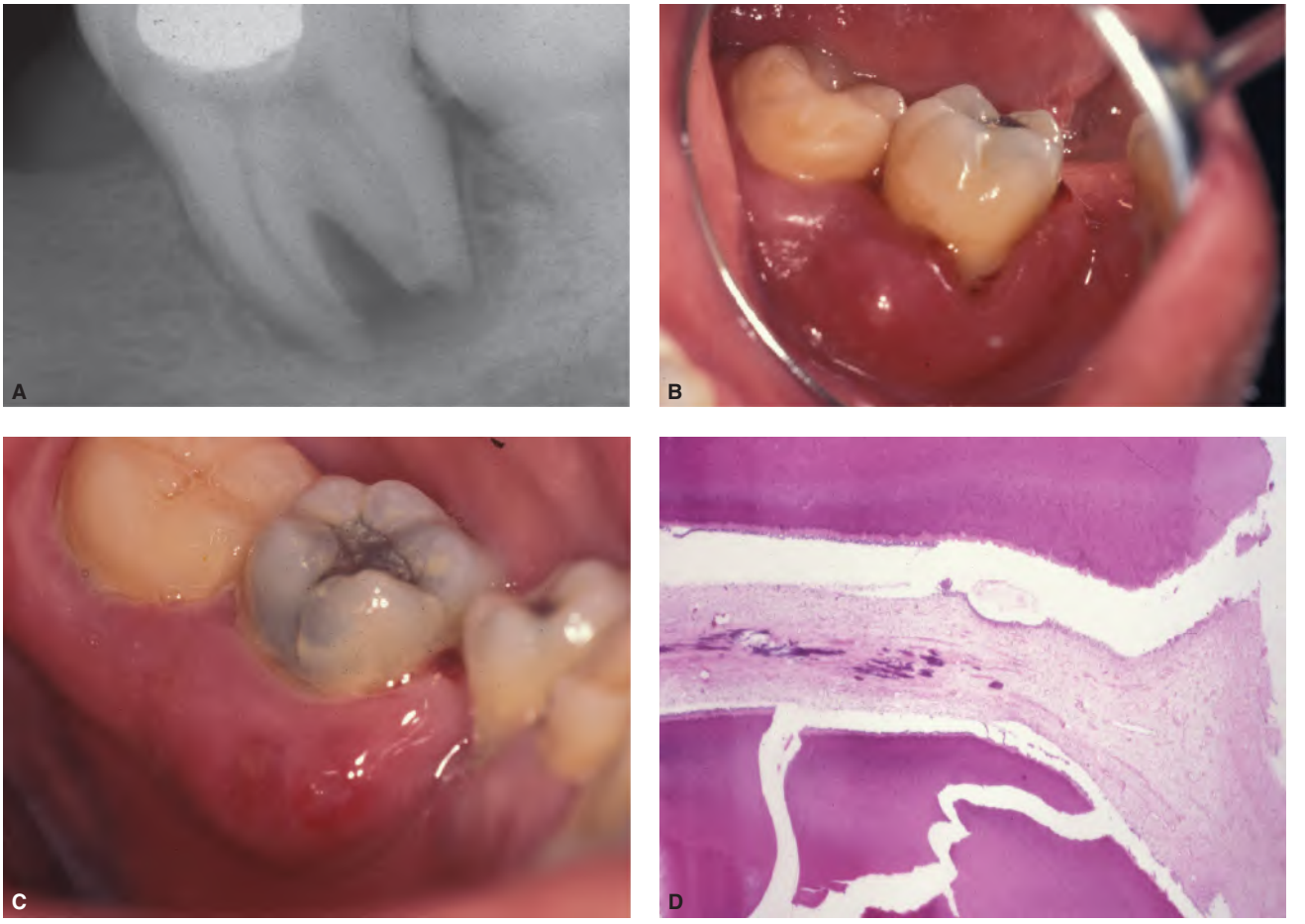

FIGURE 36-12 Primary periodontal disease in a mandibular first molar simulating endodontic lesion. ***A.*** Radiograph showing periradicular radiolucency and periapical resorption associated with the affected tooth. ***B, C.*** Buccal and lingual views. Note, gingival swelling and evidence of periodontal disease. In addition, an occulsal filling is present close to the pulp chamber. In spite of the clinical and radiographic picture, the pulp responded normal to sensibility tests indicating the radiolucency, resorption and gingival swelling are of periodontal origin. ***D.*** Microphotograph stained with H&E showing floor of the pulp chamber and entrance to the mesial canal containing normal pulp tissue (original magnification × 40).

(Figure 36-15). These may occur following traumatic injury, damaging chewing habits, excessive occlusal forces or in endodontically treated teeth restored with large post (Figure 36-16). In such cases, a local deepening of a periodontal pocket and more acute periodontal abscess symptoms can be found.

Primary Periodontal Disease with Secondary Endodontic Involvement

A periodontal pocket may continue and progress until the apical tissues are involved. In this case, the pulp may become infected due to irritants entering via lateral canals or the apical foramen and subsequently become necrotic. (Figures 36-17 to 36-19).

In single-rooted teeth, the prognosis is usually poor. In molar teeth, the prognosis may be better because not all the roots may suffer the same loss of supporting tissues. In some of these cases, root resection can be considered as a treatment alternative.

Microorganisms originating from the periodontal pocket can be a source of root canal infection. A strong correlation between the presence of microorganisms in root canals and their presence in periodontal pockets of advanced periodontitis has been demonstrated indicating that similar pathogens may be involved in both diseases.[112,113] As long as the neurovascular supply of the pulp remains intact, prospects for survival are good. If the neurovascular supply is lost to periodontal disease, pulpal necrosis will ensue.[45]

Treatment complications of periodontal disease can also lead to secondary endodontic involvement. Lateral canals and dentinal tubules may be opened to the oral environment by curettage, scaling, or surgical flap procedures. In such cases, blood vessels within a lateral canal can be severed by a curette and microorganisms introduced into the area during treatment. This may often result in pulp inflammation and necrosis.

TRUE COMBINED DISEASES

True combined diseases occur less often. They are usually formed when an endodontic disease progressing coronally joins with an infected periodontal pocket progressing

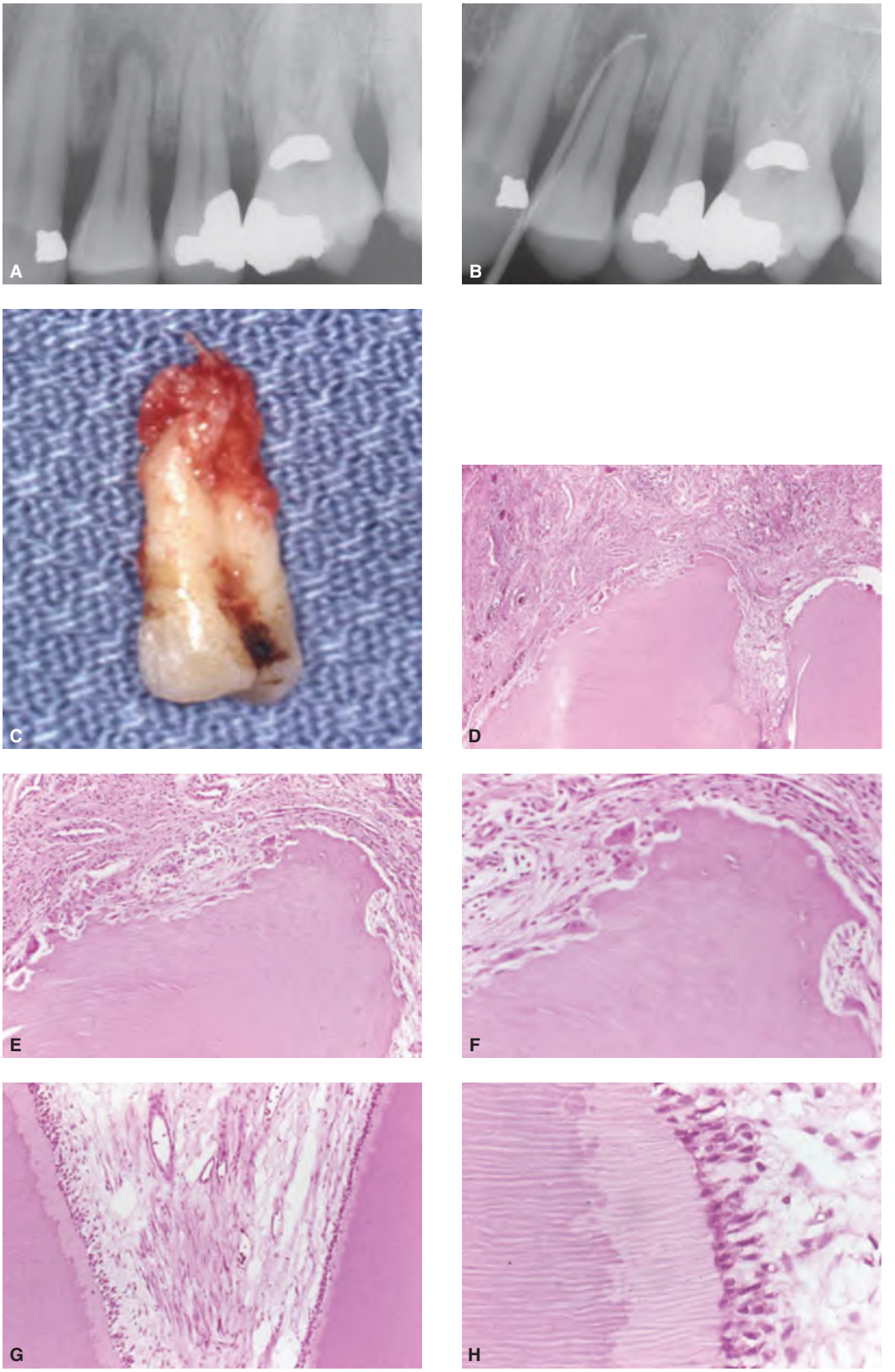

FIGURE 36-13 Primary periodontal disease in a maxillary first premolar. ***A.*** Radiograph showing alveolar bone loss and a periapical lesion. Clinically, a deep narrow pocket was found on the mesial aspect of the root. There was no evidence of caries, and the tooth responded normally to pulp sensibility tests. ***B.*** Radiograph showing pocket tracking with gutta-percha cone to the apical area. It was decided to extract the tooth. ***C.*** Clinical view of the extracted tooth with the attached lesion. Note a deep mesial radicular development groove. ***D.*** Photomicrograph of the apex of the tooth with the attached lesion. ***E, F.*** Higher magnification shows the inflammatory lesion, cementum and dentin resorption, and osteoclasts. ***G, H.*** Histologic sections of the pulp chamber shows uninflammed pulp, odontoblastic layer, and intact predentin. (Courtesy of Dr. James H.S. Simon, Los Angeles, CA, U.S.A.)

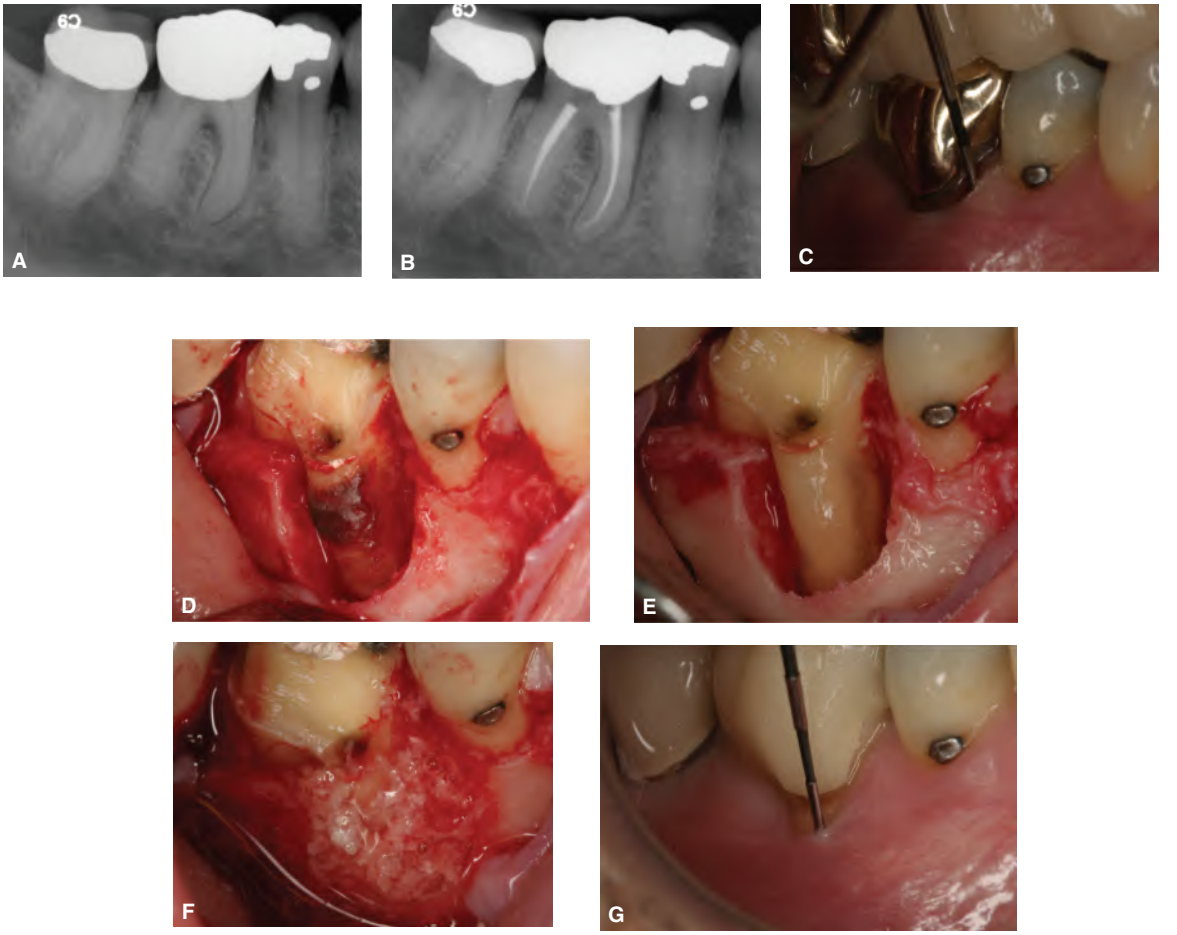

FIGURE 36-14 Primary endodontic disease with secondary periodontal involvement. ***A.*** Pre-operative radiograph of a symptomatic lower right first molar. Pulp sensibility tests were negative. Initial diagnosis of periapical periodontitis caused by a necrotic pulp was established. ***B.*** Endodontic treatment was done, however, residual deep pocket (7-9 mm) on the mesial root remained. ***C.*** Examination reveals a large mucogingival defect with minimal keratinization and absence of attached gingiva. ***D.*** Upon exploration, severe bone loss and abundant calculus on the mesial root were observed. ***E.*** The root surface was then debrided and the bony defect cleaned. ***F.*** Bony defect is repaired with bone graft material and a resorbable membrane. ***G.*** Two-month follow-up shows active healing of the defect. (Courtesy of Dr. Ziv Simon, Beverly Hills, CA, U.S.A.)

apically.[1,2] The degree of attachment loss in this type of lesions is large and the prognosis guarded in single-rooted teeth (Figures 36-20 to 36-22). In most cases, periapical healing may be anticipated following successful endodontic treatment. However, the periodontal tissues may not always respond well to treatment, and healing will depend on the severity of the condition and the efficacy of periodontal therapy. In such cases, regeneration procedures may prove beneficial.[114,115]

The radiographic appearance of combined endodontic-periodontal disease may be similar to that of a vertically fractured tooth. A fracture that has invaded the pulp space causing pulp necrosis may also be considered a true combined lesion and yet not be amenable to successful treatment. Often, it is necessary to perform surgical exploration of the affected site to confirm the diagnosis.

PROGNOSIS

Correct diagnosis of the etiology of the disease process, whether endodontic, periodontal, or combined, will determine the treatment and long-term prognosis. The main factors to consider are pulp vitality and type and extent of the periodontal condition. For example, the prognosis for a tooth with a necrotic pulp, with or without a sinus track, is excellent following adequate root canal treatment. However, the prognosis of root canal treatment in a tooth with severe periodontal disease is dependent on the success of the periodontal treatment.

Primary endodontic disease need only be treated by endodontic therapy. Good prognosis is to be expected if appropriate endodontic treatment is carried out. Primary periodontal disease should only be treated by periodontal treatment. In this case, the prognosis depends on severity of the periodontal disease and the patient's tissues response.

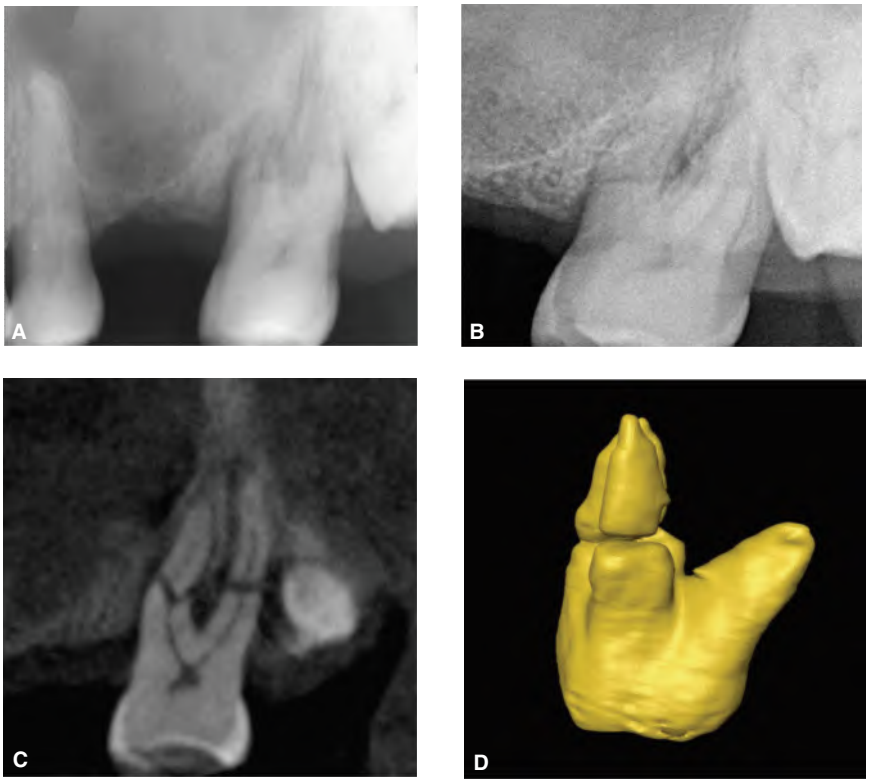

FIGURE 36-15 Primary endodontic disease with secondary periodontal involvement in a maxillary left second molar of a 48-year-old male. ***A.*** Preoperative radiograph showing periradicular radiolucency associated mainly with the mesiobuccal root. The tooth was sensitive to percussion and pulp sensibility tests were negative. The patient didn't recall any significant traumatic injury to the area. ***B.*** Additional preoperative radiograph, from a different angulation, shows possible horizontal fracture in the mesial root (arrow). ***C.*** Cone Beam Computerized Tomography (100 voxel; sagital view) indicates presence of horizontal root fractures in both buccal roots. ***D.*** Segmentation analysis (Amira Visage software) demonstrates root fractures in both buccal roots while the palatal root appears intact. (Courtesy of Dr. Borja Zabalequi, Bilbao, Spain.)

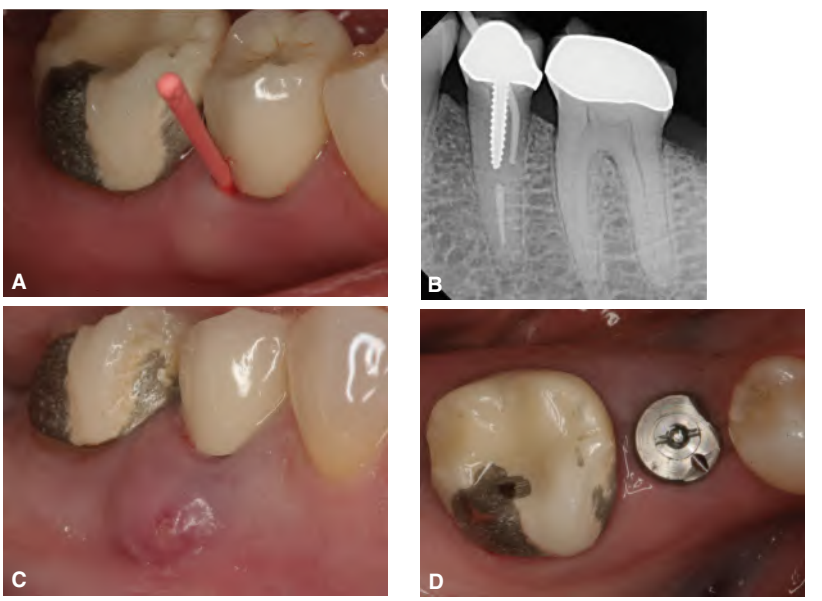

FIGURE 36-16 Complications of certain defects along the root may have similar clinical appearance of primary endodontic disease with secondary periodontal involvement. ***A.*** Patient presented with a localized deep pocket (traced with a gutta-percha cone) associated with an endodontically treated mandibular second premolar. ***B.*** Periapical radiograph showing the gutta-percha cone pointing to an area of the root associated with a screw post. ***C.*** Subsequent abscess formation on the buccal aspect of the tooth caused loss of the buccal plate. ***D.*** Surgical exploration confirmed the presence of a vertical root fracture and the tooth was extracted and replaced by an implant. (Courtesy of Dr. Ziv Simon, Beverly Hills, CA, U.S.A.)

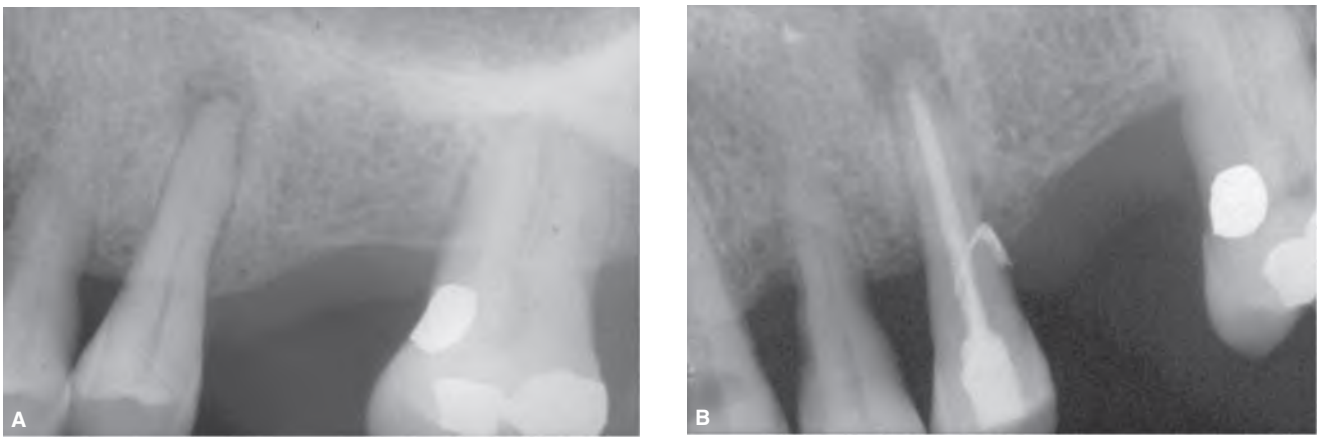

FIGURE 36-17 Primary periodontal disease with secondary endodontic involvement in a maxillary premolar. ***A.*** Radiograph showing bone loss in one-third of the root and separate periapical radiolucency. The crown was intact, but pulp sensibility tests were negative. ***B.*** Radiograph taken immediately after root canal treatment showing sealer in lateral canal that was exposed due to bone loss.

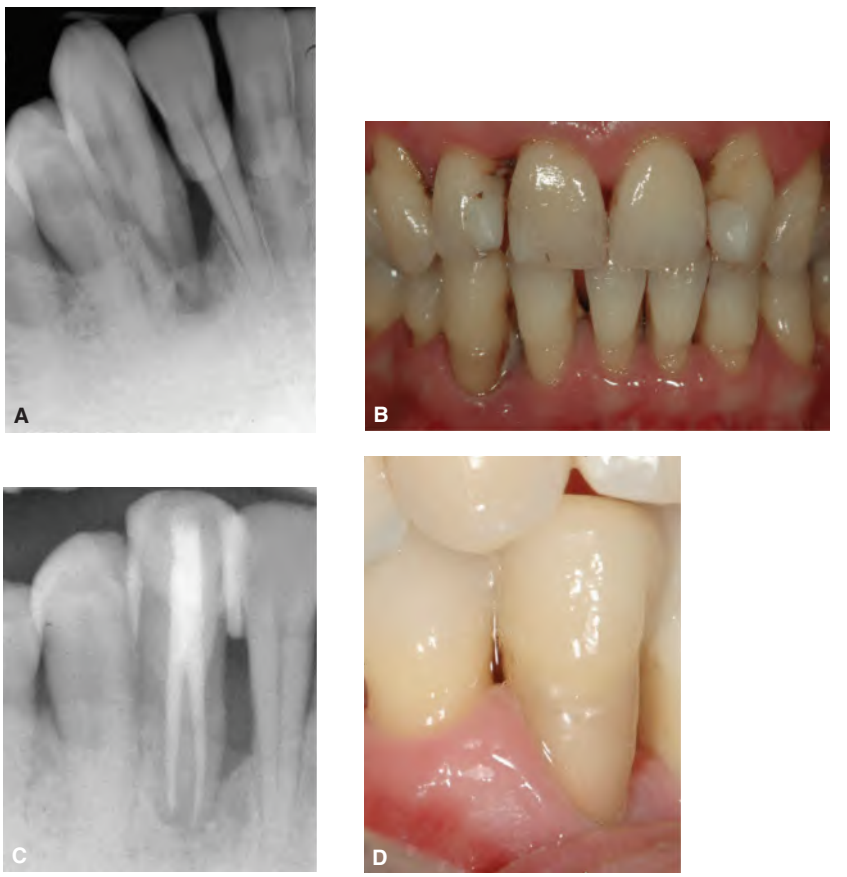

FIGURE 36-18 Primary periodontal disease with secondary endodontic involvement in a mandibular canine of a 35-year-old male. ***A.*** Preoperative radiograph showing extensive periradicular radiolucency associated with the affected tooth. The tooth was mobile and pulp sensibility tests were negative. ***B.*** Clinical view showing purulent discharge from a mesial periodontal defect associated with the canine. Endodontic treatment was done followed by periodontal therapy. ***C.*** Seven-year follow-up radiograph. The radiolucency associated with the endodontic component has resolved. The tooth was not mobile and the patient was comfortable. However, a controlled periodontal defect still remains due to the primary periodontal nature of the disease. ***D.*** Clinical view showing resolution of the purulent discharge. (Courtesy of Dr. Borja Zabalequi, Bilbao, Spain.)

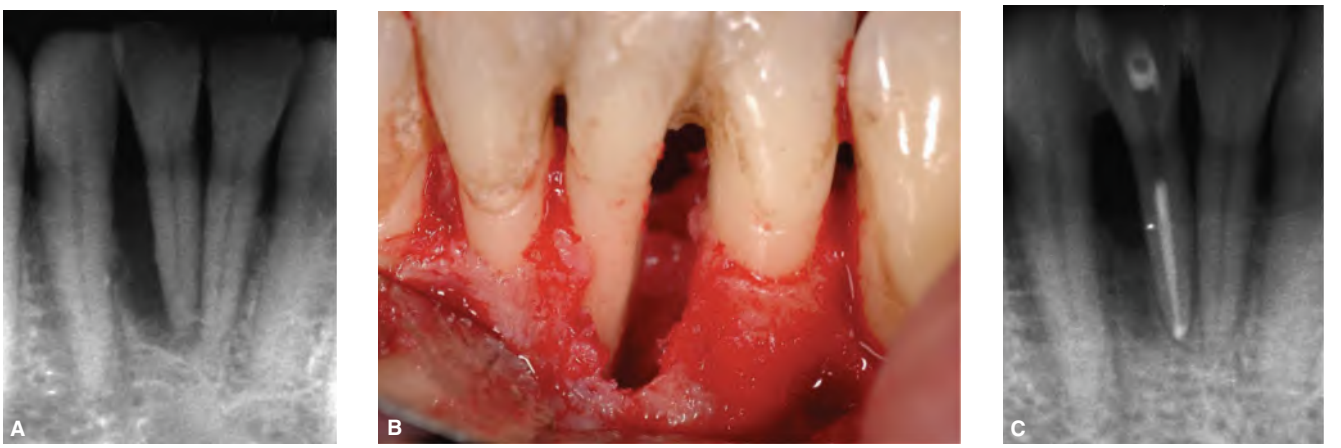

FIGURE 36-19 Primary periodontal disease with secondary endodontic involvement in a mandibular lateral incisor of a 47-year-old male. ***A.*** Preoperative radiograph showing extensive periradicular radiolucency associated with the affected tooth. The tooth was mobile and pulp sensibility tests were negative. ***B.*** Clinical exploration revealed an extensive distal bony defect. Endodontic treatment was done that was later followed by regenerative periodontal therapy. ***C.*** 18-month follow-up radiograph. The size of the bony radiolucency has been reduced significantly. The tooth was not mobile and the patient was comfortable. (Courtesy of Dr. Borja Zabalequi, Bilbao, Spain.)

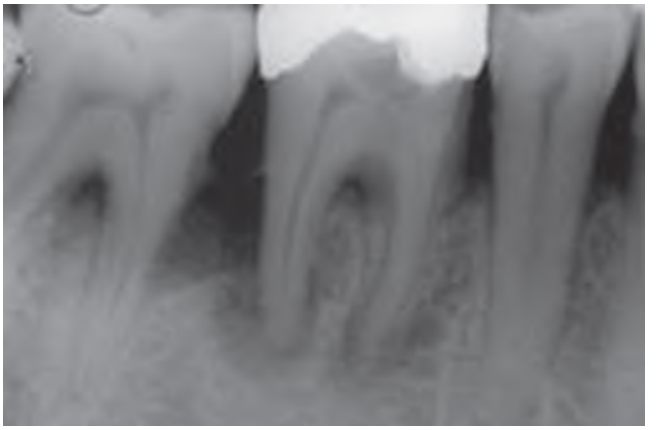

FIGURE 36-20 True combined endodontic–periodontal disease in a mandibular first molar. Radiograph showing separate progression of endodontic disease and periodontal disease. The tooth remained untreated and consequently the two lesions joined together.

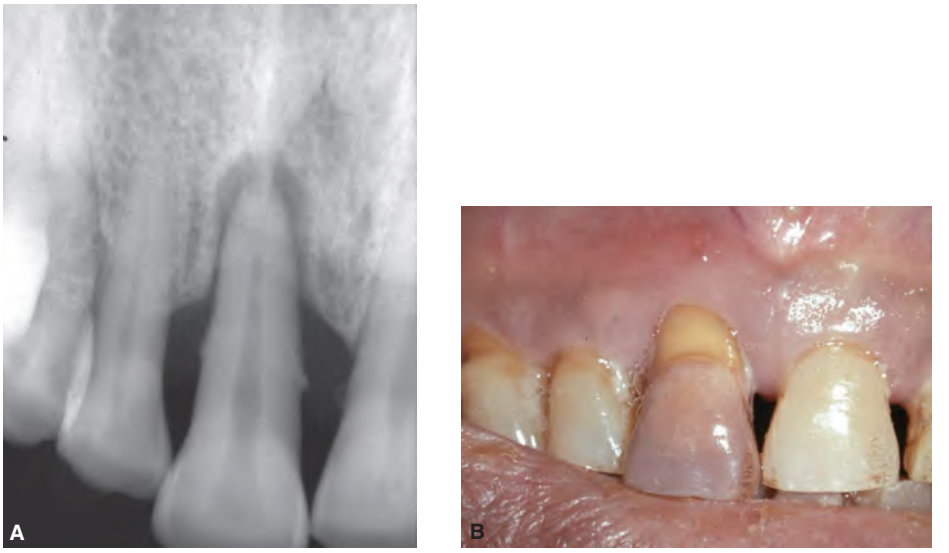

FIGURE 36-21 True combined endodontic–periodontal disease in a maxillary central incisor. ***A.*** Radiograph showing bone loss in two-thirds of the root with calculus present and separate periapical radiolucency. ***B.*** Clinical examination revealed coronal color change of the tooth involved and pus draining from the gingival crevis. Pulp sensibility tests were negative.

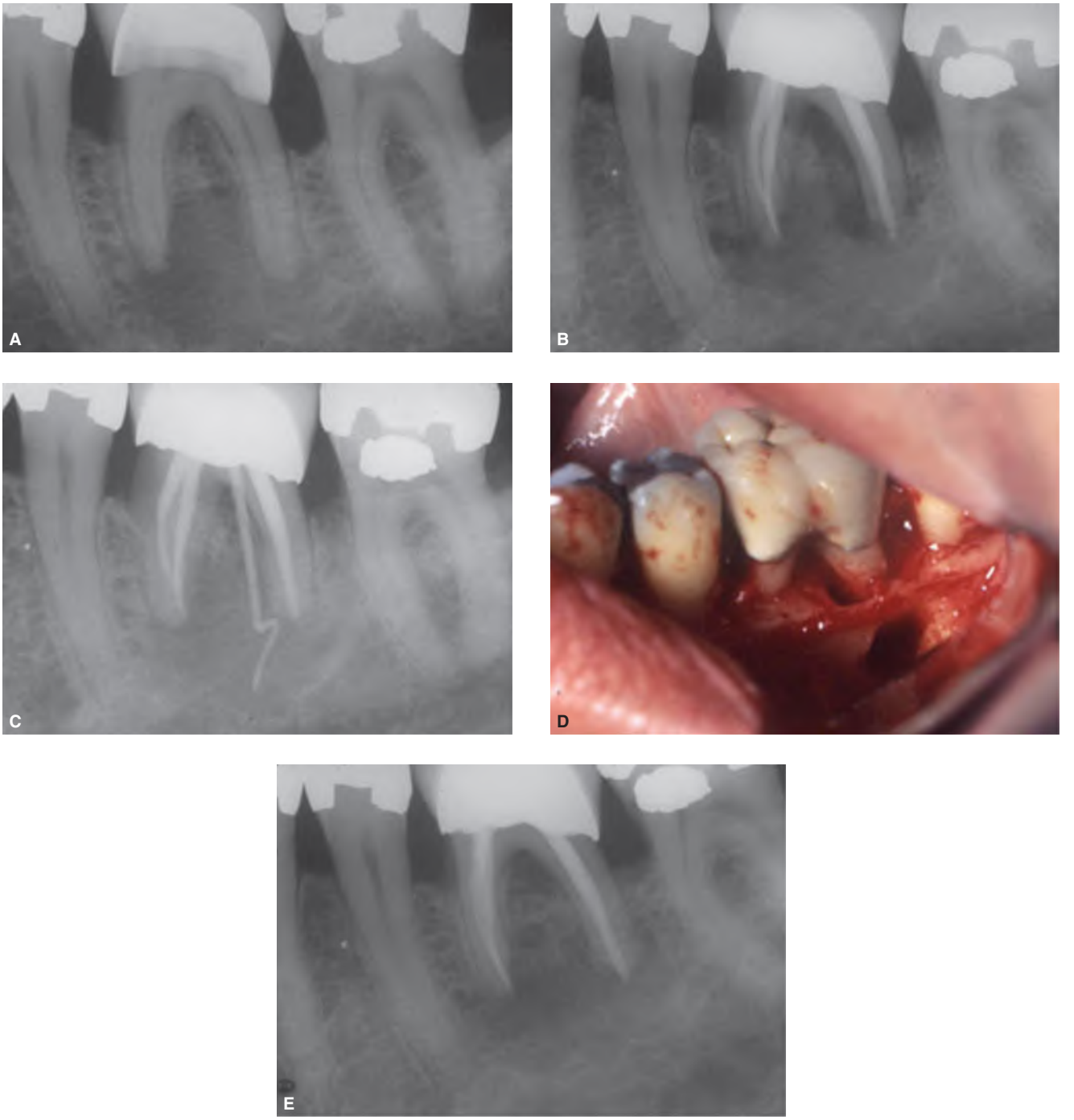

FIGURE 36-22 True combined endodontic–periodontal diseases in a mandibular first molar. ***A.*** Preoperative radiograph showing periradicular radiolucencies. Pulp sensibility tests were negative. ***B.*** Immediate post-operative radiograph of non-surgical endodontic treatment. ***C.*** Six-month follow-up radiograph showing no evidence of healing. Gutta-percha cone is inserted in the buccal gingival sulcus. ***D.*** Clinical photograph showing treatment of the root surfaces and removal of the periradicular lesion. ***E.*** One-year follow-up radiograph demonstrates evidence of active healing.

Primary endodontic disease with secondary periodontal involvement should first be treated endodontically. Treatment results should be evaluated in 2 to 3 months and only then periodontal treatment should be considered. This sequence of treatment allows sufficient time for initial tissue healing and better assessment of the periodontal condition.[116] It also reduces the potential risk of introducing microorganisms and their by-products during the initial phase of healing. Aggressive removal of the periodontal ligament and underlying cementum during interim endodontic treatment might adversely affect periodontal healing.[117] Lesions of endodontic etiology will heal, in most cases, following adequate endodontic treatment, leaving the periodontal component to be treated.

Primary periodontal disease with secondary endodontic involvement and true combined endodontic-periodontal

diseases require both endodontic and periodontal considerations. The prognosis of primary periodontal disease with secondary endodontic involvement and true combined diseases depends primarily upon severity of the periodontal disease and periodontal tissues response to treatment. True combined diseases usually have a more guarded prognosis. The prognosis of combined diseases depends mainly on the success of periodontal therapy.

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Chapter 37

Endodontic Therapy in the Elderly Patient

Thomas A. Levy, Jaydeep S. Talim, Ilan Rotstein

You can't help getting older but you don't have to get old

—George Burns

In the last decade, the number of aging patients has been constantly growing, and in the coming years, an even more rapid growth is expected (Figure 37-1). Aging population is a global phenomenon having profound implications on human life, society, and economics. In 2009, the elderly population (65 years or older) in the United States reached 40 million, approximately 15% of the population for that year.[1] By 2030, it is estimated that the number will reach about 72 million individuals.[1] In the United States, the average life expectancy also increased between the years 2000 and 2010. For women it increased by 1.7 years (to 80.6 years) and for men by 2.1 years (to 78.6 years).[2]

In other highly populated countries, such as China and India, a similar pattern occurs. China already has 180 million people over 60 years of age, and it is estimated that the number will reach 240 million by 2020, and 360 million by 2030.[3] In India the number of citizens over 60 years of age is estimated to increase from 100 million to more than 170 million by 2026[4] (Figure 37-2). A sevenfold increase in population over age 80 is also expected.[5]

As the population ages, elderly patients become a more heterogeneous social and economic group. Cognitive status, multiple chronic diseases, and medications add to the heterogeneity of this population. Consequently, physical, emotional, and psychological conditions of the elderly patient must be considered for proper and successful oral healthcare, including endodontic therapy. It is thus important to provide a practical evidence-based endodontic approach that enables appropriate patient management and maintains and/or improves oral health as part of total healthcare services.[6,7]

Elderly patients also constitute a growing share of dentistry's economy. A simple projection for the next 20 years shows the importance these patients will have on dental finances (Figure 37-3). Based on today's visit rates and average expenditures, patients age 61 and older will increase their contribution from 23% of total dental expenditures in 2006 to 28% in 2018 and to 32% in 2030. The number of endodontic procedures in the elderly population in the United States has also been growing steadily.[8] Estimates suggest that as many as 62% of people over age of 60 have at least one tooth with apical periodontitis of pulpal origin.[9] It is therefore expected that clinicians performing endodontic therapy will see more patients with increasing complexity of medical conditions. This is highlighted by a shift of disease from infectious and acute illnesses to more chronic and degenerative diseases.[2] Issues related to barriers in access to endodontic care of elderly patients will become increasingly significant.

This chapter discusses the physiological and pathological changes in the aging dentition as well as clinical considerations and expected endodontic treatment outcomes for the elderly endodontic patient.

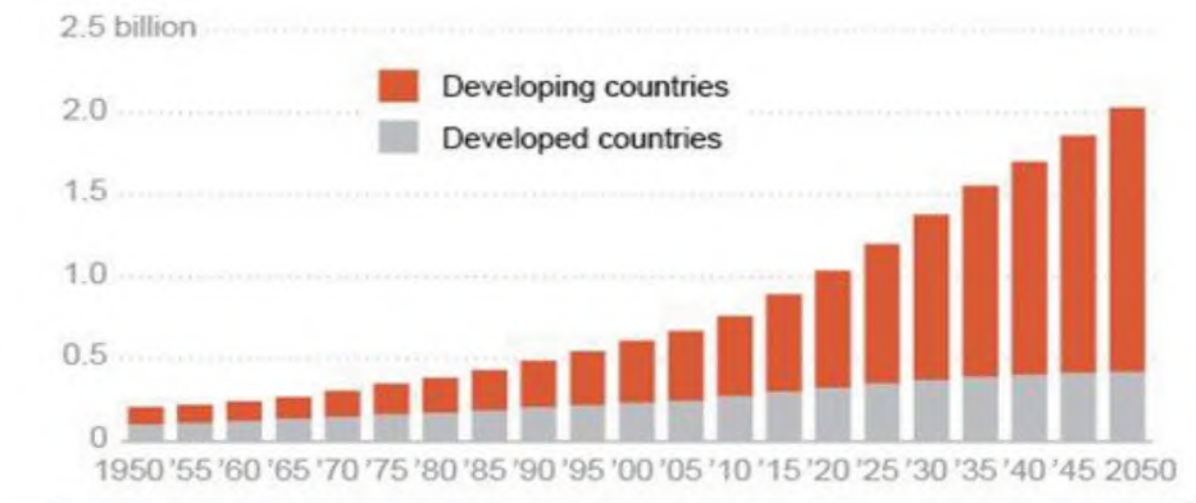


FIGURE 37-1 Total world population aged 60 and over. (Adopted from Kristen G. World not ready for aging population. Global AgeWatch, created by elder advocacy group HelpAge International and the U.N. Population Fund Global Study. 2013.)

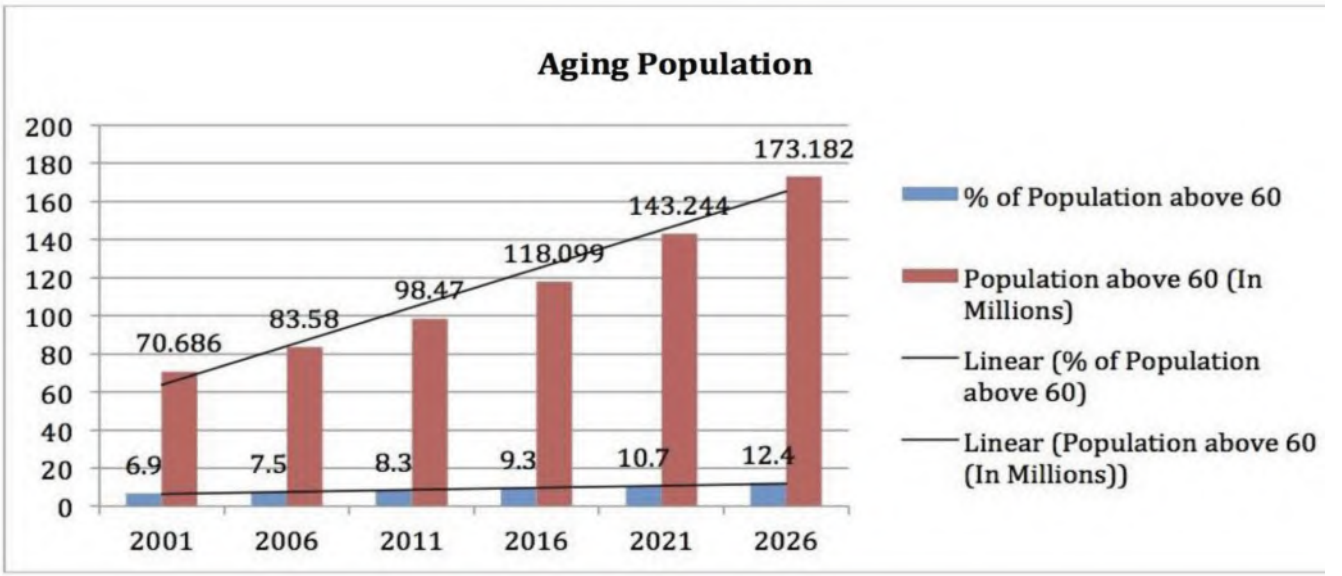


FIGURE 37-2 Linear population above 60 (In Millions). (Adopted from the United Nations Department of Economics and Social Affairs/Population Division. World Population. Aging 2013.)

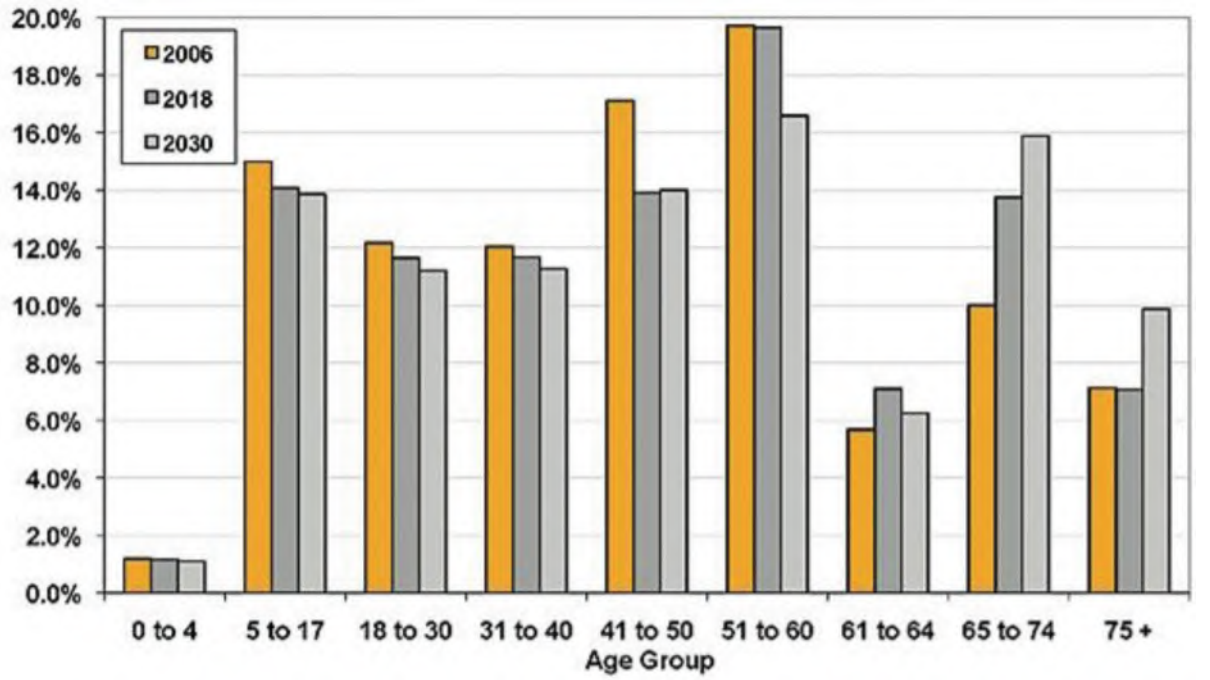


FIGURE 37-3 Projected Shares of General Dental Expenditure by Age Group. (Adopted from Center for Medical services, National Health Expenditure Projections 2008–2018, Washington DC. Centers for Medicare and Medical Services, Office of the Actuary, 2009.)

PHYSIOLOGICAL CHANGES

Dental Tissues

With age, several physiological and degenerative changes occur in the enamel, dentin, cementum, and pulp.

Enamel

Enamel is the hardest biological substance in the human body, comprising of 92% hydroxyapatite, 6% water, and 2% organic matter.[10,11] A broad spectrum of physical and chemical changes occurs in aging enamel. These changes are caused by caries, oral habits, attrition, abrasion, erosion, trauma, and dental treatment. The enamel surface becomes more susceptible to insults due to reduction in the inter-prismatic organic matrix.[12] Also, the hardness of its outer surface is decreased by almost 12%.[13] These molecular changes cause the enamel to become more brittle and may also contribute to formation of cracks and micro-fractures in elderly patients.[14]

Clinically, these changes may lead to tooth fracture by propagation and possible endodontic treatment failure due to coronal leakage from cracks harboring microorganisms.

Dentin

Dentin consists of 47% hydroxyapaptite, 30% organic matter, and 23% water.[10,11,15] It is the most voluminous structural component of the human tooth. Formation of primary dentin occurs before apex closure, while secondary dentin formation occurs after root formation and is observed throughout the patient's life.

In maxillary incisors, secondary dentin formation occurs primarily on the lingual wall of the pulp chamber while in molars it mainly occurs on the floor of the pulp chamber.[16] Tertiary dentin is formed in response to trauma of microbial, physical, or chemical origin. It can be either reactionary, formed by original surviving odontoblast cells, or reparative, formed by new odontoblast-like cells following the death of primary odontoblasts.[17] Tertiary dentin is less tubular and thus more permeable. Sclerotic dentin, on the other hand, is hyper-mineralized and less permeable resulting in a decreased sensation to cold, hot, sweet, and pain.[18]

Dentin deposition is often more marked in the coronal area of the root canal system, with deeper areas of the root canals remaining patent even into very old age. However, apical dentin contains fewer tubules and thus has potentially fewer areas for remaining microbes to reside in the post-obturation phase.[19] Therefore, a well prepared, irrigated, and obturated root canal system will have very good prognosis.

With age, several structural changes in dentin occur. Dentin of older teeth contains less water than that of younger teeth and is more likely to have cracks develop within its structures.[20] With age, the dentin undergoes reduction in fracture resistance, contributing to mechanical changes and possible structural failures.[20]

Calcifications or calcific metamorphosis in the pulp lumen can result from aging, caries, trauma, or dental treatment such as pulp capping.[21] Calcifications can be found in form of free, attached, or embedded denticles (dentin-like pulp stones) or diffuse (linear) calcifications that are atubular or less tubular.[22] Pulp stones are more prevalent in the coronal pulp, whereas diffuse calcifications tend to be found in radicular pulp. Pulp stones are usually translucent and, upon removal, reveal a darker pulpal floor with developmental fissure lines. As part of endodontic treatment, it is essential to remove pulp stones and calcific tissue, since necrotic or degenerated pulp tissue and microorganisms may be harbored in crevices between the calcific tissue and the dentin.

Certain medications, taken for systemic conditions, may cause dentin hyper-calcification and loss of vertical height of the pulp chamber. It was reported that patients taking statins to treat high blood cholesterol levels had a higher prevalence of root canal obliterations.[23] Another study reported that patients using Prostacyclin (PGI2), prescribed for Raynaud's syndrome, showed an increase in tertiary dentin formation.[24] In such cases, the teeth may appear darker.

Cementum

Cementum thickness increases threefold between the ages of 10 and 75 years.[14] The thickest layer is found at the apex. Along the root, varying degrees of thickness are found, depending on recession and wear of the root surface.[14] Cementum width ranges from 100 microns to 200 microns in young teeth and from 400 microns to 500 microns in older teeth.[25] This cementum thickening causes an increase in the distance between the anatomic apex and the apical foramen,[26] as well as the radiographic apex and cemento-dentinal junction (CDJ).[17] This may affect correct assessment of working length as it can give a radiographic appearance of the working length being short. Electronic apex locators prove very beneficial in avoiding such errors. For more details, see Chapter 19.

In addition, the number and size of portals of exit, apical, and accessory foramina, may decrease with age and become less permeable due to calcification.[27] The effect of this phenomenon on the outcome of endodontic treatment is not completely clear.

Pulp

With age, the volume of the pulp decreases and a reduction in the neurovascular components occurs.[28–30] These changes decrease cell density of the pulp tissue (fewer fibroblasts, odontoblasts, and antigen presenting cells), causing fibrosis and altering the dentinogenesis.[31,32] These events lead to increased dentin thickness, with continuing reduction of the lumen in the pulp chamber and root canal system. Such changes, depending on past history of caries, restorations and other stimuli, may be either physiologic or pathologic in nature. Generally, the number of cells in the dental pulp diminishes with age as proliferative activity peaks early in life and then decreases.[33] However, cell proliferation may be maintained well into adulthood and continue into old age.

Odontoblastic activity and continued deposition of secondary and tertiary dentin occurs throughout life. However, this activity is reduced with age. A decrease in pulp cell density may reduce the reparative capacity of the pulp after restorative treatment, but the increase in dentin thickness may aid in pulp protection.

In a study of pulp cell functional viability in Wister rats of different ages 1 to 18 months, odontoblasts and sub-odontoblast density decreased significantly with age. This reduction in density may help explain the slower secondary dentin secretion and decreased capacity of pulpal repair. This should be taken into consideration during treatment planning decisions, for example in procedures such as pulp capping.[34]

Studies on histochemical distribution in the aging dental pulp show decrease in the metabolic activity. Capillaries in the sub-odontoblastic layer decrease and there is a thinning of the odontoblast layer.[35] Also the pulp tissue endothelium becomes thinner.[36] Ikawa,[37] using laser Doppler flowmetry, found that pulpal blood flow significantly decreased with age. Interestingly, using RNA microarray technique, genetic changes have been shown to take place, whereby the genes involved in apoptosis are more highly expressed as the dental pulp ages.[38]

Much like pulp vascularity, the number of pulpal neurons is reduced as an individual ages. Bernick[28,29] showed that 90% of teeth of individuals older than 40 years have some degree of calcification of the pulp space that leads to obliteration of nerves. In a later study, Bernick[30] determined that in the aging process there is also progressive deposition of calcified mass originating in the root pulpal space leading to a decrease in the number of nerves in the coronal pulp tissue.

Studies have found that the total number of myelinated fibers decreased with age, particularly at the expense of A-delta fibers. The A-beta fibers, however, remained constant.[39] The decrease in A-delta fibers might be related to reduced sensitivity to perception of pain, as these fibers are fast conducting.[40] In addition, it was found that older patients had reduced pulpal volume that affected both myelinated and unmyelinated fibers, leading to misdiagnosis and improper treatment.[41] Also immune-histochemical findings in mice have shown marked age-related decrease in pulpal Calcitonin Gene Related Protein (CGRP) and Substance-P (SP),[42] and reduction in sensitivity to pulpal stimulation.[43] These changes in the neurovasculature and cellular content are probably the reason why pulp breakdown in the elderly often occurs without the classic symptoms of irreversible pulpitis.[43]

A degenerative pulp tissue may serve as nidus for growth of calcific inclusions. It has been found that 93% of all older teeth have caries, fractures, or defective restoration margins.[44] The majority were found on clinical and radiographic examinations prior to restoration removal.[44] Thus these factors should be taken into consideration during diagnosis, repair of structurally damaged teeth, and addressing pulpal response to restorative procedures.

Pulpal atherosclerosis has been speculated to affect pulpal blood vessels and pulpal response to injury. Interestingly, atherosclerosis could not be demonstrated to occur in the pulp.[45,46] Also, head and neck radiation therapy does not seem to affect the microvasculature of the pulp.[47]

Related Oral Tissues

Periradicular Bone

There is not sufficient data in the literature regarding unique age-related changes in periradicular bone and its response to endodontic treatment. In general, bone formation steadily declines with age leading to a significant reduction in bone mass.[48] The alveolar bone has high plasticity and under physiological conditions, it is preserved by the equilibrium between osteoblastic and osteoclastic activity influenced by parathyroid hormone (PTH), vitamin metabolites, calcitonin, estrogen, plasmatic concentration of calcium and phosphate, neurotransmitters, growth factors and local cytokines.[49] The reduction in bone formation might be due to a decrease in osteoblast-proliferating precursors or to decreased synthesis and secretion of essential bone matrix proteins. The extracellular matrix surrounding osteoblasts has been shown to play an important role in bone metabolism. A possible dysfunction of this matrix might occur concomitantly with the aging process.[49]

Osteoporosis, a multifactorial metabolic disorder, is a major disorder afflicting men and women in old age. The most common structural changes are reduction in size and number of bony trabeculae and thinning of the cortical region, particularly in the anterior region of the maxilla and the posterior region of the mandible. However, it is inconclusive whether this bone heals differently after non-surgical and surgical endodontic treatment. Also there are no documented differences between the radiographic interpretation of pre-treatment and post-treatment evaluation.[50]

Periodontal Ligament

The fiber and cellular content of the periodontal ligament (PDL) decrease with age and its structure becomes more irregular.[51] PDL cells of elderly individuals show lower rates of chemotaxy and proliferation than those of young individuals.[49] Thus the PDL is not an ideal source to obtain undifferentiated stem cells.

Clinically, progressive changes in gingival tissue, due to recession, periodontal disease or periodontal treatment, may expose lateral or accessory canals, that may act as a portal of entry for microorganisms to the dental pulp.[52]

Salivary Glands

Structural changes in both parenchymal and stromal tissues of salivary glands also occur with age. There is a reduction in the volume of acini and an increase in ductal volume, fatty and fibrous tissues, and callus formation in the salivary gland.[53] Salivary IgA, mucins, immunologic, and non-immunologic systems are also reduced in the elderly, making them more susceptible to caries and oral lesions.

Xerostomia, hypo-salivary function or dry mouth is a frequent complaint of elderly individuals and it constitutes a major clinical problem. More than 25% of patients over 65 complain of xerostomia.[54] In one-third of cases, xerostomia does not reflect a real reduction in salivary flow rate but rather the subjective feeling of a dry mouth. Only a relatively small number of patients suffering from xerostomia exhibits known etiology such as radiation therapy or Sjogren's syndrome. In the majority of cases, the etiology may be related to age, disease, and certain medications.[55] Dry mouth oral Rinse (Biotene) has proved effective in controlling xerostomia for a number of elderly patients. Radiation therapy for cancer causes a significant reduction in salivary flow predisposing patients to xerostomia, radiation-related caries, and oral fungal infections.[56]

Xerostomia-related side effects of various medications coupled with gingival recession, root exposure, hypersensitivity, leaking restorations, poor oral hygiene, and root caries may lead to an increased demand for endodontic treatment in the elderly.[57]

Temporomandibular Joint

The temporomandibular joint (TMJ) has unique anatomical and functional features that makes it different from other joints in the human body. Condylar remodeling is a physiologic process that aims to adapt the structure of the TMJ to meet the functional demands. Although structural changes are thought to be related to TMJ dysfunction, the mechanism of these changes, affected by reactive processes such as

remodeling, aging, and osteoarthritis, has not yet been completely clarified. Temporomandibular dysfunction (TMJD) caused by a compromised occlusion from multiple missing teeth, drifted teeth, bruxism, and loss of vertical dimension or arthritic disease may cause acute or chronic pain and affect the inter-incisal opening and duration of time the patient can keep the mouth open during endodontic treatment.[58] This may affect the endodontic appointment duration and limit the access for endodontic instrumentation, especially in posterior teeth.

Although most dental pain appears to be due to intraoral causes such caries, trauma, and periodontal disease,[59] atypical fascial neuralgia may be a cause for referred pain from the muscles of mastication. The accumulation of inflammatory mediators such as cytokines, eicosanoids, and neuropeptides in these muscles evoke chronic pain that may mimic endodontic pain.[60] Each affected muscle can refer pain signals to specific teeth. Diagnosis is aided, especially in elderly patients, by palpating each of the muscles of mastication individually. Pain increases as palpation is performed, however it subsides after repeated palpation or massage. For more details, see Chapter 17.

PATHOLOGICAL CHANGES

Systemic Diseases

Elderly patients may be susceptible to infections due to immunosuppression from a cumulative effect of aging, systemic diseases, and prolonged consumption of prescription drugs. Disease modifiers such as diabetes mellitus, high blood pressure, cardiovascular disease, cancer, and viral infections may alter the progression, severity, and host response to treatment and prognosis.

Diabetes: The documented effects of diabetes on apical periodontitis are characterized by higher prevalence of apical periodontitis, larger lesions,[61] twice the number of flare-ups (4.8%) as compared to non-diabetics (2.3%) and lower success rates.[62] Also preoperative presence of apical periodontitis in root canal treated teeth in diabetic patients showed lower success rate than in non-diabetics.[62]

Elevated blood glucose may have severe pathological consequences. In patients with sustained hyperglycemia, proteins, including collagen, become irreversible glycated to form advanced glycation end-products (AGEs). Altered host response to infection occurs in diabetics and may be related to accumulation of AGEs and their interaction with their receptors RAGEs in tissue, mediating chronic cellular perturbations, dysfunction, and alteration in tissues ability to respond to infection. Histologically, there is increased vascular permeability and enhanced expression of adhesion molecules onto endothelial cells and macrophages.

Different cells are affected as result of the AGE-RAGE interaction. Macrophages release pro-inflammatory cytokines and matrix metalloproteinases. Neutrophils show impaired adherence, chemotaxis and phagocytosis, whereas fibroblast exhibit increased activity of matrix metalloproteinases and impaired collagen synthesis. These events may lead to an exaggerated and sustained response to infection and diminished reparative response.[63] Recall procedures for immunologically suppressed patients may need to be extended due to delayed healing patterns.

Hypertension: It is estimated that more than 50 million people in the United States have high blood pressure or are taking antihypertensive medications. Lack of compliance is a major issue in medical treatment of hypertensive patients. Therefore, it is prudent for the clinician to measure blood pressure along with other vital signs at every visit.

The decision, whether to administer a local anesthetic agent containing vasoconstrictor to a patient with hypertension or other cardiovascular disease, is a common concern amongst clinicians. One of the primary advantages of vasoconstrictors in dental local anesthetics is to delay the absorption of the anesthetic into the blood stream. It increases the depth and the duration of anesthesia while decreasing the risk of toxic reaction. Additionally, the vasoconstrictor provides local hemostasis. Epinephrine and levonordefrin are the two vasoconstrictor agents commonly used in dental local anesthetic formulations. Although they do have slightly differing cardiac effects, they carry the same precautions for their use. For more details, see Chapters 18 and 31.

If a patient is suffering from severe uncontrolled hypertension, elective dental treatment should be delayed until the blood pressure is under control. However if emergency dental treatment is required, the clinician may elect to use one to two cartridges of local anesthetic with a vasoconstrictor. This dose will have minimal physiologic effect and will provide prolonged anesthesia. If the anesthesia wears off too soon, endogenous epinephrine produced by the patient, resulting from painful experience, will be greater than the amount of epinephrine from the dental anesthetic cartridge.[64]

Another concern is the possibility of adverse interaction between the local anesthetic agent and a patient's antihypertensive medication, particularly the adrenergic blocking agents. The nonselective beta-adrenergic drugs, such as propranolol (Inderal), pose the greatest risk of adverse interaction.[64] In these patients, an injection of vasoconstrictor-containing local anesthetic may produce marked peripheral vasoconstriction that could potentially result in a dangerous increase in blood pressure, due to the pre-existing medication-induced inhibition of the compensatory skeletal muscle vasodilation. This compensatory skeletal muscle vasodilation normally acts to balance the peripheral vasoconstriction effects in non-medicated patients. The cardio-selective beta-blockers (Lopressor, Tenormin) carry less risk of adverse reactions. For more details, see Chapters 18 and 31.

Cardiovascular disease: Cardiovascular diseases (CVDs) are known to originate from endothelial inflammatory dysfunction and are influenced by risk factors such as smoking, diabetes, hypertension, and dyslipidemia. The pathogenesis of atherosclerosis involves the formation of an atheromatous plaque on the inner walls of major blood vessels leading to myocardial infection, stroke and even death. Mature plaques are composed of lymphocytes, macrophages, and bacteria.[65]

Chronic inflammation plays a crucial role in the pathogenesis and progression of atherosclerosis and at the same time promotes acute CVD events such as plaque rupture and coronary thrombosis. Both long-standing inflammation and acute-phase response characterized by increased systemic levels of C-reactive protein (CRP),[66,67] that is associated with many systemic effects of inflammation, have been implicated with increased risk of CVD. However, well-controlled peer-reviewed studies failed to provide any direct causal relationship between endodontic infection and increased risk of CVD.[68,69]

Pacemakers and implantable cardioverter defibrillators (ICDs) are devices that regulate cardiac rate and rhythm. Both are susceptible to electromagnetic interferences. Piezoelectric instruments and electronic apex locators have shown to have no effect on these devices.[70]

Viral infections: The incidence of herpes zoster (shingles) caused by varicella-zoster virus (VZV), is higher in elderly individuals.[71] After causing chicken pox in childhood, the virus remains latent in dorsal root ganglion cells for possibly future reactivation. The herpetic lesions are self-limiting and typically follow the innervation pathway of the nerve. Patients with herpes zoster in the trigeminal nerve may present symptoms of pulpal pain without showing any pulpal disease. Sometimes the viral infection may also be followed by neuropathic pain along the pathway of the affected nerve, causing post-herpetic neuralgia that can mimic odontogenic pain.[72] A few case reports have shown pulpal pathosis (pulpitis, necrosis, or internal resorption and apical periodontitis) following the appearance of herpetic lesions.[73] Therefore, herpes zoster should always be ruled out as a potential etiology when no local irritants are present. Accurate diagnosis and management of the underlying condition rather than endodontic treatment should be the appropriate course of action.[74]

Cancer: Ignoring oral healthcare in aging patients undergoing cancer radiation and chemotherapy can predispose them to serious oral infections due to immunosuppression. Medical consultation for pre-medication and treatment and laboratory values for absolute neutrophil count must be obtained prior to any surgical procedures. In patients with a post-radiation total dose greater than 6500 cGy, endodontic treatment with de-coronation is preferred over extraction due to increased risk of osteonecrosis.[75] Preventive treatment using fluoride and artificial saliva rinses will minimize radiation-associated caries and opportunistic fungi.

Dementia and Parkinson's Disease: Movement disorders (dyskinesia), such as Parkinson's disease,[76] Dystonia, Gilles de la Tourette's syndrome, and Huntington's disease, are characterized by involuntary body movements. Some generalized dyskinesia have focal manifestations in the orofacial region. However, the available information in the literature is limited.[77]

Clinically, patients' involuntary movements while taking radiographs, performing CBCT scans, or during other diagnostic procedures may make interpretation difficult. Thus preoperative radiographic assessment of anatomy and pathology, assessment of working length, and post-treatment assessment of obturation and healing patterns may be challenging (Figure 37-4).

The clinician should accommodate the procedure for involuntary patient movements during use of any rotary instrumentation or working with the enhanced magnification. Constant patient movements may reduce the efficacy of the operating microscope.

For more detailed discussion on the medically complex patient, see Chapter 31.

Medications

Elderly patients suffering from systemic disease are usually consuming a variety of medications. Twenty five percent of all hospitalized patients are taking six or more prescribed medications.[78] Ninety percent of the general population over 70 years of age takes medications on a regular basis.[79] One-third of this population takes more than three medications.[79] Care must be taken to avoid complications and possible unwarranted interactions. Often, patients will not mention their medical condition and medications (prescription or non-prescription) since some do not see its relevance to dentistry. Therefore, patients or their family members should be thoroughly questioned regarding all prescription and non-prescription medications.

Several medications may cause osteonecrosis of the jaw. Methotrexate, an immunosuppressant used in treatment of chronic diseases such as rheumatoid arthritis, Crohn's disease, and ulcerative colitis as well as bisphosphonate, used in the treatment of multiple myeloma, Paget's disease, metastatic cancer, or osteoporosis, can cause the patient to be more susceptible to osteonecrosis of the jaw.[80] As stated earlier in this chapter, in these patients non-surgical endodontic treatment (sometimes followed by de-coronation) is the treatment of choice until extraction or surgical procedure is permitted.

Trauma

According the Centers for Disease Control and Prevention, each year millions of individuals aged 65 and older fall.[81] Falls are usually due to frailness, postural imbalance, exhaustion, or fainting. Falls can cause moderate to severe injuries, such as limb fractures and head and neck trauma. One in three individuals 65 or older fall each year but less than half inform their healthcare provider.[82] In 2013, 2.5 million non-fatal falls of older adults were treated in emergency departments and more than 734,000 of these patients were hospitalized.[83] If the patient is referred for endodontic treatment directly after trauma, it is essential to rule out any underlying cranial injury that would require urgent hospital care. The management of dental injuries is the same in the elderly patient as in the younger patient. Routine follow-up appointments, to monitor pulp status and to provide endodontic treatment before the root canal system undergoes excessive calcification, will be beneficial to both the patient and the clinician.

Sometimes, elderly patients may present in the dental office with pulp necrosis or a discolored tooth and not report any history of trauma. Careful medical history often reveals a history of general anesthesia and intubation performed a few years earlier.

Dental trauma is the second most common complication of intubation.[84] This type of trauma has been termed *silent trauma*[85] (Figure 37-5). Following silent trauma, teeth heal by laying down secondary dentin with pulp canal obliteration

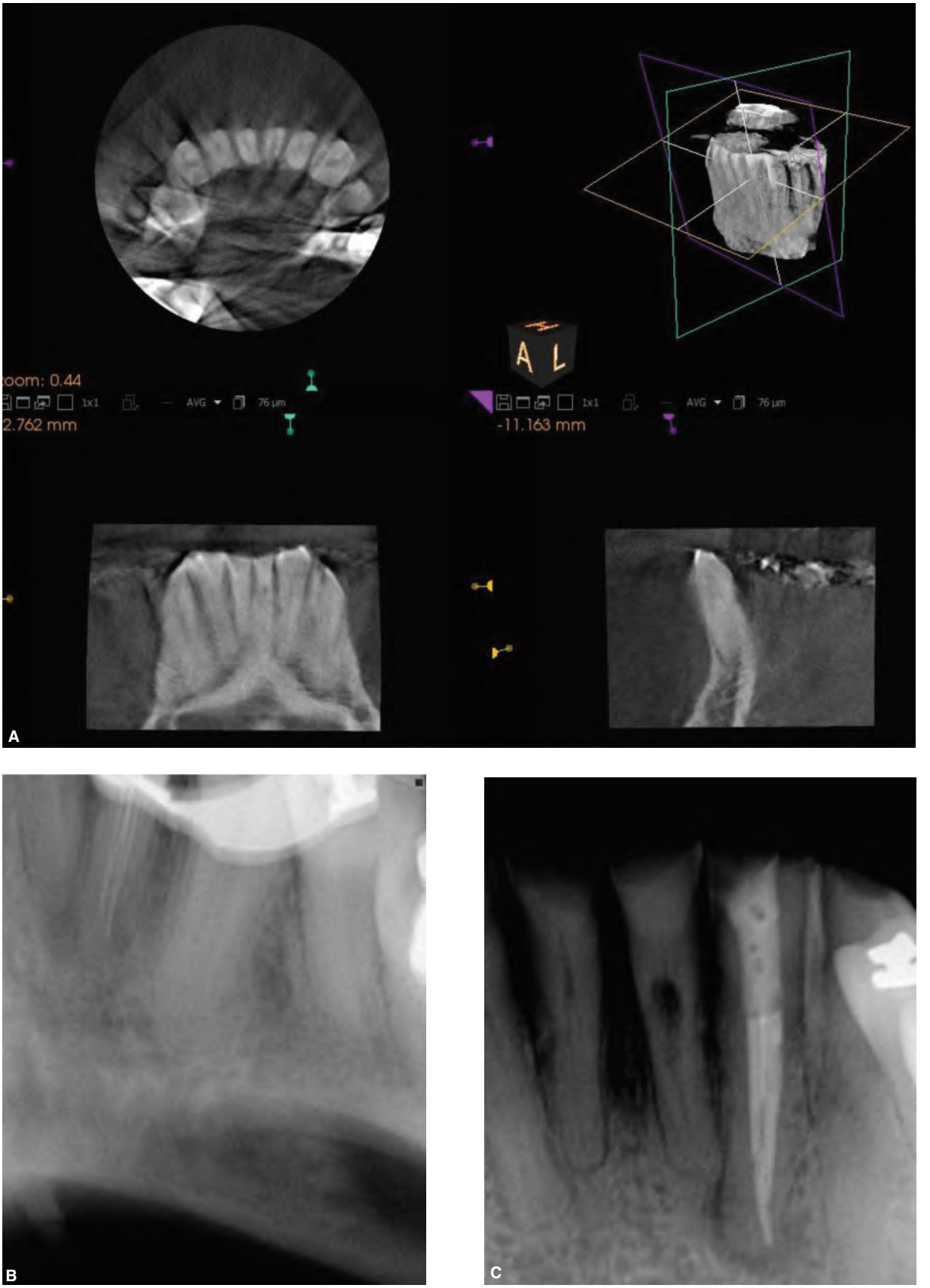
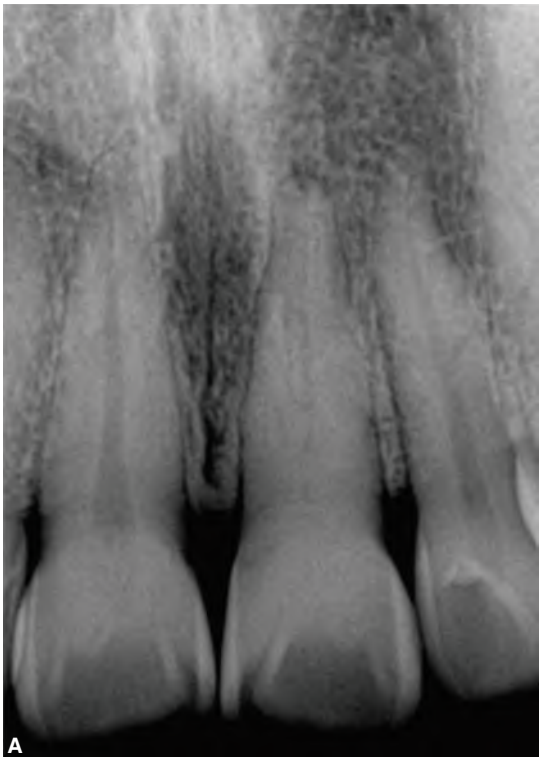


FIGURE 37-4 Radiographic distortions associated with patient's involuntary movements. ***A.*** Cone beam computed tomography (CBCT) taken on an 81-year-old male patient with Dystonia. Note the motion artifacts in the CBCT image analysis. ***B.*** Distortion of length assessment radiograph. ***C.*** Postoperative periapical radiograph of a mandibular canine showing voids in obturation, restorative material due to patient movement.



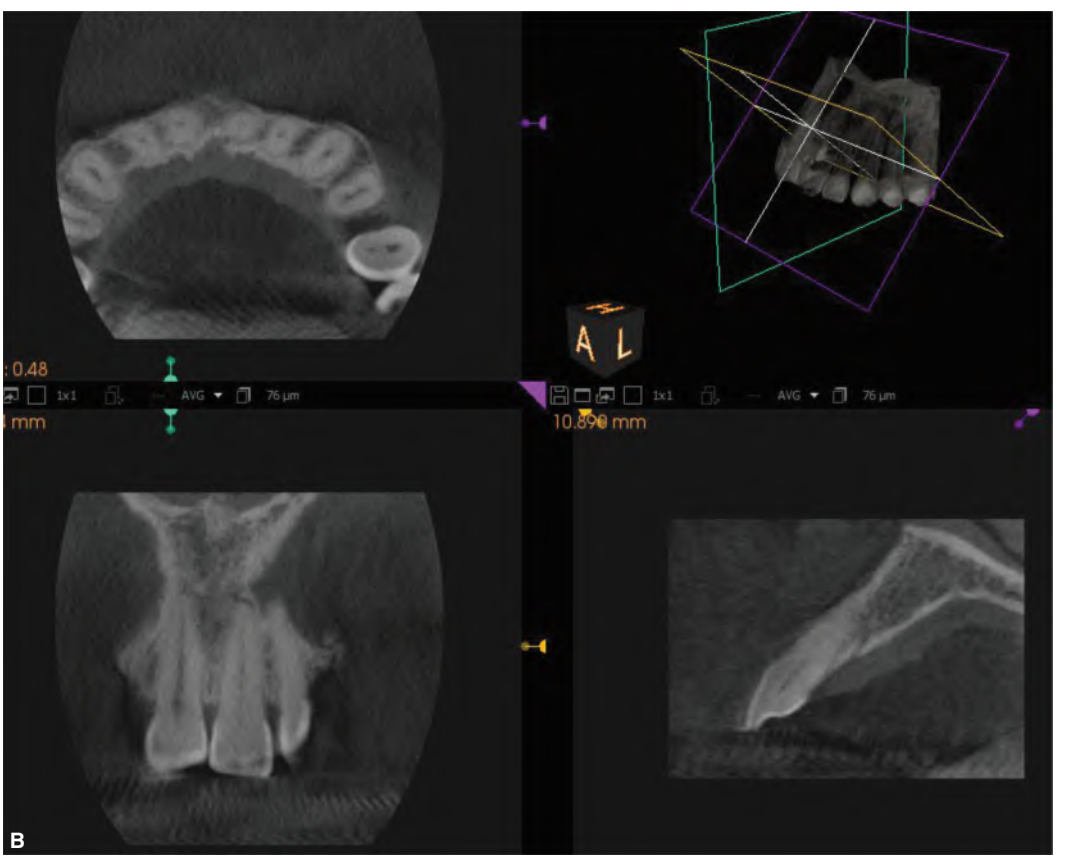


FIGURE 37-5 (*Continued on facing page*)

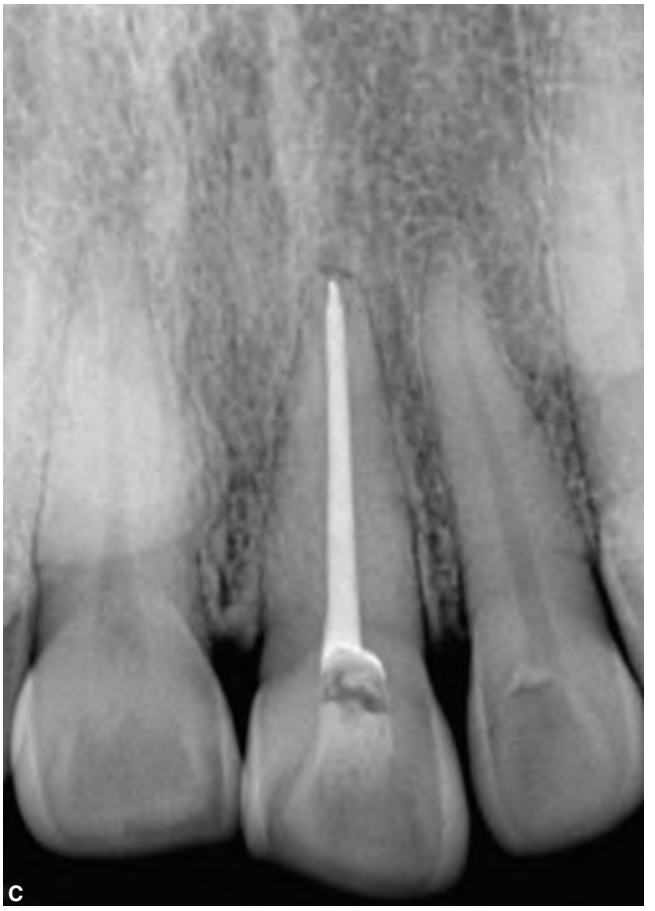


FIGURE 37-5 Patient with etiology of silent trauma. ***A.*** Radiograph of a symptomatic non-vital maxillary left central incisor in a 71-year-old female. The pulp space appears obliterated. Initially, the patient did not report of any history of traumatic injury to the tooth. After further inquiry, the patient recalled that she underwent surgery under general anesthesia 2 years previously. ***B.*** Cone Beam Computed Tomography (CBCT) views (coronal, axial, and sagittal) showing presence of patent root canal. ***C.*** Postoperative radiograph. The root canal system was prepared and obturated. Following treatment, the patient was asymptomatic.

and calcific metamorphosis, or undergo pulp necrosis, along with inflammatory resorption and/or replacement resorption.[86] Although a root canal system may appear obliterated on a periapical radiograph, histologically it may not be completely calcified and can be treated endodontically.[87]

In crown fractures with pulpal involvement, root canal treatment may be the treatment of choice, as prognosis of pulp cap procedure will be poor, due to diminished capacity of the pulp for healing.[88] In cases of crown-root fractures extending subgingivally, elderly patients may not agree to extensive treatment such as crown lengthening or orthodontic-surgical extrusion procedures to establish ferrule.

In root fracture cases treatment may include repositioning of the coronal segment and stabilization by a flexible splint for 12 weeks to help pulpal and periodontal repair. However, in root fractures associated with a persistent sinus tract or periodontal defect, the teeth will need to be extracted due to poor prognosis.

CLINICAL CONSIDERATIONS

Endodontic Treatment Outcomes

Based on epidemiological studies there is no correlation between age and outcome of endodontic treatment. Endodontic treatment can be as predictable in the elderly patient as in younger patients.[89] Also, studies have shown similar healing pattern of oral tissue in the young and the old, with a delay in healing response in older teeth.[90] Healing is primarily affected by vascularity in the damaged tissue and not age *per se*.[91]

There are no medical contraindications to endodontic therapy in the elderly except for the American Society of Anesthesiologist (ASA) type IV or type V patient, needing urgent medical attention. Age *per se* does not seem to affect the outcome or healing of endodontic treatment.[91–96] Outcomes studies have shown that treatment outcome is mainly affected by the preoperative presence of apical periodontitis, the quality and extent of the root canal filling, and quality and type of restoration following root canal treatment. However, patients with diminished healing capacity caused by conditions such as diabetes,[62] immunosuppression, or use of certain medications may influence the duration for periradicular healing.

Endodontic outcome studies have consistently shown a survival and retention rate higher than 90% in teeth treated with non-surgical root canal therapy.[97,98] Therefore, when indicated, endodontic treatment is a good alternative to extraction and tooth replacement.[99,100] The decision to perform endodontic treatment should be based upon medical health of the patient, prognosis, and cost-benefit of treatment.

Treatment prognosis in the elderly patient may sometimes depend on other factors than just long-term outcomes. Occasionally, a tooth with esthetic consideration but questionable restorability might be a candidate to be retained,[101] or a non-functional tooth may need to be retained where extraction might cause health complications (Figure 37-6).

Accessibility to Care

Elderly patients may need easy access to the dental office and operatory. Some may use accessories such as a cane, wheelchair, or a motorized cart. If the patient is ambulatory, access to the office requires a ramp and/or elevator. Waiting rooms, operatories, and restrooms need to be handicap accessible. Elderly patients should be monitored by the office staff from the moment they enter the dental operatory until discharge upon completion of the treatment. In tandem, as more elderly patients find it difficult to visit a dental office, dental care services at senior citizen community centers and mobile dental clinics should also be available.

Appointment Time and Duration

Appointments should be scheduled according to the patient's preference. Usually, morning appointments are better,[102,103] however, they should be synchronized with the patient's medication, meals, or other medical treatment regimens. Appointments should preferably be of short duration

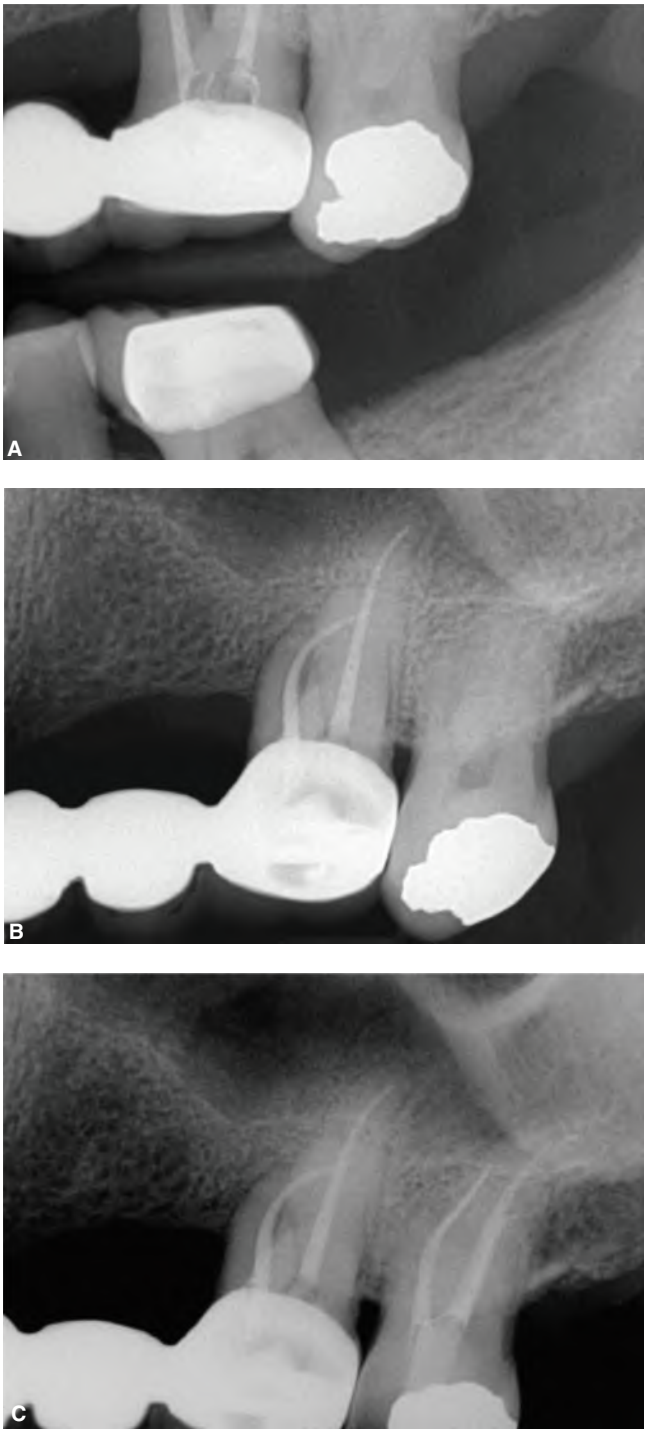


FIGURE 37-6 Medical contra-indication to extraction. ***A.*** Preoperative bitewing radiograph of a symptomatic supra-erupted maxillary left second molar in a 83-year-old male with a history of a recent open heart surgery. Although non-functional, the tooth was treated endodontically. ***B.*** Preoperative periapical radiograph. ***C.*** Postoperative radiograph. Following treatment, the patient was asymptomatic.

based on the patient's physical and medical conditions. Also, whenever possible and indicated, the number of treatment visits should be reduced since many elderly patients rely on being accompanied by others during dental visits.

Medical History

Due to visual, auditory, and cognitive impairment, the Health Insurance Portability and Accountability Act (HIPAA) of 1996 mandates a family, friend, or caregiver may need to assist in completing the medical and dental history and in describing the chief complaint. The clinician should thoroughly review the medical history, need for medical consultation, premedication, and drug interactions before initiation any dental treatment. Over the counter non-prescription medications, natural, herbal or homeopathic supplements may affect and alter the clotting mechanism especial during endodontic surgery and should be identified. For more details, see Chapter 8.

Dental History

Details of dental history of aging patients are rarely complete and may show treatment by several dentists at different locations. The clinician should make all attempts to research and review old dental records and gather as much information as possible. The number of times the tooth has been restored, the addition of restorative surfaces on a tooth over time, history of falls, and surgery under general anesthesia may be pertinent to the diagnosis. Exposure to factors that contribute to oral cancers accumulates with age and many systemic diseases may initially manifest prodromal oral signs or symptoms.[104]

Diagnostic Tests

The positioning of intraoral radiographic films and inflexible digital sensors may be difficult due to decrease in muscle tonicity, limited intra-oral opening, and presence of exostoses. Use of soft foam padding around the edges of the digital sensor or smaller pediatric sensors should be considered. A careful preoperative assessment of the bitewing and multiple angled periapical radiographs will help plan access preparation, and alert the clinician if the axial inclination of the crown does not coincide with the long axis of the root. Poorly contoured crowns or over contoured crowns made to compensate for spacing and to close interproximal contacts must be identified. Sometimes, if the tooth undergoing endodontic treatment is heavily restored and calcified, a periapical radiograph of the contralateral tooth may be beneficial in determining the anatomy of the root canal system. The clinician can also assess previous radiographs if available, to determine position and deposition of tertiary dentin over time.

Often it may be prudent to obtain prior permission from the patient and referring dentist to remove a prosthetic crown if access becomes difficult. The clinician may consider and prescribe a CBCT analysis in severe cases of calcification and retreatment after determining the benefit to risk ratio to the patient.[105]

Differential Diagnosis

Differential diagnosis, mainly of pain and its origin, is key for successful treatment planning. The occurrence of arthritic diseases such as TMJD, in either chronic or acute episodes, atypical facial pain, neuritis and neuralgia, maxillary

sinusitis, osteonecrosis of the jaw and tumors may mask or mimic odontogenic pain in elderly patients (Figure 37-7). In many instances these diseases impact quality of physical and psychological function, therefore, these complaints must be acknowledged and addressed.

There is limited evidence of age related changes in the physiological functioning of peripheral and CNS nociceptive pathways from young to older populations. Studies indicate that the aging dental pulp showed alterations in its ability to respond to pain stimuli.[28–30] There are fewer cells, blood vessels, and nerve bundles in the pulp and a marked decrease in the density of nerves is evident. As mentioned earlier, with age there is a subsequent decrease in neuropeptides such as CGRP and Substance P in the pulp. Also calcification and reduction in pulp volume cause a decrease in response to environmental stimulation or induced stimulation of diagnostic tests. Thus, following an insult, the pulp in the elderly patient may undergo necrosis without intermediate symptoms. For more details, see Chapters 8 and 17.

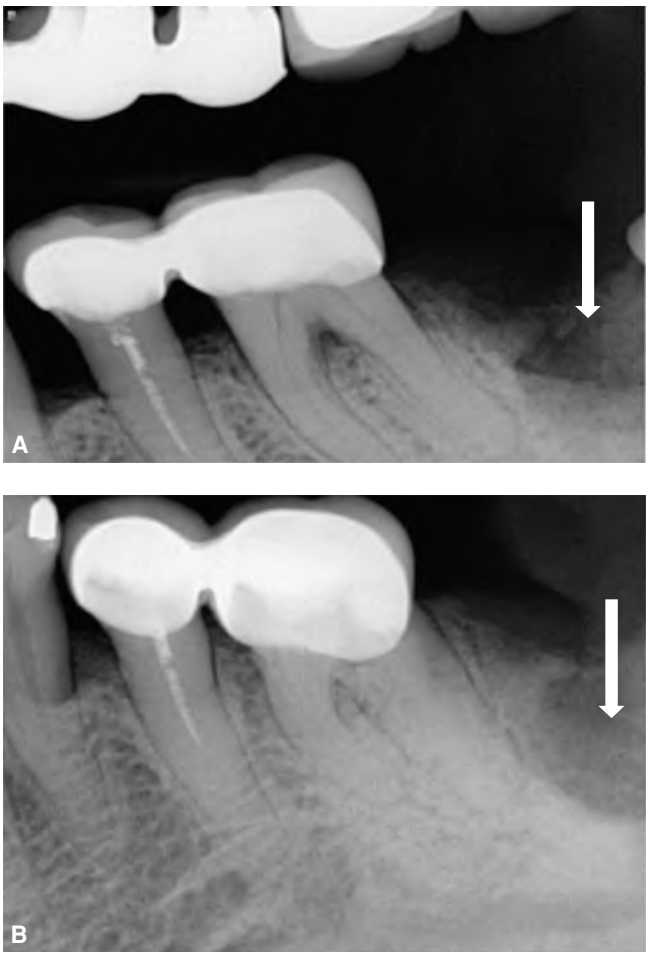


FIGURE 37-7 Pain mimicking endodontic symptoms in a 83-year-old female with osteonecrosis of the jaw (ONJ). The patient was referred for root canal treatment in the mandibular left first molar. Both the mandibular left first molar and second premolar were asymptomatic and responded normally to pulp testing. The patient has been receiving medical treatment for ONJ that developed after extraction of the mandibular left second molar. ***A.*** Preoperative bitewing radiograph and ***B.*** Preoperative periapical radiograph showing area of osteonecrosis in the lower left mandible.

Additionally, a significant number of elderly patients may have multiple restorations on the same tooth over a period of time with reduced pulpal healing capacity and greater tendency to undergo pulpal necrosis.[106] Establishing the culprit tooth in a patient with multiple tooth restorations can be challenging due to decreased response to stimuli.

Case Selection

In most cases, there are no contraindications to endodontic treatment. However, in some cases, case selection should be done cautiously taking into consideration the physical limitations of the patient. On the other hand, in certain medically compromising situations, endodontic treatment should be selected as the treatment of choice. For example, patients presenting with osteoradionecrosis during radiation therapy or those with bisphosphonate-related osteonecrosis of the jaw (BRONJ).

Prophylactic root canal treatment may be indicated in asymptomatic healthy pulps when restorative considerations such as overdenture abutments or in cases of root amputation in teeth with localized periodontal involvement.

Patient Positioning and Comfort

Elderly patients may require special positioning during treatment. They should be seated comfortably in the dental chair equipped with an adjustable headrest and pillows. Some patients may have difficulty reclining in an extreme supine position due to conditions such as osteoarthritis or neck and back problems (Figure 37-8). At the end of the endodontic procedure bring back the patient from supine position in a gradual manner in order to avoid orthostatic hypotension. Bite blocks should be used to stabilize the jaw especially in patients with conditions affecting muscle tonicity such as Parkinson's disease or dystonia. Bright operatory and surgical

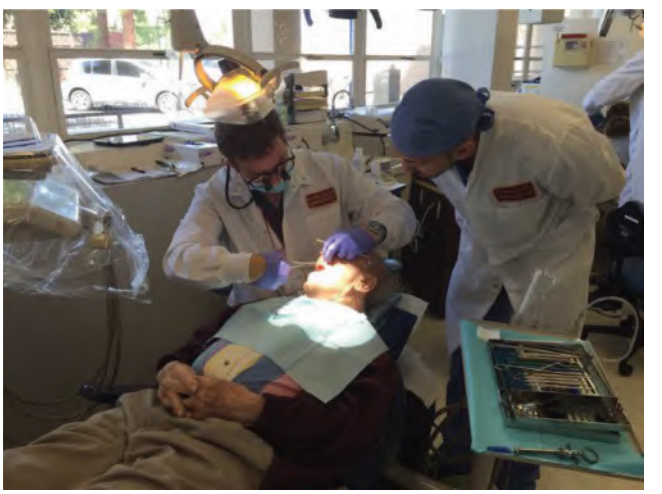


FIGURE 37-8 81-year-old patient undergoing endodontic treatment in a minimally reclined position.

operating microscope lights may be shielded from the elderly patient's eyes by using dark glasses. Also, blankets may be necessary if the temperature in the operatory is too low for the patient.

Prior to discharging the patient, allow them time to recover both mentally and physically. Verbal and written instructions should be given to the patient and to the accompanying individual. Reemphasize the importance of maintaining good oral hygiene and regular recall appointments.

Anesthesia

Older patients tend to be less anxious, have a higher threshold of pain tolerance, and are more likely to prefer less anesthetic, especially if the tooth is asymptomatic. There are no contraindications to use vasoconstrictors in dental local anesthetics in routine endodontic procedures in elderly patients.

Supplemental anesthesia such as intra-osseous or intra-ligamentary injections should only be administered as the last option, after informing and educating the patient of anticipated heart rate increase. It may be advisable to use non-vasoconstrictor local anesthetics such as 3% mepivacaine for such procedures. For more details, see Chapters 18 and 31.

Tooth Isolation

All root caries and defective restorations should be removed to evaluate tooth restorability and to determine if the tooth is salvageable. Placing a clamp may be challenging in patients with decreased muscle tonicity, or with limited oral opening. Often the dental dam may need to be placed after access and location of canal orifices. Avoid clasping teeth that appear weak from extensive caries or restorations especially in the anterior dentition.

Elderly patients may need to be given frequent breaks during the appointment to facilitate breathing or use of restroom. The dental dam may need to be taken off during this time. The tooth should ideally be temporized with cotton and temporary restorative material during this phase. For more details on tooth isolation, see Chapter 19.

Access Cavity

Enhanced illumination and magnification will greatly enhance visibility and aid in access. The clinician needs to be aware, that due to modified patient positioning in the dental chair, the orientation of the crown or the root may change during visualization and access. "Walking" a periodontal probe along the cemento-enamel junction (CEJ) and using the "Laws of Access Preparation."[107] will provide a systematic approach to locating root canal orifices.

Initial access into a calcified pulp chamber may be gained without a dental dam to aid in visualization of the pulp chamber and to avoid iatrogenic procedural errors (Figure 37-9). Orientation should be verified and confirmed visually and radiographically. The traditional "drop into the pulp chamber" may not be felt, making careful exploration with an endodontic explorer and the measurements from occlusal surface to the roof of the pulp chamber more relevant. A radiopaque marker such as gutta-percha from a down-pack gun or calcium hydroxide paste at the depth of the exploration can reveal critical information on a bitewing radiograph (Figure 37-10).

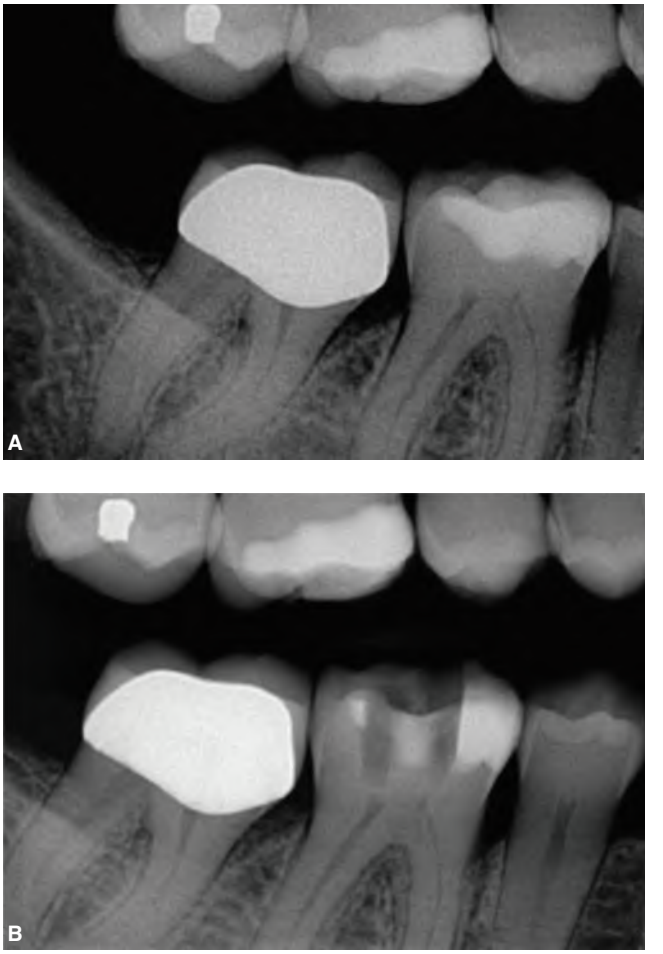


FIGURE 37-9 Access preparation assessment prior to placement of the dental dam in a 69-year-old patient. ***A.*** Preoperative bitewing radiograph of the mandibular right first molar. The pulp chamber appeared partially obliterated. ***B.*** Intraoperative bitewing radiograph taken without the dental dam for orientation purposes.

Often the access preparation may need to be modified or enlarged in order to locate the canals orifices. As soon as the canals are located it is important to place the dental dam. As stated earlier even if the canals appear calcified on a radiograph, histologically a root canal space is always present[87] (Figure 37-11). If the canals cannot be located, it may be wise to schedule for the patient to return or consider other treatment options.

Canal Length Determination

As described earlier, age-related cementum thickening may alter the original locations of apical foramen.[17,27] Electronic apex locator readings, together with good radiographic assessment, tactile sensation, and wet-dry paper point test to

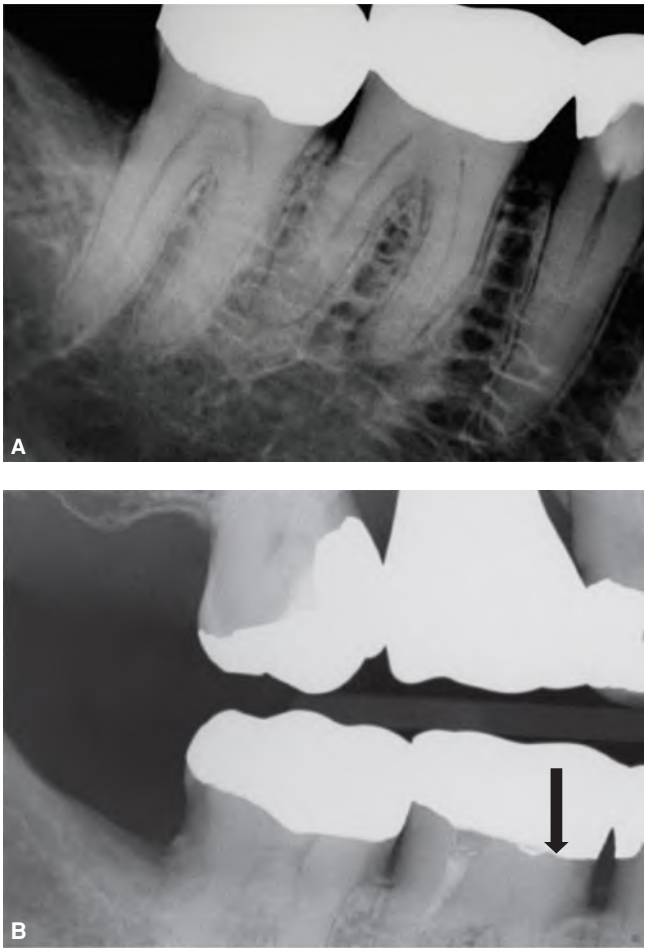


FIGURE 37-10 Use of radiopaque tracer for assessment of access preparation in a tooth with a calcified pulp chamber. ***A.*** Preoperative radiograph of the mandibular right first molar in a 73-year-old male showing a full coverage restoration and apparent obliteration of the pulp chamber. ***B.*** Intra-operative bite-wing radiograph showing access orientation with aid of radiopaque calcium hydroxide.

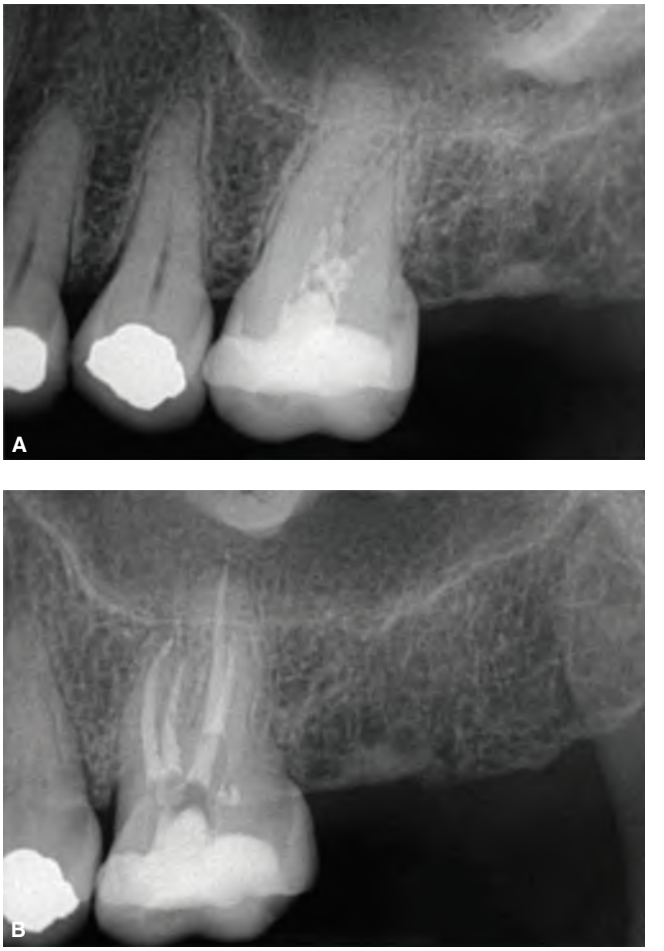


FIGURE 37-11 ***A.*** Preoperative radiograph of the maxillary left first molar in an 82-year-old female. The root canals appear partially obliterated. The patient was referred to the endodontist after an attempt to fully access and negotiate the canals was unsuccessful. ***B.*** Postoperative radiograph showing complete obturation of the root canal system.

determine the working length, are important. Patency may not be achievable in all cases.

Cleaning and Shaping

There are no specific considerations for cleaning and shaping procedures in the elderly patient. After establishing a glide-path and working length, the clean and shaping can be completed using standard protocol. It should be noted that the root canal lumen is expected to be smaller and the dentin tissue more sclerotic. Copious use of chelating agent such as Ethylene Diamine Tetra Acetic acid (EDTA) with specific files such as "Stiff files" (Brasseler), C (Dentsply), and C-plus files (Dentsply/Maillefer) to help negotiation and establish a glide path may be helpful. Some rotary files such as ProGliders (variable taper) and PathFiles (0.02 taper) (Dentsply) may help expedite this initial enlargement of the canal, but are to be used only after establishing a safe glide path for the rotary file. Frequent irrigation with sodium hypochlorite will help soften and dissolve the fibrous pulp tissue greatly facilitating the shaping and cleaning procedure.

Obturation

No special treatment modifications are needed for cold or warm obturation techniques. For more details, see Chapter 22. When using the down-pack and backfill for the warm vertical obturation techniques, the clinician should be conscious of the fact that the elderly patient may not have an adequate intra-oral opening and may move during the procedure. Sometimes a different obturation system such as carrier-based or a single cone technique may have to be considered as an alternative.

Tooth Restoration

Elderly patients may present with teeth that have had multiple restorations or insults over time. At this point, these teeth often need extensive restorations such as full coverage

or onlays. Due care needs to be given to avoid aggressive preparations since these cumulative factors can tilt the balance in favor of pulpal pathosis.

It is essential to place a coronal seal immediately after root canal therapy to prevent failure of treatment due to coronal leakage.[108–110] Older patients are more likely to have previous cervical or sub-gingival root caries. Often, the final restoration may not extend to cover the pre-existing sub-gingival restoration. Ease of application and potential fluoride releasing properties of glass-ionomer or glass-ionomer resin-modified restorative materials makes them preferable over the more technique-sensitive composite restorations. For more details, see Chapter 35.

Retreatment

With age, the incidence of endodontic retreatments may increase, due to coronal leakage from loss of seal, recurrent caries or fracture of teeth, and restorations.[111] Also, overlooked or missed canals are a more common cause of failure in older patients. An isolated symptom, heat sensitivity is usually indicative of a missed canal.[112] Retreatment indications and considerations are the same as in younger patients. For more details, see Chapter 23.

Apical Surgery

Often, with age, the position of anatomic structures such as the maxillary sinus, the inferior alveolar nerve canal, mental foramen, and incisive foramen may change as a result of multiple missing teeth and residual ridge resorption over time. The changes in position of these anatomical entities should be observed and considered during the treatment planning.

Elderly patients may be taking prescription anti-coagulants or self-prescribed low-dose aspirin. A thorough medical history evaluation and questioning will provide key information on allopathic, homeopathic or herbal anti-coagulant agents. Studies have shown that anti-coagulant therapy should preferably not be stopped and hemorrhage during endodontic surgery should be controlled by local hemostatic agents.[113] Post-operatively, patients must be informed about the possibility of ecchymosis. This sub-epithelial hemorrhaging presents itself as purple-red discoloration and is self-limiting. Tissues regain normal color in few weeks.

Sometimes, due to periodontal disease, root resorption or crown attrition, periodontal and endodontic surgery may need to be combined to get adequate crown root ratio and tissue attachment.

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Chapter 38

Endodontic Therapy in the Pediatric Patient

Enrique Bimstein, Yaara Y. Berdan

JUSTIFICATION FOR ENDODONTIC THERAPY IN THE PRIMARY DENTITION

Dental caries is the most common chronic disease of children in the United States, and has been described as a silent epidemic that restricts activities in school, home, parent's work, and often diminishing the quality of life (Figure 38-1).[1–3] Although there has been a reduction in dental caries in permanent teeth over the past few decades, caries prevalence in the primary dentition has only stabilized and may have even increased slightly in some population groups.[1]

Early childhood caries can begin soon after eruption (Figure 38-2) and, if neglected, may rapidly deteriorate to virulent forms of caries, leading to unrestorable teeth, pulp inflammation, and ultimately pulp necrosis and a lasting detrimental impact on the dentition (Figure 38-3).[4]

The American Academy of Pediatric Dentistry (AAPD) states that dental care is medically necessary to prevent and eliminate orofacial disease, infection, and pain, to restore the form and function of the dentition, and to correct facial disfiguration or dysfunction.[5] Dental neglect is defined as dental caries, periodontal diseases and other oral conditions that, if left untreated, can lead to pain, infection, loss of function (Figure 38-3),[2] and in extreme situations may endanger the child's life (Figure 38-4). This represents a most relevant problem in the United States of America as reflected in a recent manuscript that provided representative estimates of hospital-based emergency department (ED) visits during the year 2008 for dental caries, pulp and periapical lesions, gingival/periodontal lesions, and mouth cellulitis in patients who were ≤21-years-old.[6] A total of 215,073 ED visits with dental conditions occurred among children; 41% of them included the diagnosis of pulpal and periapical conditions; while 3% were diagnosed with oral abscess/cellulitis. An additional study done with the purpose of determining the prevalence, types, and treatment of dental emergencies that presented to a tertiary care pediatric ED indicated that, among 247 non-traumatic dental complaints, 59% were in children younger than 5 years old, 8% required hospitalization for intravenous antibiotics, and 82% were discharged from the ED with oral antibiotics.[7] Prevalence of oral disease in children, as well as available data, may vary from continent to continent and from country to country. Readers should check the available publications pertaining to their individual countries.

Primary teeth are more susceptible to the carious process than permanent teeth, due to their smaller size, thinner enamel and dentin, and larger pulp horns (Figure 38-5).[8–10] Additionally, this higher susceptibility is related to the fact that primary teeth show a lower calcium content, slightly

FIGURE 38-1 A child in pain from dental origin.

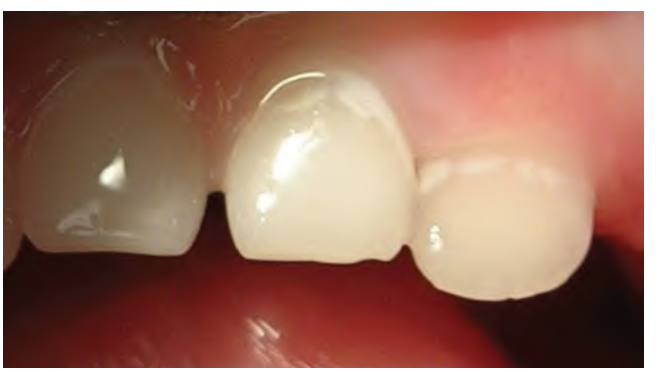


FIGURE 38-2 Early childhood caries in an infant. Note the white spot lesions at the gingival third of the crowns. (Courtesy of Dr. Martin S. Rayman, San Rafael, CA, U.S.A.)

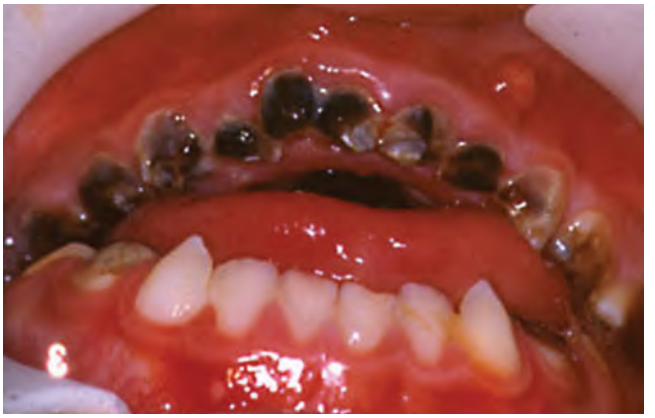


FIGURE 38-3 Severe early childhood caries. Note the extensive crowns destruction and the parulis located at the area apical to the primary maxillary left lateral incisor.

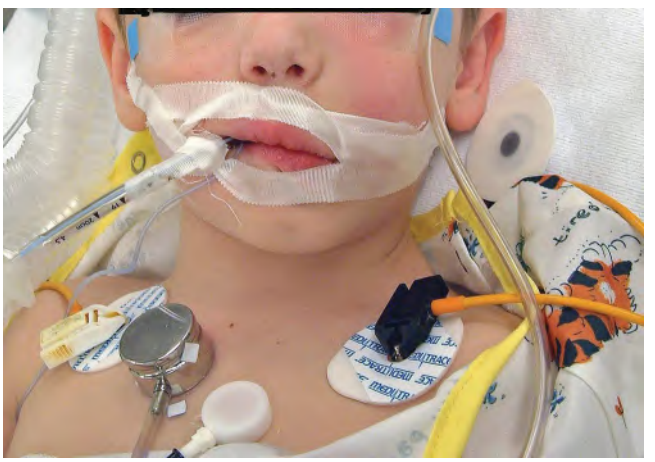


FIGURE 38-4 Child under general anesthesia for emergency treatment of an abscessed primary tooth. Note the facial redness and cellulitis under the left eye.

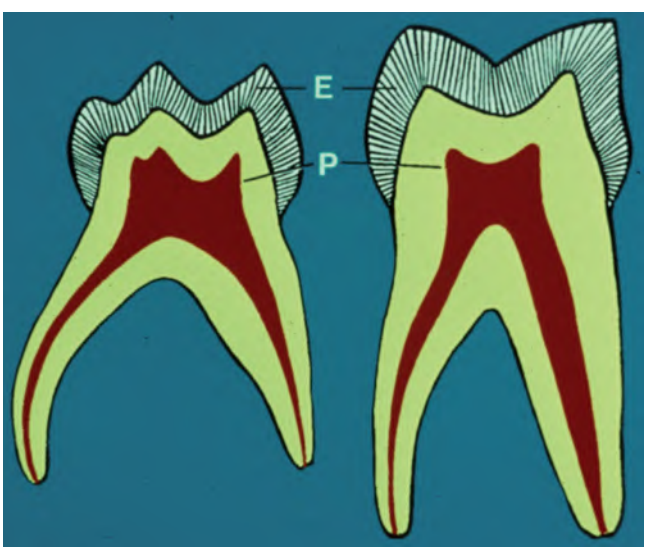
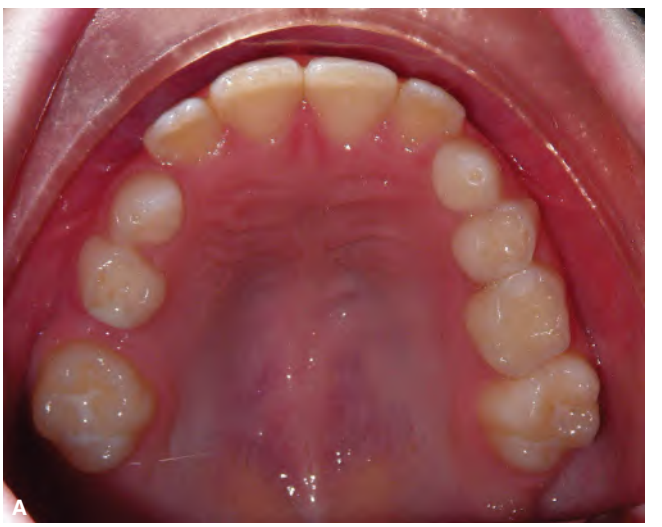


FIGURE 38-5 Contrasting primary and permanent molars. Note large pulp space and thin dentin in the primary molar (E- enamel; P- pulp).

lower calcium-to-phosphate ratio, higher water content, and higher organic content than permanent teeth. Consequently, early childhood caries progresses rapidly owing to the lower degree of mineralization, higher carbonate content, and higher porosity of the mineralized tissues of the primary teeth.[11–13] Clearly, prevention or the early restoration of primary teeth ensuring the omission of pulpal inflammation and infections are fundamental components of the practice of pediatric dentistry.

The assumption that primary teeth are not important since they exfoliate, and that attempts to maintain them with pulpal therapy are unnecessary, fails to take into account the important functions of deciduous teeth. Primary teeth enable children to eat solid foods, aid in speech development, and maintain space for the permanent dentition (Figure 38-6A&B).[8,14] Children's confidence



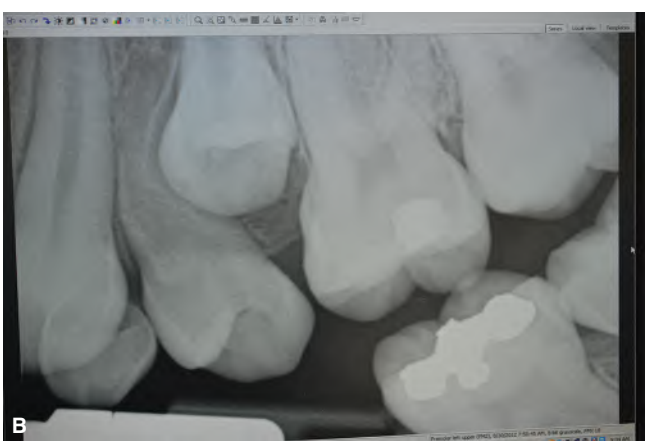


FIGURE 38-6 ***A.*** Clinical picture of a child in the mixed dentition period after early extraction of the primary maxillary right second molar, and mesial drift of the permanent first molar. ***B.*** Periapical radiograph of an adolescent after early extraction of the primary maxillary left second molar and impaction of the succedaneous tooth due to mesial drift of the permanent first and second molars and distal drift of the first premolar. (38-6A, courtesy of Dr. Shaul Yehezkel, Los Angeles, CA, U.S.A.)

and self-esteem are enhanced by the appearance of their teeth. Keeping primary teeth healthy is important not only to prevent a child from experiencing pain and disease, but to enhance their social development, esthetics, quality of life, development and eruption of the permanent successors, and the prevention of the development of detrimental oral habits.[8,14–17] Although space maintainers can be used successfully to reduce the prevalence and severity of malocclusion following premature loss of primary teeth, they are not without complications. Adverse effects associated with space maintainers include: 1) dislodged, broken, and lost appliances; 2) plaque accumulation; 3) caries; 4) damage or interference with the successor tooth eruption (Figure 38-7); 5) undesirable tooth movement; 6) inhibition of alveolar growth; 7) soft tissue impingement; and 8) pain.[18] Therefore, pulpally involved primary teeth should be retained when possible with appropriate endodontic treatments.[19]

Because of the significant value of primary molars and the potential harm of early extraction, it may even be justified to treat a primary molar with a poor prognosis with the intent to maintain the tooth until it is no longer needed as a space maintainer. However, this decision needs to be balanced with the maintenance or creation of a favorable environment that does not interfere with the normal exfoliation of the primary tooth, and the development and eruption of the succedaneous tooth. While alternative strategies for posterior space maintenance and occlusal guidance are frequently required in cases when it becomes imperative to extract primary molars, the loss of a primary incisor after the eruption of the primary canines does not result in space loss, but may be an esthetic concern.[15,16]

According to the AAPD Guideline on Pulp Therapy for Primary and Immature Permanent Teeth, the primary objective of pulp therapy in the primary dentition is to maintain the health and integrity of the teeth and their supporting structures.[18] Several vital (e.g., pulp cap or pulpotomy) or non-vital pulp (e.g., pulpectomy) therapies are possible for primary teeth.[18] Accurate assessment of pulp condition that indicates which treatment is the most appropriate in each individual case may be the most challenging aspect of treatment. This chapter presents updated information on pulp diagnosis, and on selection of the matching pulp treatment for primary teeth, emphasizing the need to take into consideration the individual characteristics of each case, in the best interest of the child.

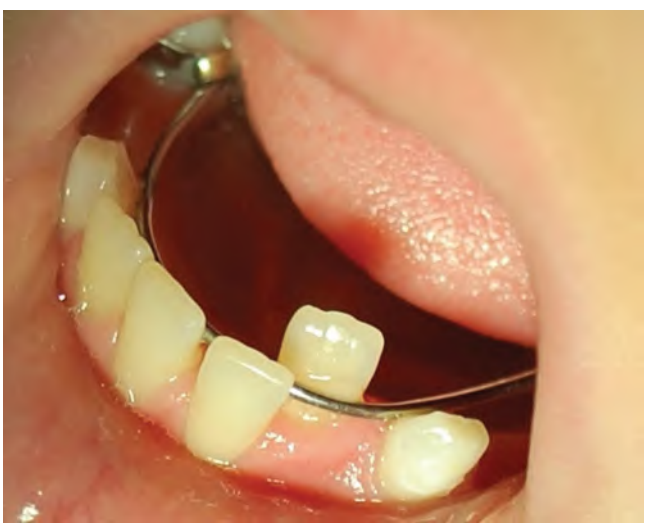


FIGURE 38-7 Ectopic eruption of the permanent mandibular left lateral incisor behind a lingual arch that is preventing its physiological alignment.

RATIONALE FOR SELECTION OF ADEQUATE PULP THERAPY IN THE PRIMARY DENTITION

The treatment of endodontic conditions in primary teeth should take into consideration the child's medical and dental histories, subjective evaluation of the area associated with the current symptoms/chief complaint (as informed by the child and the parent), objective clinical and radiographic findings, value of each tooth in relation to the child's overall oral development and occlusion, potential of inflamed pulp tissues to recover, restorability of the tooth, as well as the child's physical and mental development and behavior, and alternatives to pulp treatments.[18,19] Recognition of the healing potential of the inflamed dental pulp, and awareness of the improvements in dental materials and techniques, has contributed to the development of endodontic primary teeth treatments that have high success rates. Sound relationship between the extent of the carious lesion, the degree of pulp involvement, and the type of treatment are important factors.[18,19]

In order to deliver the most adequate dental care to pediatric patients, the following dynamic variables should be considered:

a. *Growth and development of the dentition*:
The "life cycle" of the primary tooth involves its intra-alveolar development, eruption, completion of root development, and root resorption that eventually facilitates the natural exfoliation of the tooth, harmonizing with the development and eruption of the permanent successor, or in some cases acquiring developmental disturbances.[8,10,20–22] Therefore, it is essential for the pulp therapy of the primary tooth not to interfere with the proper development of the primary tooth and its permanent successor, or with the transition from the primary to the permanent dentition. Furthermore, the selection of the type of therapy must take into consideration the stage of development of the primary tooth and its successor.
Table 38-1 summarizes the average ages for the various stages of dental development. However, the clinician must be aware that these data represent only mean values and significant variations from the timing of the normal developmental stages, requiring that each case be evaluated

TABLE 38-1 Developmental Stages of the Primary and Permanent Dentition[8,21]

Dentition	Arch	Tooth	Enamel completed	Eruption	Root completed	Exfoliation
Primary	Maxillary	Central incisor	1 ½ mo	7 ½ mo	1 ½ yrs	7 yrs
		Lateral incisor	2 ½ mo	9 mo	2 yrs	8 yrs
		Cuspid	9 mo	18 m	3 ¼ yrs	11 yrs
		1st Molar	6 mo	14 mo	2 ½ yrs	9 yrs
		2nd molar	11 mo	24 mo	3 yrs	11 yrs
	Mandible	Central incisor	2 ½ mo	6 mo	1 ½ yrs	6 yrs
		Lateral incisor	3 mo	7 mo	1 ½ yrs	7 yrs
		Cuspid	9 mo	16 mo	3 ¼ yrs	10 yrs
		1st Molar	5 ½ mo	12 mo	2 ¼ yrs	9 yrs
		2nd molar	10 mo	20 mo	3 yrs	10 yrs
Permanent	Maxilla	Central incisor	4–5 yrs	7–8 yrs	10 yrs	
		Lateral incisor	4–5 yrs	8–9 yrs	11 yrs	
		Cuspid	6–7 yrs	11–12 yrs	13–15 yrs	
		1st Premolar	5–6 yrs	10–11 yrs	12–13 yrs	
		2nd Premolar	6–7 yrs	10–12 yrs	12–14 yrs	
		1st Molar	2 ½–3 yrs	6–7 yrs	9–10 yrs	
		2nd Molar	7–8 yrs	12–13 yrs	14–16 yrs	NA
	Mandible	Central incisor	4–5 yrs	6–7 yrs	9 yrs	
		Lateral incisor	4–5 yrs	7–8 yrs	10 yrs	
		Cuspid	6–7 yrs	9–10 yrs	12–14 yrs	
		1st Premolar	5–6 yrs	10–12 yrs	12–13 yrs	
		2nd Premolar	6–7 yrs	11–12 yrs	13–14 yrs	
		1st Molar	2 ½–3 yrs	6–7 yrs	9–10 yrs	
		2nd Molar	7–8 yrs	11–13 yrs	14–15 yrs	

mos = months, yrs = years.

individually.[8,10,21] The dental age, rather than the chronologic age, must be carefully evaluated for teeth present in the mouth, amount of root resorption of the primary tooth, amount of bone between the primary tooth and its permanent successor, and the degree of development of the permanent tooth.

b. *Child's ability to cooperate with dental treatment and/or extent of treatment*:
The risks and benefits involved in advanced behavior guidance techniques, such as pharmacologic sedation or general anesthesia, should be considered in favor of a less conservative approach, diminishing the possibility of failure, and the need for re-treatment. For example, performing an extraction instead of a pulpotomy or a pulpectomy on a tooth with a doubtful prognosis. On the other hand, it is up to the clinician's judgment to evaluate the risk and benefit of an "extra effort" to maintain a pulpally involved primary tooth in specific situations. For example, preventing space loss caused by extraction of a second primary molar before the eruption of the first permanent molars (Figure 38-6A&B), or when a primary molar has no permanent successor.

c. *Anatomy and morphology of the primary teeth*:
Due to the fact that in primary teeth the enamel and dentin are much thinner and the pulp horns much larger than in the permanent teeth, dental caries, restorations and traumatic crown fractures often come close to, or involve, the pulp (Figures 38-5 and 38-8).[8–10] The clinician must be familiar with the root anatomy of primary teeth. While

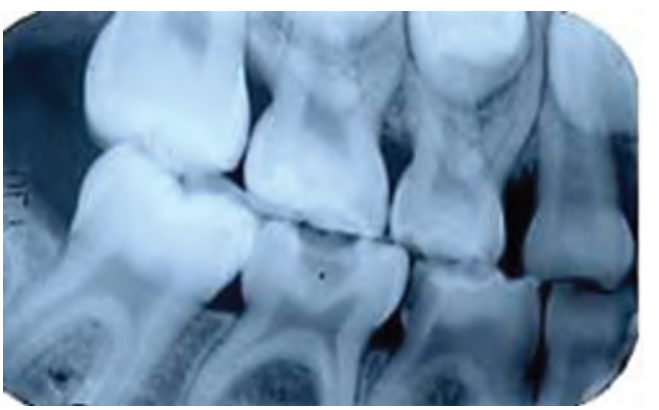


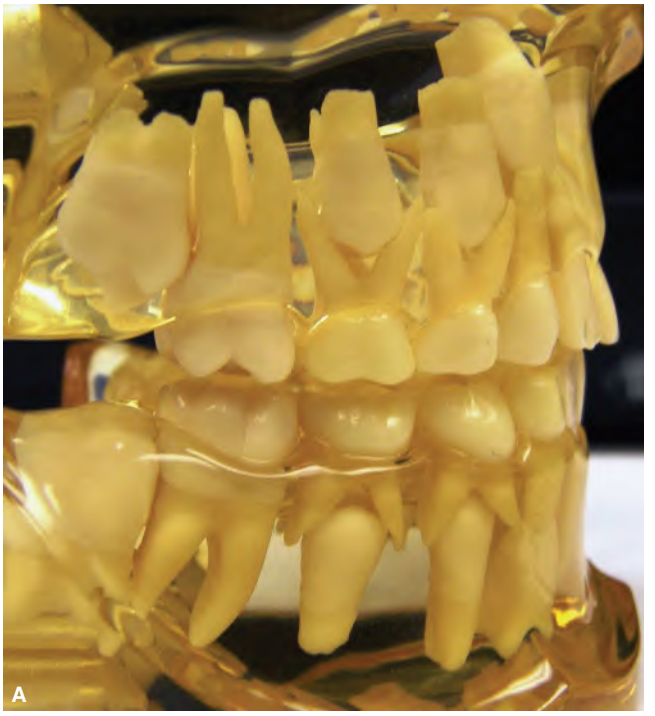
FIGURE 38-8 Bite-wing radiograph showing deep caries in proximity to the pulp in both primary mandibular right molars.

the root canal of anterior primary teeth is normally a large single canal, the molars usually have three root canals; two canals in the mesial root and one in the distal root of mandibular molars, and one canal in each of the three roots in maxillary molars. Two canals have also been found in the mesio-buccal and disto-buccal roots, and some reports have indicated a relatively high incidence of fusion of the distobuccal and palatal root canals.[8–10,23]

The canals are the least complex when the teeth are newly erupted. Narrowing of the canal system takes place with the formation of secondary dentin, and the apical root canal system is modified by the physiological resorption

that has been reported to start immediately after the completion of primary root formation.[8,23] Roots of primary molars have a greater flare than those of permanent teeth, since the primary teeth accommodate the developing succedaneous premolars (Figure 38-9A&B).[8–10,23]

d. *Comprehensive medical history*:
The patient's systemic evaluation is an indispensable component of the oral evaluation and treatment of children, since a systemic condition may, in some cases, be the main factor for choosing the most adequate treatment of pulpally involved primary teeth.[18] In every case, but more emphatically in children with systemic diseases, endodontically treated teeth should be clinically and radiographically monitored periodically for signs of internal resorption (in the case of pulp capping or pulpotomy), or failure due to pulpal, periapical, and/or furcal infections.



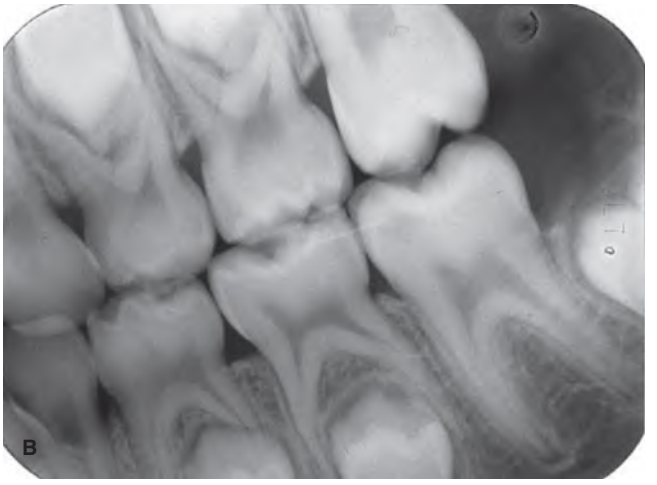


FIGURE 38-9 ***A.*** Transparent model showing the close relationship between the erupted primary teeth and their developing permanent successors. ***B.*** Bite-wing radiograph of the mixed dentition in which the close relationship between the primary teeth and their permanent successors is evident.

The presence and treatment of the following systemic conditions, among others, must be taken in consideration when deciding the adequate treatment of a particular tooth:

1. Cancer disease and therapy
Pediatric patients at all stages of chemotherapy, bone marrow transplantation, and/or radiation therapy require a specially tailored oral/dental treatment program that takes into consideration the systemic impact of the disease and its therapies, and the impact an odontogenic infection would have on their overall health. Immunosuppressed children are at high risk for septicemia that may be caused by oral infections, and require a more radical treatment such as tooth extractions.[24–27]

   Consistent with this rationale, the AAPD guidelines indicate that although there were no studies that address the safety of performing pulp therapy in primary teeth prior to the initiation of chemotherapy and/or radiotherapy, many clinicians choose to provide a more definitive treatment in the form of extraction in order to prevent any residue of oral infection in immune suppressed children, that may lead to pulpal/periapical/furcal infections during immune suppression periods and be life threatening.[25] On the other hand, a recent study by Halperson et al.[24] suggested that pulpotomy treatments in children undergoing therapy for cancer did not increase the risk of these children to develop bacteremia or systemic complications from an oral origin, that there was no significant difference between the success rates of pulpotomy treatments between children receiving therapy for cancer and healthy children and therefore, recommended the re-evaluation of the AAPD guidelines for treating carious teeth involving the pulp in the pediatric oncological patient.[24] Nevertheless, the clinician should be aware that the blood supply may be compromised in patients undergoing radiotherapy, resulting in an impaired repair mechanism following infection and extraction and a significant risk of osteonecrosis.[24] Space maintainers have not been recommended for immunosuppressed patients, increasing their risk of space loss and permanent tooth eruption problems.[24,27] In patients with hemophilia, pulp treatments may be considered more desirable than extractions since it prevents an increased risk for bleeding.[25] Pulp therapy in all chronically ill children may be done only after taking into consideration the systemic treatment, the prognosis of each child's general health, the prognosis of the pulp therapy, and the relevance of the affected tooth.[24–27]

2. Immunosuppression
In addition to children with cancer, those with conditions that increase the possibility of subacute bacterial endocarditis like nephritis, cyclic or chronic neutropenia, or conditions that depress the granulocye and polymorphonuclear counts should not be subjected to the possible development of an acute infection.[26]

3. Congenital heart disease
   There are no recommendations from the AAPD with respect to pulp therapy in a patient with congenital heart disease (CHD) of any severity, but the Guidelines of the Australasian Academy of Pediatric Dentistry state that a vital pulpotomy is contraindicated in these patients.[28] However, this recommendation is empirical and is only based on the rationale to avoid any potential negative sequel from an unsuccessful pulp treatment in a patient with CHD. It is notable that a survey done in 2013 indicated that AAPD members preferred extraction over pulp treatment for patients with irreversible pulpitis suffering from CHD.[29]
4. Diabetes
   A case report indicates that odontogenic infections may be very dangerous in children with diabetes or in those patients with immune diseases since they may lead to ketoacidosis.[30]

e. *Chief complaint and history of pain*:
Subjective evaluation of the affected oral area of the child is based on the current symptoms/chief complaint and by questioning the child and the parent on the location, intensity, duration, stimulus, relief, and spontaneity of the complaint.[18] Subjective symptoms are important factors when establishing a diagnosis, but since young children may be unable to communicate their symptoms, or have a limited ability to recall their pain history, there is a need to rely on the parents to get a more accurate history. In addition, the clinician must take into account that it is not uncommon for a pulp to degenerate to the point of abscess, or developing a pulp polyp, without the child's recalling any discomfort (Figure 38-10A&B).[26]

The information regarding a correlation between histological inflammatory changes within the pulp and reported pain history is inconsistent.[31–36] While several reports have failed to prove a strong correlation between the two,[31–35] most clinicians agree with the concept that when pain is stimulated by an external irritant and resolves when the noxious stimulus is removed, it indicates that the pulp is reversibly inflamed, and that spontaneous pain, throbbing pain, or pain that continues long after the causative factor has been removed, or that keeps the patient awake at night usually signifies an irreversible pulpal inflammation.[26,36] In an interesting study, Guthrie et al.[36] showed an association between a spontaneous toothache and extensive degenerative changes extending into the root canals, as reflected in an hemogram of the exposed pulp.[36] It is also of general belief that pain upon biting and the need for analgesics are indicative of an irreversibly inflamed pulp.[18,36] However, pain upon biting may also be elicited by food impaction into a chronic periodontal defect adjacent to proximal caries; the periodontal defect may heal after tooth restoration with no need for pulp treatment.[37]

f. *Extraoral clinical examination*:
Extraoral patient evaluation should begin by observing the patient when entering the room, and his/her demeanor and extent of discomfort can be evaluated while reviewing the medical information with the parents. A visual examination and palpation of the head and neck are important in helping to determine whether swelling is present and if there is any lymphadenopathy. Odontogenic infections rarely spread beyond the periapical space, however, on some occasions the infection may spread into contiguous anatomic areas through bone, fascial, or muscular barriers.[38]

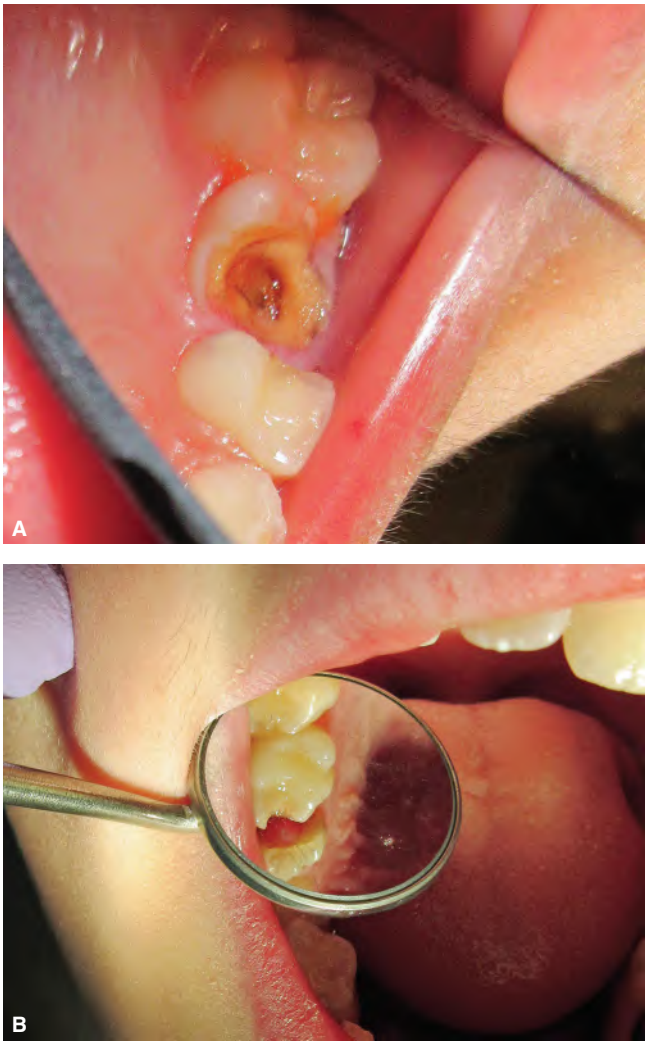


FIGURE 38-10 ***A.*** Clinical (mirror) picture of a primary maxillary left second molar with deep caries and pulp exposure. The child did not report any pain. ***B.*** Clinical (mirror) picture of a primary maxillary right first molar with deep caries and a pulp polip. The child did not report any pain.

Cellulitis is associated with significant morbidity in children. Oral health providers may encounter children with odontogenic infections that may progress and develop systemic signs and symptoms including fever, malaise, tachycardia, dehydration, periorbital and orbital cellulitis (Figure 38-4), Ludwig's angina, and intracranial abscesses.[39–42] These maladies have the potential to lead to death as in the tragic case of Deamonte Driver (reported in The Washington Post, February 28, 2007), a 12-year-old boy who died from a brain infection of odontogenic origin. These signs and symptoms are even more

dangerous in children with chronic or acute systemic diseases and therefore, extraction of the involved tooth after proper medication with antibiotics, rather than pulp therapy, should be the treatment of choice.[26]

g. *Intraoral clinical examination*:
Intraoral clinical examination may begin with the child pointing with his/her finger to the tooth/area where he/she feels the pain and by that leads the clinician to the offending oral lesion. Accordingly, one should avoid misdiagnosis since the offending tooth may not always be the one with the largest carious lesion, or the pain may not be from dental origin as in the case of an aphthous ulcer. Signs that may be associated with pulp pathology include swelling of the vestibulum, parulis, and sinus tract (Figures 38-3 and 38-11).

The intraoral clinical examination should include inspection for the presence of carious lesions, tooth discoloration, palpation of the tissues surrounding the tooth to evaluate sensitivity to touch, tissue fluctuation, abnormal mobility (normal or increased accompanied by pain when tested), swelling and presence of an exudate from the marginal sulcus or from a sinus tract, and/or swelling of the attached gingiva or oral mucosa.[26] Sensitivity to percussion or pressure in a primary tooth with a deep caries lesion may suggest some degree of pulpal disease, however, the possibility of chronic or acute periodontal disease should also be taken in consideration.[18,37,43,44]

Accounting for the behavioral aspects involved in pediatric dentistry, it is possible to perform a "kind" percussion test in primary teeth by tapping on the tooth with a finger instead of with the back of a dental mirror.[44] Most importantly, during the intraoral clinical examination, comparison of both sides of the mouth should always be done.

Although infrequent, periodontal abscesses in the primary dentition are possible and should be differentiated from pulp originated abscesses by the clinical and radiographic signs. A periodontal abscess may manifest with pus draining from the gingival sulcus rather than producing pronounced buccal swelling, and a periodontal abscess may not produce an interadicular radiolucency (Figure 38-12A&B).[43]

h. *Radiographic examination*:
Intra-oral radiographs are essential for the overall evaluation of the condition of the primary dentition. These provide unique information about the extent of the carious lesion, the proximity of large restorations to the pulp, presence of periapical or interadicular radiolucencies, degree of pathological or physiological root resorption, amount of bone between the primary and its permanent successor, and the presence and degree of development of the permanent successor.

Pulp inflammation and necrosis in primary teeth may lead to periapical radiolucencies in the anterior teeth (Figure 38-13), interadicular, or interadicular and periapical radiolucencies in the primary molars (Figures 38-14, 38-15 and 38-16A&B),[18,26,44,45] atypical root resorption (Figure 38-13), and in some cases, damage to the permanent successor (Figures 38-15 and 38-16A&B). The finding that pulp necrosis in primary molars usually manifests in the radiograph at the interadicular area (Figure 38-14) and not

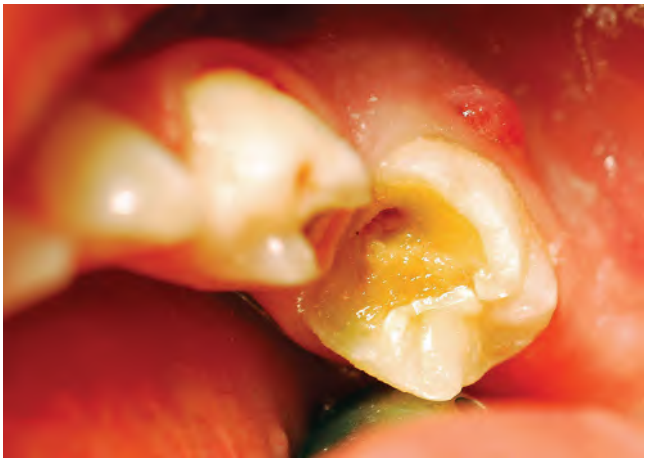
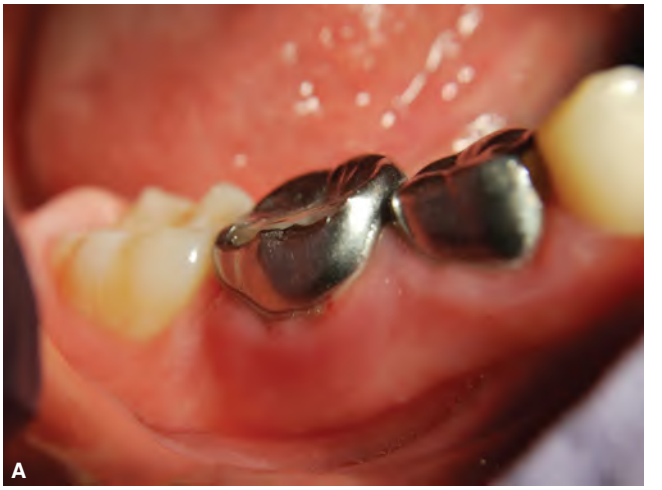


FIGURE 38-11 Clinical picture showing a primary maxillary left second molar with deep caries, pulp exposure and a buccal parulis.



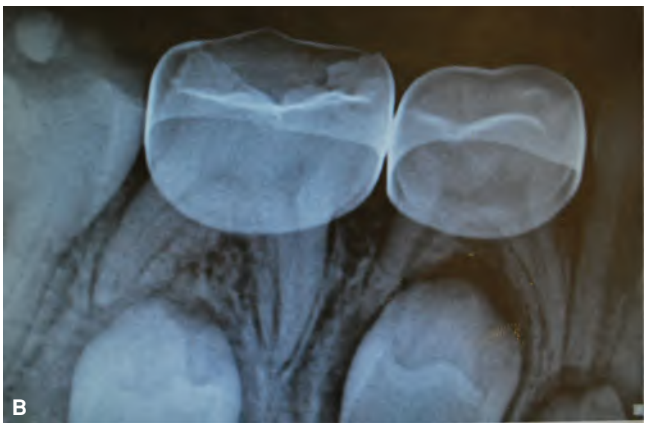


FIGURE 38-12 ***A.*** Clinical picture of a child who developed a periodontal abscess adjacent to an ill-fitted stainless steel crown in the primary mandibular right second molar. Note the purulent exudate coming out between the marginal gingiva and the crown, and the gingival inflammation. ***B.*** Periapical radiograph of the involved tooth shows no evidence of bone pathosis.

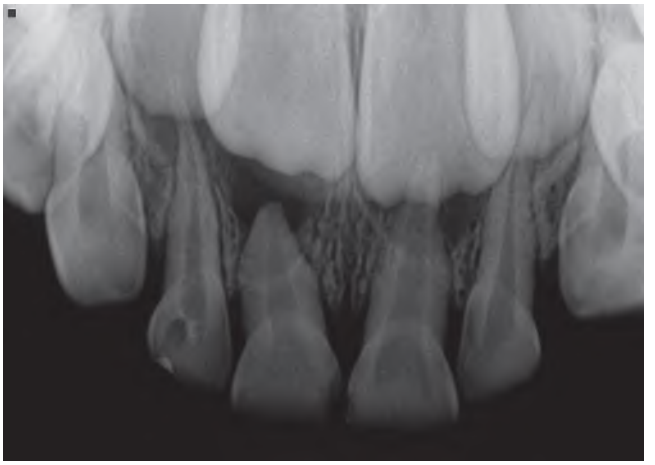


FIGURE 38-13 Anterior maxillary periapical radiograph showing a periapical radiolucency and atypical root resorption associated with the primary right central incisor.

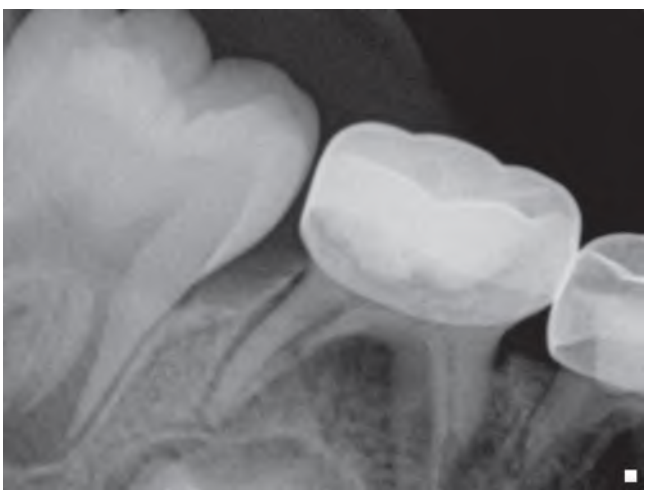


FIGURE 38-14 Periapical radiograph of a primary mandibular right second molar that developed an interadicular radiolucent area subsequent to being restored with a stainless steel crown.

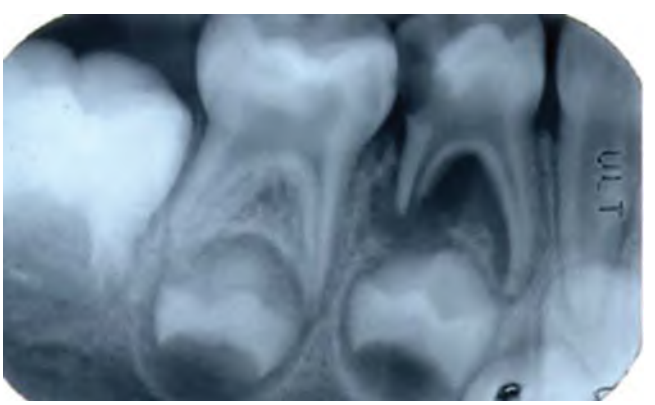
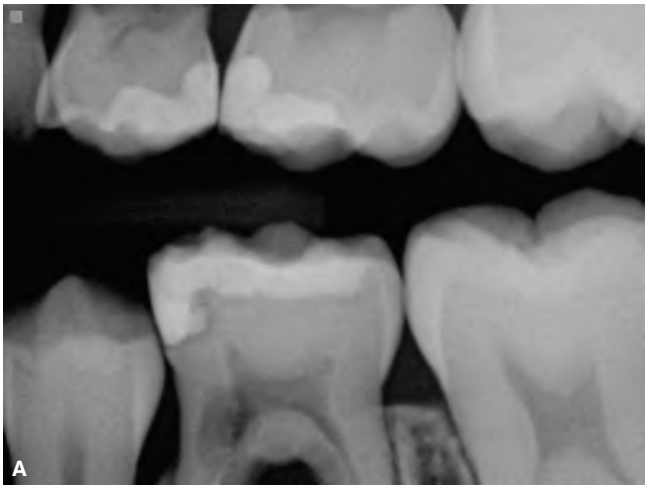


FIGURE 38-15 Periapical radiograph showing an interadicular and periapical radiolucent area associated with the primary mandibular right first molar. Note that the radiolucent area has already included the bone compacta surrounding the follicle of the permanent successor.



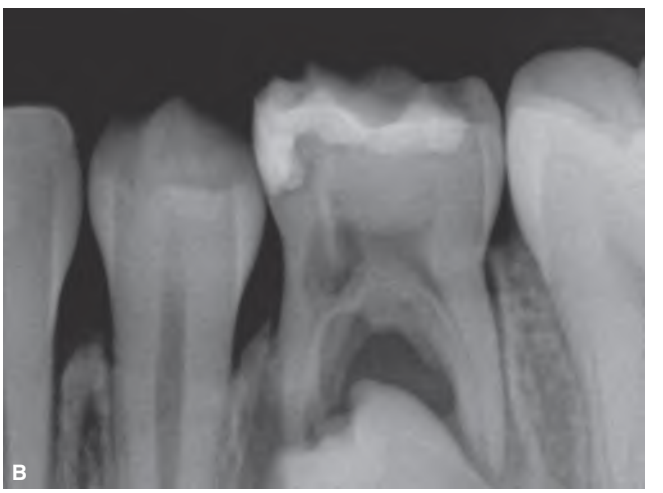


FIGURE 38-16 ***A.*** Bitewing radiograph showing internal resorption and an interadicular radiolucent area associated with the primary mandibular left second molar. ***B.*** Periapical radiograph of the same tooth. Note the large interadicular radiolucent area and the displaced permanent successor tooth.

at the periapical area is related to the common presence of furcal canals in primary molars.[45]

Pulp inflammation may lead to internal dentin resorption at the pulp chamber or root canal (Figures 38-16A&B and 38-17).[36] Due to the thinness of the roots of primary teeth, internal resorption may easily result in root perforation and subsequent alveolar bone resorption necessitating extraction to prevent damage to the alveolar bone and the permanent successor (Figure 38-16A&B).[45,46] Evidence of pulp inflammation in primary teeth may also include obliteration of the pulp cavity with dentin (Figure 38-17).

i. *Pulp testing:*
Thermal and electrical pulp testing are seldom used in the primary dentition because of unreliable results. For these tests to be effective, the pulp must have a sufficient number of mature neurons. Primary teeth are not fully innervated with A-type myelinated axons, the neural components that are responsible for the sensation of pulpal pain, as physiologic resorption begins shortly after

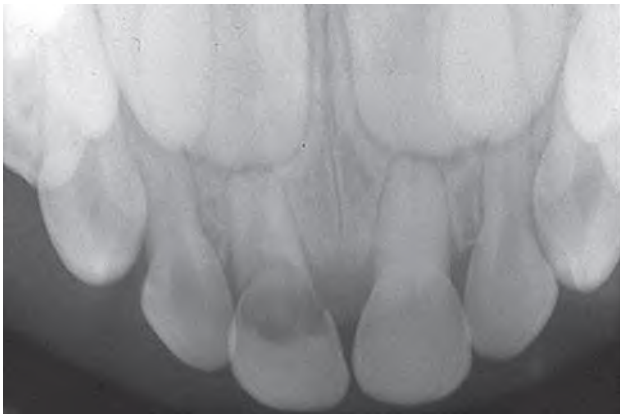


FIGURE 38-17 Periapical radiograph of primary maxillary incisor area after trauma in the past. While the primary left central incisor shows obliteration of the pulp cavity, the primary right central incisor shows a large area of internal resorption.

the root is fully developed.[47] This can be compared to the finding that immature bicuspids are less responsive to stimuli due to a reduced number of pain receptors and therefore, more likely to produce a false negative response from electric and thermal tests.[47]

Thermal and electric pulp testing in primary teeth may only indicate the presence of necrosis, and do not reflect the degree of inflammation of the pulp. It is also possible to get a positive response from a necrotic pulp in which the content of the pulp canal is liquid.[18] In addition, the child's apprehension or inability to comprehend the test may lead to erroneous evaluation.[18] It may, therefore, be summarized that despite a study that reported that electric pulp testing may be reliable for the diagnosis of the pulp status in primary teeth,[48] pulp testing is mostly not recommended in primary teeth and eliciting pain in a small child may trigger compliance problems.

In attempts to eliminate the problem of low reliability of children in the subjective evaluation of pain caused by electric or thermal pulp testing, the objective evaluation of the pulp condition of primary teeth has been tested with pulse oximetry or laser Doppler flowmeter.[49–52] Pulse oximetry estimates the arterial hemoglobin saturation indicating the presence of blood flow in the pulp.[49] A study on primary and immature permanent teeth concluded that pulse oximetry is applicable to primary and immature permanent teeth where patient cooperation and the incomplete innervation reduces the effectiveness and reliability of thermal and electric pulp testing methods.[50] Munshi et al.[49] reported zero pulse oximeter measurements of 10 known non-vital teeth, immature vital permanent teeth recorded average values of 98% and vital primary teeth recorded average values of 93%. They concluded that pulse oximeter has the ability to differentiate between vital and non-vital teeth.[49] Still, the lack of probes that are optimized for dentistry and the insulation of the pulp by the surrounding hard tissues are obstacles to the pulse detection and the use of pulse oximetry in clinical dentistry.[49]

A study of laser Doppler flowmetry, designed to measure the velocity of red blood cells in capillaries, in 119 primary incisor teeth of 32 children between the ages 4 and 10 years old concluded that laser Doppler flowmeter may be considered a noninvasive, nonpainful, direct, objective, and acceptable method of intrapulpal blood flow in primary incisors.[51] It is important to consider that this study was done on primary incisors with at least two thirds of their root, in children under general anesthesia, before and after extraction or before and after a pulpectomy, and was based on the comparison between the dental flowmeter output and the electrocardiogram signal.[51] However, the reliability and the price of the laser Doppler flowmeter have impeded its routine use.[52]

j. *Operative diagnosis:*

In addition to the initial clinical and radiographic examination, the diagnosis of the pulp condition is defined during treatment by direct evaluation of the exposed pulp tissue. For example, the need for a pulpectomy, or extraction, is indicated by the finding of a necrotic pulp or by a dark color and/or profuse bleeding and the inability to obtain hemostasis during a pulpotomy.[26]

The key to successful pulp therapy is accurate diagnosis. The clinician must be mindful to the fact that the patient's systemic and oral histories, clinical evaluation, and radiographic findings must be integrated in order to be able to achieve a precise pulpal diagnosis and hence the most appropriate treatment. Pulpal and periapical diagnosis should not rely solely on a single diagnostic test or one piece of information. Inaccurate diagnosis of pulpal status may result in inappropriate treatment, questionable prognosis, and in the case of the primary dentition, potential damage to the developing dentition (Figures 38-15, 38-16B, and 38-18).

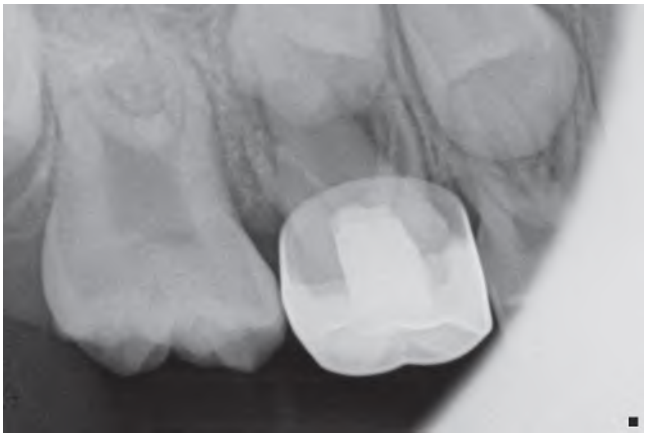


FIGURE 38-18 Periapical radiograph of a primary maxillary right second molar in which internal resorption and an interadicular radiolucent area developed after a pulpotomy. Note that the bone compacta surrounding the crown of the permanent successor has been lost.

PULP THERAPY FOR PRIMARY TEETH

A primary objective of pulp therapy is to maintain the integrity and health of teeth and their supporting tissues, and in the case of the primary dentition allow for the normal exfoliation of the primary teeth and eruption of the succedaneous teeth.[18,26,44] The dentin-pulp complex of the primary teeth has a similar response to dental caries as the permanent teeth, and pulpally involved primary teeth are amenable to comparable pulp therapy modalities as permanent teeth, with procedures such as direct or indirect pulp capping, pulpotomy and pulpectomy, depending on the extent of the caries, the pulp condition, and tooth restorability.[18,26,44] The indications, objectives, and type of pulpal therapy depend on whether the pulp is normal, reversibly inflamed (reversible pulpitis) and thus capable of healing, irreversibly inflamed (irreversible pulpitis) and thus incapable of healing, or necrotic.[18,26,44]

Dental caries involving more than half the dentine thickness in primary teeth is associated with variable degrees of reversible pulpal inflammation. The objective of pulp treatment should be to maintain the vitality of the pulp of a tooth affected by caries, or other causes affecting the pulp, such as traumatic injuries.[53–55] Therefore, a non-aggressive approach of caries removal in primary teeth should be taken in consideration with the aim of avoiding pulp exposures, maintaining the vitality of the pulp, and allowing pulp healing.[53–57] Invasion of the dentinal tubules by caries pathogens triggers defense and repair mechanisms within the dentin-pulp complex to contain pathogen spread and reduce dental pulp tissue damage. Odontoblasts situated beneath infected dentinal tubules secrete reactionary dentin, accompanied by neuronal sprouting and immune cell invasion into the reactory collagen matrix.[58] As the result of this process, the pulp may recede and a pulp exposure may be prevented (Figure 38-19). However, this is not always the case and the dental pulp may eventually become exposed by caries (Figures 38-10A&B, 38-11, 38-15, and 38-20).

Vital pulp therapies are those procedures that allow the affected pulp to heal, maintain the vitality of the pulp, and protect the pulp from an exposure. It can be defined as any restorative dental treatment that is focused on minimizing trauma to the dental pulp.[18,59] The objectives of vital pulp therapy are to eradicate the potential for infection, maintain the tooth in a quiescent state, preserve space for underlying permanent teeth, and retain the primary tooth when the permanent tooth is congenitally absent.[18,59] These conservative procedures, aimed at preserving pulp vitality, can only be effective when the status of the pulp is accurately assessed as normal or reversibly inflamed.[60] Non-vital pulp therapy for the primary dentition are performed when the radicular pulp tissue is diagnosed with irreversible pulpitis or necrotic.[18,59]

Dental Materials Commonly Used in Vital Pulp Therapies in Primary Teeth

Dental materials used for vital pulp therapy ideally should have the following properties: a) inhibit bacterial growth, b) induce mineralization, c) establish a tight bacterial seal, d) resist long-term bacterial leakage, and e) stimulate the remaining pulp tissue to return to a healthy state while promoting the formation of dentin.[61] While some materials, or techniques, are limited to one specific pulp treatment (e.g., formocresol and ferric sulfate are limited to be used in pulpotomies), some materials may be used for various treatments such as indirect or direct pulp capping, and pulpotomy. These include calcium hydroxide ($Ca(OH)_2$), mineral trioxide aggregate (MTA) and Biodentine™. These materials are used in vital pulp therapy procedures that aim to maintain the vitality of the pulp.

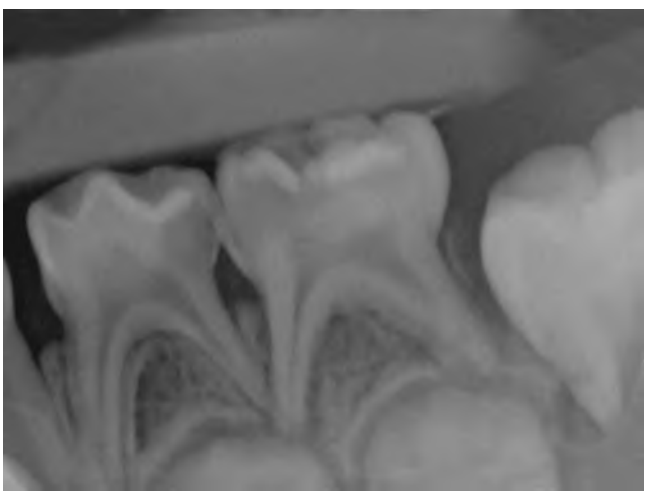


FIGURE 38-19 Periapical radiograph of primary mandibular left molars with deep caries. Note the recession of the distal portion of the pulp in the primary first molar as a defensive process from the carious injury.

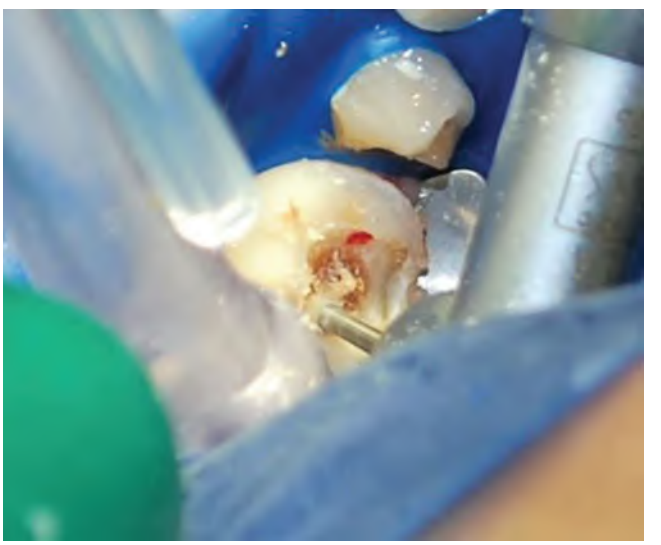


FIGURE 38-20 Primary second molar in which a carious pulp exposure became evident during removal of carious dentine.

Calcium Hydroxide

$Ca(OH)_2$ was introduced to the dental profession in 1921 and was considered the "gold standard" of pulp capping materials for several decades.[62] It is a white odorless powder classified as a strong base, its main actions come from the ionic dissociation of Ca^{2+} and OH^- ions and their effect on vital tissues. The high pH of calcium hydroxide provides antimicrobial activity and encourages tissue repair by promoting tertiary dentin secretion.[63] The therapeutic effect of calcium hydroxide may also be due to its extraction of growth factors from the dentin matrix. Once released, these growth factors may play a key role in tertiary dentinogenesis.[64]

Although $Ca(OH)_2$ has been considered the gold standard for pulp capping with the advent of new research and materials, the disadvantages of calcium hydroxide are becoming evident. Calcium hydroxide degrades over time, has a low compressive strength, and has poor sealing properties.[65] Multiple tunnel defects were found in 89% of dentin bridges after calcium hydroxide pulp capping, and 41% of these bridges were associated with recurring pulpal inflammation or necrosis, and with the presence of inflammatory cells and stained bacterial profiles.[66] Tunnel defects in dentinal bridges under calcium hydroxide can act as pathways for bacterial microleakage.[67] Microbial microleakage can impede the formation of the dentinal bridge by odontoblast-like cells.

Glass Ionomer Cements

Glass ionomer cements (GICs) were introduced to dentistry in the early 1970s. Glass ionomer consist primarily of alumina, silica, and polyalkenoic acid and are self-curing materials. GICs form chemical bonds to tooth structure, are biocompatible, and release fluoride ions for uptake by enamel and dentin.[68] Because of glass ionomer's ability to chemically bond to tooth structure, it can prevent the diffusion of potentially toxic materials through dentin to the pulp. GIC provides an excellent seal against microorganisms and shows good biocompatibility when used in close approximation but not in direct contact with the pulp.[69]

Resin-modified glass ionomer cement (RMGIC) materials such as Vitremer (3M ESPE) and Fuji II LC cement contain glass-ionomer powder, to which a light-cure resin is added. RMGIC not only have improved physical characteristics, but the photopolymerizable resin component reduces initial hardening time substantially. Conversely, RMGICs are more cytotoxic to the pulp cells than conventional GICs due to the presence of unpolymerized monomers, and should not be applied directly to the pulp tissue.[70] Clinical evidence of RMGI promoting reparative dentin has not been reported. However, Farooq et al.[71] reported 93% success of indirect pulp therapy IPT over an average of four years with the use of a RMGI as a liner or base without the use of $Ca(OH)_2$.[71]

Mineral Trioxide Aggregate

Mineral trioxide aggregate (MTA) is a fine hydrophilic powder. It consists of tricalcium silicate, tricalicum aluminate, tricalcium oxide, silicate oxide, and bismuth oxide.[72] Studies analyzing the constituents of both gray and white ProRoot® MTA have conclusively shown that both materials are similar to Portland cement but with bismuth oxide added, presumably to make the material radiopaque.[73] Each pack of ProRoot® MTA comes with a pre-measured unit dose of water for convenience in mixing. In the presence of moisture, MTA sets into a hard mass by forming $Ca(OH)_2$ and silicate hydrate gel.[72]

MTA was originally developed to seal communications between the dental root and the surrounding surfaces.[74] Over the years, further research of the material resulted in its application in various clinical situations,[73] and MTA has been used as a material for vital pulp therapy in both primary and permanent teeth due to its favorable properties.[75] The rationale behind the use of MTA is that it prevents micro-leakage, is biocompatible, promotes regeneration of original tissues when it is placed in contact with the dental pulp or peri-radicular tissues, and has not been found to induce internal resorption that has been observed in teeth treated with some other medicaments.[73] MTA has a pH of 10.2 immediately after mixing that increases to 12.5 after 3 hours of setting. It is similar to the pH of calcium hydroxide. MTA does not show signs of solubility contributing to the good seal achieved with MTA and prevention of bacterial contamination of the pulp.[76,77] MTA induces a dentin bridge formation that is faster and with better integrity than that formed by calcium hydroxide. This is achieved with more favorable pulpal tissue reaction as there is less pulpal inflammation, hyperemia, and necrosis of the pulp than has been associated with the use of calcium hydroxide.[78]

Since the set time for MTA is several hours, the manufacturer recommends the placement of the final restoration at a second appointment to assure the set of the material. In the first visit, a wet cotton pellet is applied over the MTA, and the tooth is temporized. In the second visit, the cotton pellet is removed, and the permanent restoration applied after the MTA has sufficiently hardened. However, it would be beneficial for patients and dentists alike if the final adhesive restoration could be placed over MTA during the same visit, especially in pediatric dentistry. Eid et al.[79] concluded that resin-modified GIC can be successfully applied on freshly mixed MTA in a single visit with no expected adverse reactions between the two materials.

Interestingly, an in vivo study comparing the clinical and microbiological outcomes of MTA, medical Portland cement, and calcium hydroxide as a liner for protecting the dentin–pulp complex of permanent and primary teeth, subjected to two-step indirect pulp therapy found no difference in outcome.[80] This finding demonstrates the potential of indirect pulp treatment and that a well-sealed restoration is fundamental for treatment success. Incidentally, MTA was intended for direct capping of pulp exposures and the manufacturer does not advocate its use for indirect pulp capping.

The original MTA was gray in color but in 2002, a white MTA (WMTA) was marketed due to esthetic concerns of

gray MTA (GMTA) staining tooth structure. The WMTA contains less iron, aluminum, and magnesium oxides than its GMTA counterpart. GMTA has been investigated more than the white MTA, and while some reports suggest that GMTA may invoke a more desirable biologic response than WMTA, existing reports as a whole are insufficient.[81] WMTA, however, may still cause tooth discoloration, most likely due to reaction of MTA with blood.[82]

Biodentine™

Biodentine™ is a tricalcium silicate-based cement that was introduced to the market in 2009 as a dentine replacement material and has gained popularity in recent years as a biocompatible and bioactive dentin substitute.[83] Biodentine™ is similar to MTA, formulated using MTA-based cement technology while improving some of the properties of MTA, such as physical properties and handling.[83] Biodentine™ is a powder and liquid system that, when combined, forms a gel structure with an initial set time of 9-12 minutes but with a longer final setting time.[83,84] Biodentine™ is utilized for pulp capping, repair of endodontic root perforations, apexification and retrograde fillings, and has also been suggested for use in pulpotomy treatments of primary teeth.[59,83,85]

Numerous studies have shown Biodentine to have favorable properties, including biocompatibility, ease of handling, negative solubility, and high compressive strength, while providing a good seal without any dentin conditioning.[84,85] When Biodentine™ was used for direct pulp capping, the tertiary dentin was well formed without tunnel defects, and without evidence of adjacent pulpal inflammation.[86] Biodentine™ has been shown to form hydroxyapatite on its surface as a result of contact with tissue fluids.[84] This property produces a material that is not soluble and is dimensionally stable. Although more studies are needed, Biodentine™ holds promise for use in clinical procedures as a biocompatible and easily handled product with a short setting time.[83]

VITAL PULP THERAPY FOR THE PRIMARY DENTITION

Protective Liner

A protective liner (PL) is a thinly applied flowable material, placed on the pulpal surface of a deep cavity preparation to cover the exposed dentin tubules after complete caries removal (Figure 38-21). Its purpose is to act as a defensive barrier between the restorative material and the pulp, minimize postoperative sensitivity, preserve the tooth's vitality in teeth with a normal pulp, promote tissue healing and tertiary dentin formation, and minimize microbial microleakage.[18,59] Though the materials used as cavity liners have no direct contact with the pulp tissue, these materials should possess the same qualities as those used for direct pulp caps and promote healing, be biocompatible, induce dentin formation, minimize bacterial leakage, and inhibit bacterial growth.[61] The PL should therefore seal the dentin and prevent microorganisms and their by-products from contaminating the pulp.[18] At the discretion of the clinician, the

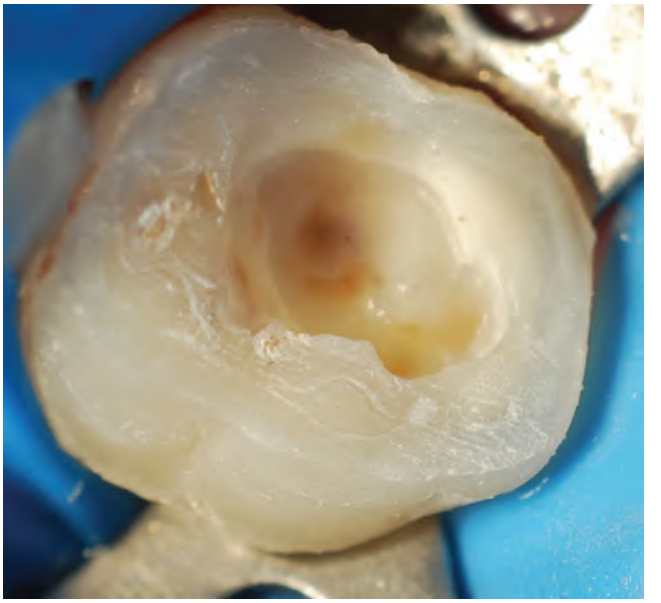


FIGURE 38-21 Primary mandibular left second molar after complete deep carious removal and cavity preparation for a stainless steel crown, prior to placement of a protective liner.

PL may be $Ca(OH)_2$, a dentin bonding agent, or a glass ionomer cement.[18,59,62,87] Adverse post-treatment clinical signs or symptoms such as sensitivity, pain, or swelling should not occur.[18,59]

A recent review of current restorative concepts of pulp protection revealed that the main function of liners is to protect the pulp from microbial penetration along the tooth restoration interface, as opposed to the commonly held belief that the liners protected the pulp from the restorative materials.[87] Other studies have also found that the presence, or absence, of liners has no effect on postoperative sensitivity or pulpal complications.[88,89] While the use of a PL is similar to indirect pulp capping, PL includes the complete removal of caries, while the indirect pulp capping does not involve removing the deepest part of the carious lesion.[18,59]

Indirect Pulp Capping

Bjørndal[19] quoted Dr. GV Black: "it is better to expose the pulp of a tooth than to leave it covered only with softened dentin." Indeed, for many years the complete removal of caries and subsequent restoration of the tooth was the standard method of treating carious lesions in the primary dentition.[19] However, this approach may easily result in pulp exposures in primary teeth due to their anatomical characteristics.[8–10] Yet, in the same publication, Bjørndal indicated that a different interpretation of the very same clinical situation may result in a completely different treatment approach, based on the fact that it is desirable to avoid pulp exposures and facilitate pulp healing.[19]

Indirect pulp capping (IPC), also named indirect pulp therapy, is the intentional incomplete removal of carious dentin in order to avoid a pulp tissue exposure, and treatment of the decay process with a biocompatible material and adhesive restorations that provide a good seal (Figure 38-22).[18,56,57,59] IPC is based on the following assumptions:[18,56–59,90–92]

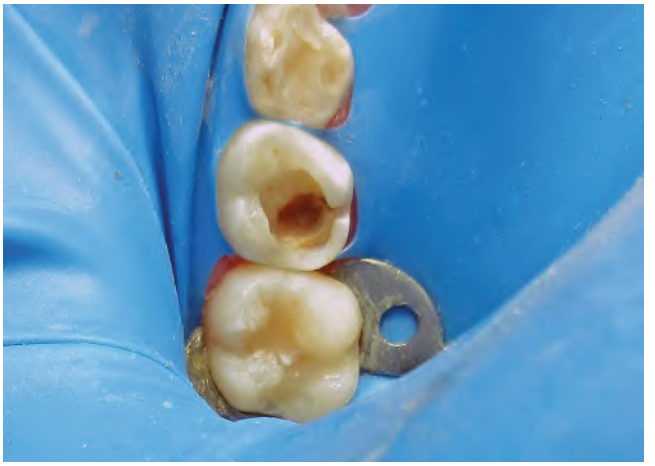


FIGURE 38-22 Primary maxillary right second molar after removal of the softened and infected dentin. The remaining partially demineralized stained carious dentin will be covered with an indirect pulp capping biocompatible material to prevent pulp exposure.

a) the presence of microorganisms in dentin itself is not a determining factor of caries progression but dependent on the metabolic activity of the biofilm present on the dental surface. The demineralization of enamel and dentin reflects this metabolic activity; b) changes in the biofilm caused by the IPC tend to arrest the sealed carious lesion and stimulate pulp healing and repair. Lin and Langeland[93] showed that when no pulp exposure occurred, the pulp's repair capacity was excellent, but after exposure, it was questionable and unpredictable. In addition, the risk of displacing infected dentin chips into the pulp with total pulp excavation increases the risk of pulpal inflammatory breakdown.[94]

Clinical procedures involving incomplete caries removal are also based on the concept that carious dentine lesions are composed of two distinct layers.[95] The outer layer is highly infected with cariogenic bacteria, structurally disarranged, and presents no mechanical properties. It is considered irreversibly damaged and its complete removal is recommended. The inner layer, or affected dentin, is only partially demineralized, is able to remineralize under a well-placed and sealed restoration, and should be maintained to avoid exposure of the pulp.[95–99] The caries close to the pulp is then covered with a biocompatible liner material such as a dentin bonding agent, RMGIC, $Ca(OH)_2$, zinc oxide eugenol (ZOE), GIC, MTA or Biodentine™.[18,56,57,90–92] In any case, it is most important to completely remove the decay from the dentin-enamel junction, along the axial walls of the cavity, as this has shown to improve the integrity and marginal seal of the restoration.[99]

A systematic review of pulp preservation studies through 2008 found that most investigations used calcium hydroxide for indirect pulp capping, yet, other materials were also found to reduce cariogenic microorganisms and promote remineralization as effectively as calcium hydroxide.[91] When $Ca(OH)_2$ is utilized, a GIC or reinforced zinc oxide/eugenol material should be placed over it to provide a seal against microleakage, since $Ca(OH)_2$ has a high solubility, poor seal, and low comprehensive strength.[18] Currently, it appears that the use of GIC for IPC is more popular than $Ca(OH)_2$ since GIC has the additional advantage of inhibitory activity against cariogenic microorganisms,[18] and the ability to reduce permeability and sensitivity.[100] Other studies have indicated that the presence or absence of protective liners has no effect on postoperative sensitivity or pulpal complications.[88,89]

GIC has the ability to infiltrate the smear layer with resin, during placement, and to penetrate dentin tubules by forming resin tags in acid etched dentin. The IPC pulp healing effect promotes remineralization of the demineralized remaining dentin.[101,102] Particularly in pediatric dentistry, the use of RMGIC is advantageous since these materials may act as a base and restorative material. This is due to their ease of handling, advantageous mechanical characteristics, compared to conventional glass ionomer cements, and their high success rate when used as IPC materials.[101] The question of whether different materials used for IPC produce different degrees of remineralization on the remaining dentin under an IPC has been addressed by comparing the dentin under IPC treatments in primary molars accomplished with Ca(OH)2, RMGIC, gutta-percha, or a self-etching primer. Regardless of the protective material used, there was mineral gain by the affected dentin after IPC.[101–103]

As our knowledge of caries pathogenesis and pulpal response improves, it is becoming possible to plan the treatment of carious primary molars with a more evidence-based approach. A recent systematic review concluded that incomplete caries removal was advantageous, particularly in teeth with caries close to the pulp. This treatment was found to significantly reduce the risks of pulpal exposure and postoperative pulpal symptoms, compared with complete caries excavation.[104] A 2013 update of a 2006 systematic review of complete or ultraconservative removal of decayed tissue concluded that "the current evidence demonstrates that in symptomless, vital, carious primary or permanent teeth, stepwise and partial excavation, reduced the risk of pulp exposure".[105]

It has been considered that there is insufficient evidence to determine whether it is necessary to re-enter and excavate further in the stepwise excavation technique, however, the studies on teeth in which the cavity was not re-entered, reported no adverse consequences.[105] Furthermore, the findings that there is remineralization of the remaining dentin, based on an increase of calcium and phosphorous, that few viable microorganisms remain in the deep dentinal layers after the tooth has been properly sealed, and that RMGICs can simultaneously serve as a base and restorative material with high clinical, radiographic, ultrastructural and microbiological success rates, suggest that there is no need for cavity reopening with the purpose of removing the remaining carious dentin after an IPC.[56,100–102] Recognition of the success of IPC[71,105–108] is reflected in a 2007 survey of primary tooth pulp therapy that found that 83% of American dental schools use, or teach indirect pulp capping as opposed to 70% that used or taught that procedure in 1997.[109] Furthermore, IPC

has been shown to have a higher success rate than pulpotomy in long-term studies. Therefore, it has been suggested that IPC is preferable to pulpotomy when the pulp is normal or has a diagnosis of reversible pulpitis.[7,18,106,108]

Atraumatic Restorative Technique and Interim Therapeutic Restoration

Variations of IPC are the atraumatic restorative technique (ART) and the interim therapeutic restoration (ITR) as defined by the World Health Organization (WHO) and the AAPD respectively. ART and IRT involve the partial removal of caries without anesthetizing the tooth and placing a glass ionomer restoration.[110,111] ART has been endorsed by the WHO, as a means of restoring and preventing caries in populations with little access to traditional dental care.[110] ITR has been recommended by the AAPD when circumstances do not permit traditional cavity preparation or placement of traditional dental restorations, or when caries control is necessary prior to placement of definitive restorations, as part of the comprehensive dental care in the dental home.[111] The reported success rates of ATR depend on the material used for restoration, length of the follow-up studies, and on the location and extent of the glass ionomer restorations; some studies indicate success rates from 57.1% to 100%.[112–114]

The success of the ART is also dependent on the antimicrobial properties of freshly prepared restorative materials, since restorative materials used in ART have a potent effect against cariogenic microorganisms and the prevention of secondary caries.[115] It has been demonstrated that the microhardness of dentin under ART increases over time, but does not reach the degree of hardness of healthy dentin.[116,117] A recent study investigated the effect of a GIC as a liner, over infected unexcavated dentin, after 60 days, and at 10 to 15 months. After 60 days, the dentin already exhibited a better organization, fewer bacteria, and signs of remineralization.[118] The study concluded that: a) the lining material to be placed on infected dentin is not fundamental for caries arrestment; b) when the cavity is properly sealed, carious dentin tends to reorganize within 60 days, and the remineralization process still continues for a longer period of time. Moreover, it has been considered that with ITR there is no conclusive evidence indicating that it is necessary to re-enter the tooth to remove the residual caries, as long as the tooth remains sealed from additional microbial contamination. Evidence of the success of ITR should include no radiographic evidence of pathological internal or external resorption or other pathologic changes, and there should be no harm to the permanent successor.[111,112]

The Hall technique, developed by Dr. Nora Hall in the 1980's, is a conservative treatment alternative for carious primary molars.[119] This controversial non-invasive technique involves the use of SSCs with glass ionomer cement to seal over caries lesions in primary molars without administration of local anesthetic, no caries removal, and no crown preparation. This technique showed a similar success rate for SSCs placed using the traditional technique that includes administering local anesthetic, caries removal, and crown preparation.[119]

Direct Pulp Capping

Direct pulp capping (DPC) is the treatment of an exposed vital pulp by sealing the pulpal wound with a material placed directly on the pulp to facilitate the formation of reparative dentin and maintenance of pulp vitality.[120] In primary teeth, DPC is indicated for small mechanical or traumatic exposures.[18] The clinical intra-operative judgment is most important since proper evaluation of the size of the exposure, the appearance of the pulp, and the amount of bleeding are valuable observations in the diagnosis of the condition of the pulp and treatment decision-making. DPC is not recommended for carious pulp exposures (Figure 38-20),[18] as teeth with carious pulp exposures are unlikely to be completely vital and have compromised healing potential due to infection of the pulp that causes irreversible pulpal inflammation.[121,122]

Biocompatibility of dental materials used for direct pulp capping is a major factor since the material is in direct contact with the connective tissue and has the potential to damage the viability of pulpal cells. The creation of the dentin bridge is beneficial as it provides natural protection to the pulp and is also a sign of biological recovery represented by odontoblast or odontoblast-like cell activity.[123] Calcium hydroxide has been the standard against which all other pulp capping materials have been compared, due to its induction of mineralization and inhibition of bacterial growth.[123–125] However, cells in contact with calcium hydroxide are killed due to its alkaline pH, forming a necrotic zone that causes a mild irritation to the adjacent vital pulp tissue, initiating an inflammatory response. In the absence of infection, the pulp will heal with a hard tissue barrier.[125]

Although the research on the biocompatibility of MTA and Biodentine™ is rather limited, existing information suggests these materials to be less toxic compared to glass ionomer cement and calcium hydroxide.[83] Biodentine™ has been found to promote healing when placed directly in contact with the pulp by enhancing the proliferation, migration, and adhesion of human dental pulp stem cells, confirming the biocompatible and bioactive characteristics of the material.[126] In addition, clinical and histological evidence from a study performed on molars indicated that MTA and Biodentine™ have similar efficacy in pulp capping, inducing complete dentinal bridge formation with minimal or no inflammation.[86]

A 12-month follow-up study, in which hemostasis was attained with various solutions prior to the DPC in primary molars, indicated a radiographic success rate of 100% when hemostasis was attained with octenidine dihydrochloride, 94.7% with sodium hypochlorite, 93.3% with chlorhexidine digluconate, and 84.2% with a saline solution.[127] A recent study indicated that the success rates of DPC in primary teeth after a 12-month follow-up was about 75% when the pulp was capped with calcium sulfate or $Ca(OH)_2$.[128] Yet, it has recently been stated that no good quality long-term studies are available on the success rates of DPC in primary

teeth, and the recommendations for DPC in primary teeth are based on clinical experience and expert opinions that assume that DPC may have a low success rate in primary teeth.[129] Therefore, the use of DPC in primary teeth is considered to have limited application for teeth with a small, iatrogenic, non-carious pulpal exposure and within 1 or 2 years prior to exfoliation.[129]

Clinical, radiographic, and histological analysis of the effects of MTA used in direct pulp capping and pulpotomies of primary teeth showed favorable clinical and radiographic pulp response.[130] It can be concluded that the success rate of DPC in primary teeth is dependent on contamination before, during and after the clinical procedure, and the success rate is increased by limiting treatment to non-carious exposures, use of an antiseptic to obtain hemostasis, and an hermetic restoration that prevents leakage. By the same token, DPC in cariously exposed primary molars is contraindicated due to a high incidence of internal resorption.[129] The vital pulp treatments alternatives that the clinician may consider for a carious pulp exposure are either an indirect pulp capping or a pulpotomy.

Vital Pulpotomy

The treatment of choice for carious or mechanical pulp exposures in primary teeth, larger than pinpoint, is cervical pulpotomy. This rationale is based on the notion that iatrogenic exposures larger than a pinpoint and infected pulps may have very limited potential for recovery and need to be removed.[18,59,129] A pulpotomy is preferred to a pulpectomy in primary teeth since a pulpectomy in primary molars is complicated by unpredictable and tortuous root canals that may be difficult, or impossible, to clean, shape, and properly obturate (Figure 38-23). It is also beneficial to keep the length of children's appointments as short as possible without compromising the quality of the treatment.[18,59,129] A pulpotomy is contraindicated when the radicular pulp is diagnosed with irreversible pulpitis or necrosis. In such cases, a pulpectomy or extraction should be considered.[18,59,129]

A pulpotomy in a primary tooth should allow the maintenance of the tooth in a symptomless state (no sensitivity, pain, or swelling), with healthy supporting tissues, and without eliciting any damage to the permanent succedaneous tooth, or interfering with the transition from the primary to the permanent tooth.[18,129] Internal resorption may sometimes lead to root perforation and it may be an indication for extraction.[46] However, internal resorption may also be self-limiting and stable, its advancement depending on microbial stimulation of the clastic cells involved in hard tissue resorption; without this stimulation, the resorption will not advance.[131] Therefore, clinical decision for treatment should be based on longitudinal monitoring of the internal resorption and removing the affected tooth if perforation causes loss of supportive bone or clinical signs of infection and inflammation.

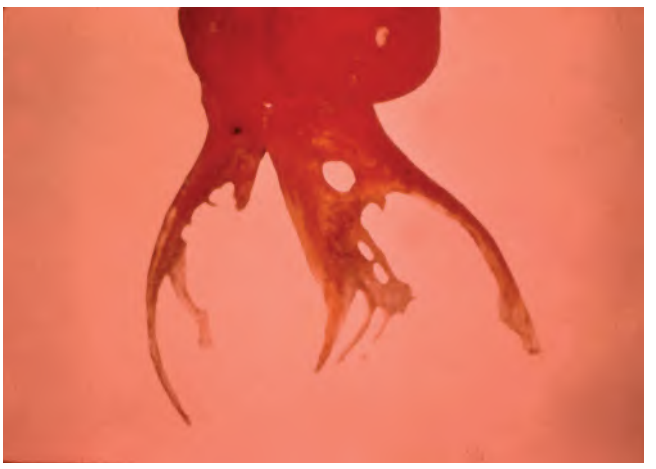


FIGURE 38-23 Root canal system of a primary molar.

Many options for pulpotomy treatment of primary teeth have been reported in the literature, among them calcium hydroxide, formocresol, glutarhaldehyde, electrosurgery, collagen, MTA, Biodentine, sodium hypochloride, and laser and electro-fulguration.[18,26,44,59,129] After any of these methods is applied to the radicular pulp stumps, the pulp chamber is filled with reinforced, or non-reinforced, zinc/oxide eugenol, or other suitable base, and the tooth is restored with a well-sealed restoration.[18,26,59,130,132,133] Reports on the influence of the final restoration on success rates of pulpotomy treatment in primary teeth indicate that a higher success rate is obtained with stainless steel crowns than with amalgams. Therefore, it has been recommended that pulpotomized primary teeth should be restored with one surface amalgam only if their natural exfoliation is expected within less than two years.[18,132,133]

Pulpotomy Technique

Pulpotomies in primary teeth are mostly performed in molars but may also be carried out in anterior teeth. While permanent teeth may be treated with partial coronal pulpotomy, or cervical pulpotomies,[134–136] a pulpotomy in primary teeth involves the complete amputation of the coronal pulp.[18,59,132,133] The pulpotomy technique in primary molars includes (after proper anesthesia and dental dam isolation) the complete removal of carious tissue, using a large round bur, at low speed, and/or with a sharp spoon excavator, starting from the dentin-enamel junction, and finalizing at the center of the cavity. This is done in order to minimize the contamination of the pulp chamber with infected carious dentin (Figure 38-24).

When the carious pulp is exposed, the pulp chamber is unroofed with a 330-tungsten bur, mounted in a water-cooled high-speed turbine, producing a funnel-shaped access to the entrance of the root canals (Figure 38-25). The opening of the pulp chamber must be wide enough to allow proper access and complete removal of the pulp tissue from the pulp chamber, and complete amputation of the coronal pulp (Figure 38-25). Tags of pulp tissue under ledges of dentin may bleed profusely and lead to an erroneous diagnosis of irreversible pulp inflammation of a normal pulp.[26] The complete removal of the pulp tissue is done with either a sharp excavator, or a clean large round bur, with care not to cause a perforation of the pulp chamber floor (Figure 38-26).

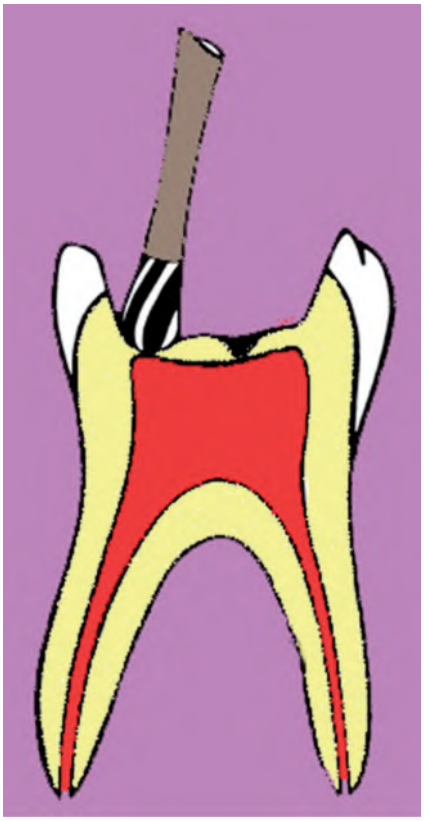


FIGURE 38-24 Caries removal.

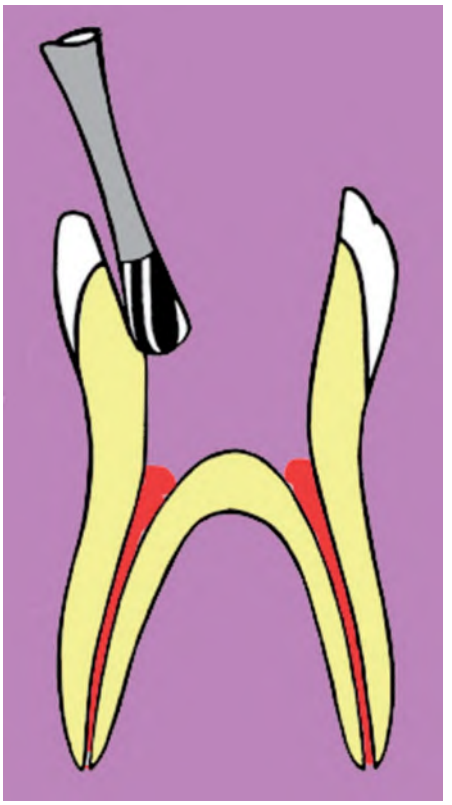


FIGURE 38-25 Removal of the roof of the pulp chamber.

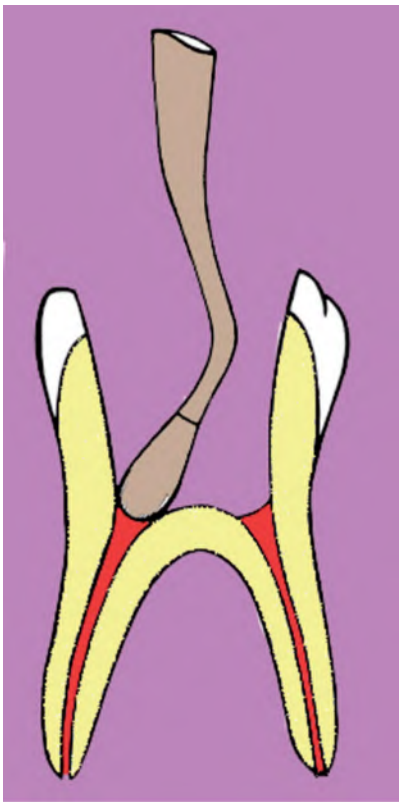


FIGURE 38-26 Removal of the coronal pulp with a spoon excavator.

The pulp chamber in primary molars is comparatively shallow and can be easily perforated.[26,137]

During the opening of the pulp chamber, and after the removal of the pulp tissue, the clinician must assess the condition of the pulp based on the appearance and consistency of the pulp tissue and bleeding amount. Lack of bleeding is usually indicative of necrosis; profuse bleeding, or a dark blood color, is usually indicative of irreversible pulpal inflammation.[36] In addition, bleeding from the pulp stumps at the entrance of the pulp canals should stop with the help of a dry or wet (with water, saline, sodium hypochlorite or chlorhexidine), cotton pellet applied with pressure for a few minutes (Figure 38-27). If the hemorrhage is readily controlled and the pulp stumps appear normal, it can be assumed that the pulp tissue in the canals is normal or capable of healing.[26]

Pharmacologic Pulpotomies

Ideally, the pharmacologic pulp dressing materials that are applied to the radicular pulp stumps for primary teeth pulpotomies should be biocompatible.[26,59]

a. *Calcium hydroxide*
The rationale of using calcium hydroxide as a pulp dressing material for vital pulpotomy in primary teeth is that it is expected to facilitate the formation of a dentine bridge and promote healing of the radicular pulp tissue.[137] However, studies indicate that this is not the case. It appears that about two-thirds of primary teeth treated with this technique fail as the result of pathologic internal resorption, which has been

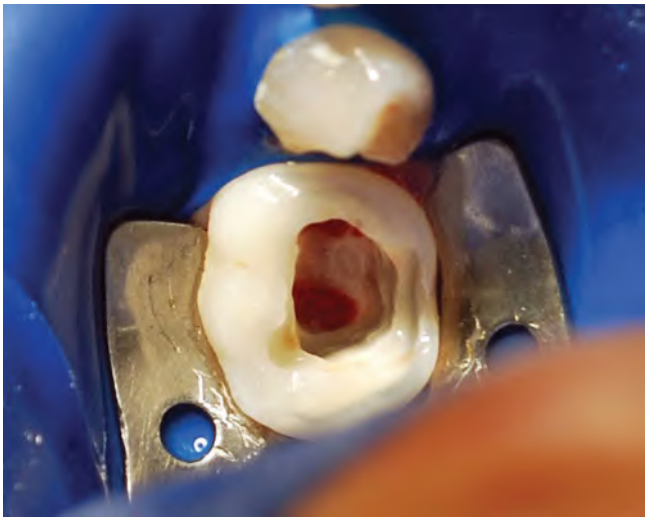


FIGURE 38-27 View of the floor of the pulp chamber and the pulp stumps of a primary mandibular left second molar after removal of the coronal pulp. Note the reddish appearance of the pulp stumps after achieving hemostasis with a cotton pellet.

related to the calcium hydroxide having no beneficial effect on the inflamed pulp and the creation of an extravascular blood clot.[138–140] This was confirmed by a recent review and network meta-analysis indicating that after 18–24 months, formocresol, ferric sulfate, and MTA showed significantly better clinical and radiographic outcomes than calcium hydroxide and laser therapies in primary molar pulpotomies.[141]

b. *Formocresol*

The initial use of formocresol (FC) as a pulp medicament is attributed to J. P. Buckley, who from 1904 to 1906 outlined the use of a mixture of formalin and cresol to treat necrotic pulps.[137] Subsequently, Dr. Sweet developed a multi-appointment pulpotomy procedure that eventually led to the current single-appointment pulpotomy for primary teeth.[137] The original Buckley's solution was composed of 19% formaldehyde, 35% tricresol, 15% glycerin, and 31% water.[137]

The treatment of primary teeth with FC pulpotomy has a history of excellent clinical success despite its inability to promote healing, and the release of formaldehyde that may diffuse through the pulp and fixate (mummify/fixate) adjacent pulp tissue.[137] Berger,[142] using a one appointment FC pulpotomy, covered pulp stumps of cariously exposed primary molars with a zinc oxide-eugenol cement in which formocresol had been added to the eugenol liquid (equal parts). This treatment was 97% successful on the basis of radiographic examination and 82% on the basis of histologic examination.[142] At the amputation site, a layer of superficial debris was observed. Below this is a zone of fixation, consisting of a darker staining area and compressed tissue with good cellular detail. Below the zone of fixation, the pulp appears to be more cellular with the odontoblastic layer well preserved. At the apical region, there is minimal cellular change, with a tendency for ingrowth of fibrous connective tissue.[142] It has also been claimed that, subsequent to formocresol application, fixation occurs in the coronal third of the radicular pulp, chronic inflammation in the middle third, and vital tissue in the apical third. The rationale and success of fixation is the creation of a tolerable irritation that replaces an intolerable infection.[137,142]

Despite the reported high clinical success rates of FC pulpotomies in primary teeth (70% to 97%), histologic studies have suggested that FC may be cytotoxic, devitalize the pulp, and cause chronic inflammation and tissue necrosis. Additional concerns include the possible diffusion of formaldehyde from the pulpal site into the systemic circulation and potential mutagenic and carcinogenic effect in humans.[143–146] Therefore, the need for finding a replacement for FC pulpotomies has been repeatedly advocated.[144–148] With the recognition of the high clinical success achieved with FC, whilst attempting to reduce its deleterious effects, several studies have investigated the use of diluted FC, or shorter applications of FC, on the pulp stumps.[149–156] The use of a one-fifth diluted FC has been recommended since it is expected to achieve the desired cellular response of the full strength FC, allow a faster recovery of the affected cells, is a safer medication with less systemic toxic potential, and has similar success rates than the full strength FC.[149–153] The one-fifth concentration is prepared by mixing three parts of glycerin with one part of distilled water, then adding four parts of this mixture to one part of the original Buckley's formocresol.[151–153] However, a survey by King et al.[154] on the concentration of formocresol used by pediatric dentists in primary tooth pulpotomy, found that 84% of 422 pediatric dentists used formocresol for pulpotomies, and that among those, only 27% used the diluted formocresol.[154]

Research on one-minute application of FC has indicated that its success rates are comparable to five-minute diluted or full-strength solutions; however, the one-minute technique showed a tendency toward early exfoliation, no effect on clinical management, and no increase in incidence of defects on succedaneous teeth.[155,156] Even so, due to FC potential for cytotoxic, mutagenic, and carcinogenic effects in humans, a continuous quest for a replacement of FC pulpotomies has taken place, resulting in various pharmacologic and non-pharmacologic alternatives, some of which are described below.

c. *Glutaraldehyde*

Glutaraldehyde (GA) is an excellent disinfectant that is active against vegetative and spore forms of bacteria and is considered superior to formaldehyde preparations.[18,148,157–161] This is due to: a) links to protein instantaneously allowing the presence of normal pulp tissue beneath the pulpotomy site; b) less volatility than formocresol; c) less apical damage and less necrosis; d) no evidence of ingrowth of granulation tissue into the apex; e) less dystrophic calcification; f) less antigenicity; and g) lower toxicity. Similar to FC, a 2% GA solution is applied to the radicular pulp stumps for four to five minutes. Although GA seems to compare favorably with formocresol, it has not substituted FC.[18,148,157–161]

d. *Ferric sulfate*
Fei et al.[140] indicated that ferric sulfate (FS) was first utilized as a hemostatic agent before the placement of $Ca(OH)_2$ in monkey teeth pulpotomies, with promising results. Later, FS was used as a pulpotomy dressing agent by itself.[140] When FS comes in contact with blood, a ferric ion-protein complex is formed and the membrane of this complex seals the severed blood vessels producing hemostasis thereby preventing pulpal hemorrhage. FS precludes the problems previously encountered due to clot formation when using $Ca(OH)_2$ and minimizes inflammation and internal resorption. However, its ability to induce a favorable histological result in the form of secondary dentin and dentinal bridging is questionable.[140,159–163] Papagiannoulis[163] reported that the internal resorption present in some ferric sulfate treated teeth did not progress or even remineralize. After removal of the coronal pulp tissue, the technique of ferric sulfate pulpotomy includes the application of FS to the pulp stumps, for 15 seconds, with a felted tip (or a small cotton pellet) (Figure 38-28A). The FS is then rinsed with water and after the water is removed, the pulp stumps are evaluated for hemostasis (Figure 38-28B). The pulp chamber is then sealed with zinc oxide eugenol, and the tooth restored, preferably with a stainless steel crown.[26,59,160,162–164]

e. *Mineral Trioxide Aggregate (MTA)*
In the MTA pulpotomy technique, after amputation of the pulp from the pulp chamber, the pulp stumps are covered with a paste obtained by mixing MTA powder (ProRoot® MTA, Dentsply, Tulsa, OK, U.S.A.) with sterile water at a 3:1 powder to water ratio according to manufacturer's instruction. It is followed by filling of the pulp chamber with a zinc oxide eugenol paste. The tooth is then restored, preferably with a stainless steel crown.[164] Previously, the clinical guidelines for use of MTA required the placement of a wet cotton pellet to allow the material to set.[165,166] Current instructions specify the placement of a light-cured resin or glass-ionomer liner to cover the ProRoot® MTA material, prior to tooth restoration. Although one study showed no detriment to placing glass ionomer on MTA prior to it setting,[79] another study showed MTA breakdown under glass ionomer cement placed for 15 minutes after MTA application.[166]

MTA pulpotomies have been found to provide similar, or better, results than pulpotomy dressed with other materials, and has been suggested to be a suitable replacement for formocresol.[73,86,164,165,167–169] The results from a meta-analysis showed that after 9–12 months, MTA had significantly better clinical and radiographic outcomes than formocresol and calcium hydroxide, and that calcium hydroxide had more failures than formocresol and ferric sulfate.[141] After 18–24 months, formocresol, ferric sulfate, and MTA had significantly better clinical and radiographic outcomes than calcium hydroxide and laser therapies in primary molar pulpotomies.[141]

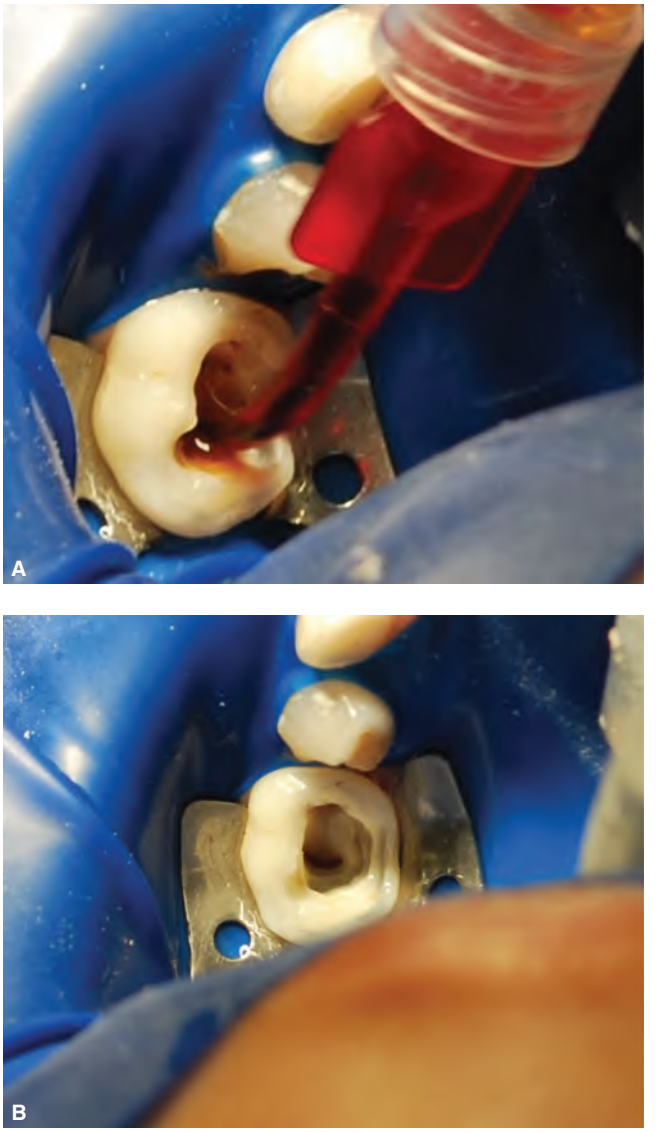


FIGURE 38-28 ***A.*** Application of ferric sulfate with a felted tip on the pulp stumps. ***B.*** After application of ferric sulfate, pulp stumps appear brown instead of red.

Challenges with ProRoot® MTA are the prolonged setting time and cost. Since Portland cement (PC) has a similar composition to MTA (PC differs from MTA by the absence of bismuth ions and the presence of potassium ions, but both have comparable antibacterial activity), PC has been considered as a possible substitute for MTA in endodontic treatments, after being sterilized with ethylene oxide and mixed with sterile water.[170,171]

At least two publications reported the use of PC as a pulpotomy dressing material in children. Conti et al.[171] reported success of PC pulpotomies in a 7-year-old boy and in a 6-year-old girl. A clinical research study by Sakai et al.[170] compared the use of MTA and PC as pulp dressing material in 30 carious human primary teeth of children aged 5–9 years old. Clinical and radiographic results after 6, 12, 18 and 24 months lead to the conclusion that all the pulpotomy treatments were successful. The only

statistically significant difference ($p<0.05$) between MTA and PC was that the beginning of mineralized material deposition could be radiographically detected after six months in 100% and 57.14% of the teeth treated with PC or MTA pulpotomies, respectively.[170]

A concern has been expressed about the possible arsenic content in PC and MTA. A review of the literature, however, suggested that the arsenic level in PC and MTA is low and therefore, unlikely to cause toxic effects.[172,173] A recent comparison of the metal contents in Type V gray and structural white PC, ProRoot® MTA, and MTA showed increased levels of calcium, nickel, and zinc in structural white PC.[174] Type V PC presented the highest levels of arsenic, chromium, copper, iron, lead, and manganese ($P < 0.05$). Bismuth was found in all cements, and the lowest concentration levels were observed in Portland cements, while the highest were observed in ProRoot® MTA.[174] Despite the potential use of PC in primary tooth pulpotomies, additional studies and approval of government regulatory offices are required before PC can be considered for use in human.

A study of pulp capping in dogs' teeth showed that a calcified bridge could be seen 1 week after treatment with white or gray MTA.[175] While there are no significant differences between the treatments, white MTA has not been found to have a significant higher toxicity than gray MTA, and both products were considered to be biocompatible.[176] Gray and white MTA presented high levels of clinical and radiographic success, but gray MTA showed significantly higher number of dentine bridge formations than white MTA.[175–177]

f. *Biodentine™*

Concerns regarding toxicity and mutagenicity of FC and the cost of MTA have led to research on the use of Biodentine™ as a pulp capping and pulpotomy dressing material in primary teeth,[83,86,178,179] An histologic study in pigs' teeth, where the pulpal response to Biodentine™ and MTA was evaluated, concluded that both materials have bioactive properties, encouraged hard tissue regeneration, and provoked no signs of moderate or severe pulp inflammation response.[179] An additional study on pulp capping of mechanically exposed pulps of permanent intact human molars indicated that Biodentine had similar efficacy as MTA.[86] The clinical use of Biodentine™ and MTA pulpotomies is the same.

g. *Sodium hypochlorite*

Sodium hypochlorite (NaOCl) has been successfully used for many years in endodontic therapy as an irrigant for permanent teeth. At low concentrations it has been found to be biocompatible, non-irritating, and hemostatic to exposed pulpal tissue. It has the ability to eliminate bacteria, superficial inflamed tissue, and dentine debris from exposure sites, while leaving the deeper healthy pulp tissue unharmed.[143,159,180–184]

The technique of NaOCl pulpotomy includes removal of the coronal pulp, achieving hemostasis, placing a cotton pellet saturated with 3% NaOCl for 30 seconds, rinsing with sterile saline and filling the pulp chamber with ZOE. The success rate for this treatment is similar to that of formocresol.[143,185,186]

The use of sodium hypochlorite as a disinfectant material of the pulp chamber, before application of calcium hydroxide, or mineral trioxide aggregate, on top of the pulp stumps in primary teeth, has also been examined in at least three studies.[187–189] The first study concluded that the use of NaOCl does not affect the success of calcium hydroxide or MTA pulpotomies.[187] Additionally, regardless of the cleansing agent, MTA was found to be superior to calcium hydroxide and it, therefore, appears that the selection of the proper medicament is more important than the choice of the cleaning agent in primary teeth pulpotomies.[187]

The second study indicated that NaOCl pulpotomy results are comparable to those with FC.[188] The third study concluded that the use of sodium hypochlorite, as an antimicrobial agent, prior to application of the pulpotomy dressing, improved the success rate of calcium hydroxide pulpotomies, to equal the success of MTA pulpotomies for observation periods of up to 12 months.[189]

h. *Collagen*

The use of collagen as a pulp dressing material, over pulp stumps of pulpotomized teeth, is based on the following facts:[190–194] a) collagen has a potent hemostatic property and ability to aggregate platelets, facilitating wound maturation by enhancing initial blood clot and fibrin linkage formation; b) collagen functions as a chemotactic agent for fibroblasts and improves their migration and attachment through its fibrillar structure, thus enhancing cell migration into the space between the collagen membrane and the pulp wound; c) collagen fibers are able to induce mineral formation and orient hydroxyapatite crystals; d) collagen has a proven rate of success in dentistry when used for guided tissue regeneration, root conditioning, hemostatic, and wound dressing agent; e) collagen has a low immune response and toxicity, the ability to promote cellular growth and attachment, homeostasis, and the added advantage of antibiotic incorporation, making it a prospective product for vital pulp therapy.

Formation of a dentinal bridge has been demonstrated in animal teeth after application of an enriched collagen solution (that contained some vitamins, amino acids, and other nutritive factors) on the pulp amputation site.[190–191] Yet, another study, using plain collagen, showed that human osteogenic protein had good characteristics as a direct pulp capping material. On the other hand, collagen matrix itself did not initiate mineralization nor created dentinal bridge, thus indicating that some kind of a stimulative chemical factor is necessary for the development of protective reaction of traumatized dental pulp, and that the mere contact with biocompatible non-stimulative matter is not sufficient.[193] More recently, a randomized, controlled, prospective two-year clinical study was done on 40 teeth of 20 children in the age group of 7–10 years.[194] Collagen particles were mixed with saline into paste-like consistency, then carried into

the pulp chamber and compressed using a damp cotton pellet. It concluded that collagen, used for covering the radicular pulp remnants after pulpotomy, was proven to be a promising alternative as a pulp medicament.[194] The use of collagen as a pulp dressing material in children's pulpotomies requires additional scientific evidence, and the process and expense involved still presents a hindrance.

NONPHARMACOLOGIC PULPOTOMY TECHNIQUES

The characteristics of non-pharmacologic pulpotomy techniques (that do not involve the placement of a pulp dressing material over the radicular pulp stumps) for primary teeth should be inexpensive, simple to use, bactericidal, completed efficiently in one appointment, and produce a high clinical and radiographic success rate. These techniques include electrosurgery/electrofulguration and laser applied to the radicular pulp stumps.

a. *Electrofulguration*
Electrofulguration, or the controlled surgical destruction of tissue with an electric current, has been reported as a nonpharmacological pulpotomy technique, with clinical and radiographic success rates that range from 54.6% to 100%.[195–200] In this technique, after removal of the pulp from the pulp chamber and hemostasis, the electrosurgery dental electrode (i.e., HyfrecatorR 705A, Birtcher Medical Systems, Irvine CA, U.S.A.) is placed 1–2 mm above the radicular pulp stumps and allowed to produce an electrical pulse to each pulp stump for 1 second, in a rotational sequence to avoid heat build-up in any one area of the tooth. After the electrofulguration of each pulp stump, a sterile cotton pellet is placed, with pressure, on the next pulp orifice to be electrosurgically treated, to absorb any blood or tissue fluids before the next current application.[195,197] The electrofulguration of all the pulp stumps can be repeated up to three times with cool down periods of five seconds between each application of the current. When properly completed, the pulpal stumps appear dry and completely blackened.[195,197]

Two studies that evaluated the histological effect of electrosurgery in dog teeth presented conflicting results; while one study indicated that the conventional formocresol pulpotomy technique is histopathologically superior to the electrosurgery pulpotomy technique,[199] the second study indicated that mechanical or electrosurgical coronal pulp removal exhibited less histopathological reaction than the teeth treated by formocresol pulpotomy.[200] Clinical research studies on electrosurgery pulpotomies, performed in children, have compared pulpotomies done with $Ca(OH)_2$, formocresol, ferric sulfate or diode laser pulpotomies and indicated inconsistent results. Nevertheless, they suggested a general trend that electrosurgery has a similar success rates to the other techniques, and that placing ZOE or $Ca(OH)_2$ over the pulp stumps following electrofulguration doesn't cause significant differences in the success rates.[196–198]

b. *Laser*
The application of lasers as a non-pharmacologic hemostatic procedure for the pulpotomy technique has a potential to increase healing, stimulate odontogenesis, and preserve vitality of the dental pulp.[201–208] In fact, an Nd:Yag laser has been evaluated in several studies, with excellent results. Low-level laser therapy (LLLT) has shown a potential for hemorrhage control, absence of mechanical contact, healing of dental tissue by the stimulation of regenerative cells, pain reduction, and edema regression with consequent anti-inflammatory effects.[207,208] From a biological point of view, LLLT offers low energy density for the target cell, and stimulates membranes, or organelles, leading to positive biomodulation and biostimulation.[207,208] After the initial removal of the pulp tissue from the pulp chamber, the technique achieves complete hemostasis by direct multiple applications of the laser beam, until the pulp is ablated at, or in, the canal orifice. Subsequently, the pulp chamber is filled with an intermediate restorative material (IRM), zinc oxide eugenol,[204,205] or a layer of MTA (laser-MTA technique). The MTA is covered with Vitrebond to achieve a firm foundation that prevents disturbance of the unset MTA.[203]

SUCCESS RATES OF THE VARIOUS PULPOTOMY DRESSING MATERIALS AND TECHNIQUES

The determination of a successful clinical and radiographic outcome with a pulpotomy in primary teeth includes: no spontaneous pain and/or sensitivity to pressure/percussion, no swelling, no sinus tract, or pathologic mobility, no furcation or periapical radiolucencies, no widening of the periodontal ligament, no internal or external pathologic internal or external resorption, and normal exfoliation of the primary tooth and eruption of the permanent successor.[18,26,59]

Formocresol (FC) and ferric sulfate (FS) have shown in retrospective and prospective studies that they have equivalent clinical and radiographic success rates; the clinical success rates ranging between 84% to 100% and 89% to 100% respectively, and their radiographic success being 73% to 96% and 74% to 97% respectively. The success rate of NaOCl pulpotomies is similar.[143] The two most common pathologic findings reported in the literature for pulpotomized primary teeth treated with FS and FC are internal resorption and furcation bone destruction. In the case of NaOCl, internal resorption, furcation bone destruction, and external root resorption were found, with many of the failures demonstrating more than one pathological outcome (Figures 38-18, 38-29, and 38-30).[143]

Many studies have attempted to evaluate the success rates of the various pulpotomy techniques, producing inconsistent results. A systematic review and network meta-analysis of 37 research studies (22 of them included in the final meta

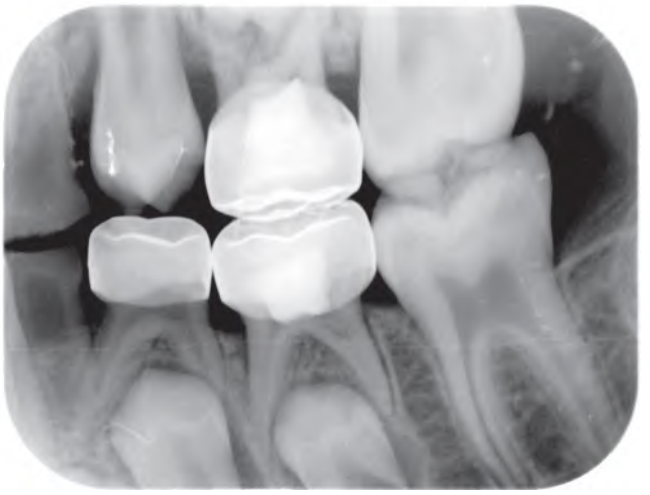


FIGURE 38-29 Bite-wing radiograph taken several months after pulpotomies were performed in the primary maxillary and mandibular left second molars. Note the obliteration of the root canals in both molars, and internal resorption, partially calcified, at the distal root of the mandibular second molar.

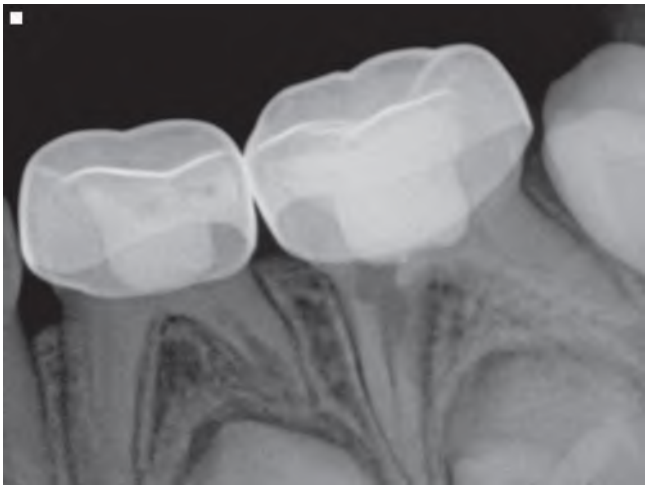


FIGURE 38-30 Follow-up periapical radiograph of pulpotomized primary mandibular left molars. The mesial root of the second molar shows internal resorption. The distal root of the first molar shows atypical internal resorption that may be related to physiologic resorption process and/or pulp inflammation.

analysis) on success rates of vital pulpotomies in primary teeth using formocresol, ferric sulfate, calcium hydroxide, and MTA indicated that: a) the success rates of the vital pulpotomy technique depend on the properties of the material itself and on the biocompatibility to the remaining radicular tissue; b) after 18–24 months teeth treated with laser therapy were two to three times more likely to fail compared to formocresol, ferric sulfate, and MTA; c) MTA has higher success rates than calcium hydroxide and laser therapies after 18–24 months and better success rates than formocresol, calcium hydroxide and laser therapies after 9–12 months.[141] Sonmez et al.[209] compared four pulpotomy techniques in primary teeth and found that after a two-year period, the success rates were 76% for formocresol, 73.3% or ferric sulfate, 46.1% for Ca(OH)2, and 66.6% for MTA.

Biodentine™ is a relatively new material and therefore, the research on its clinical, radiographic and histologic success in the pulpotomy treatment of primary teeth is limited. Nevertheless, the available information shows promising results. A study on 28 caries-free maxillary and mandibular permanent intact human molars were mechanically exposed and a pulp capping was performed with either Biodentine™ or MTA. The teeth were extracted after six weeks and examined with light microscopy. The majority of the specimens showed complete bridge formation and the absence of an inflammatory cell response.[86] A study that compared NaOCl and FC pulpotomies indicated a 100% clinical success rate for both medicaments after three months and at six months, and after 12 months NaOCl showed 95% and 87.5% clinical and radiographic success rate respectively, while FC showed 95% clinical and radiographic success rates.[210] A study by Durmus & Tanboga[211] comparing FC, FS, and diode laser pulpotomies found that after 12 months, the clinical success rates were 97%, 95%, and 100% respectively, and the radiographic success rates were 87%, 79%, and 75% respectively.

They concluded that when considering the radiographic success rates, laser pulpotomies may not replace formocresol or ferric sulfate pulpotomies in primary molars.[211] On the other hand, the opposite conclusion was reached in a nine-month follow-up study by Yadav et al.[198] in which an 86.6% clinical success rate was found for FS whereas 100% clinical success rate was found in electrosurgical and diode laser groups. Radiographically, an 80% success rate was found in all the three groups with internal resorption being the most common cause of failure.[198] A systematic review of pulp therapy has suggested that ferric sulfate, electrocautery, and formocresol pulpotomies have similar success, and that MTA pulpotomies have a very high success rates with the gray and white formulations having success rates of 90% and 100% respectively.[129]

It can therefore be concluded that there is no clear and sufficient evidence to identify one superior pulpotomy medicament and technique. Nevertheless, two medicaments may be preferable: MTA or FS. The cost of MTA may preclude its clinical use in some countries and therefore, FS could be used in most situations.[129,212] Another point that needs to be noted is that FC is no longer used in Canada and the Netherlands mainly due to safety concerns.[212]

DEEP PULPOTOMY TECHNIQUE

The clinician may face situations in which, after complete removal of the coronal pulp tissue, it is difficult to control bleeding from the pulp stumps, indicating the possibility that the radicular pulp (at least in its most coronal portion) has irreversible inflammation. In these cases, the clinician has several options: perform a deep pulpotomy (or partial pulpectomy), pulpectomy, or extraction.[18,26,59] A deep pulpotomy is accomplished by penetrating the root canals with

a small round bur to a depth of about 2–3 mmm, rinsing with water, or saline, and if hemostasis is achieved a regular pulpotomy is carried out. If hemostasis is not achieved, a pulpectomy or extraction should be performed.[18,26,59]

NON-VITAL PULP THERAPY FOR PRIMARY TEETH

Pulpectomy

A pulpectomy in a primary tooth is indicated when the tooth is diagnosed with irreversible pulpitis and/or pulp necrosis, as indicated by the child's symptoms, clinical or radiographic findings, or when clinical signs of irreversible pulpitis are present.[18] Knowledge of the pulp system morphology of the primary teeth facilitates optimal pulpal treatment. Although the pulp of a primary tooth is histologically similar to the pulp of a permanent tooth, the morphology of deciduous teeth is different in many respects from that of the succedaneous teeth, with the primary molar roots being relatively longer and more slender, that is, mandibular molar roots are narrower mesio-distally, the maxillary mesio-bucal and disto-buccal roots are narrower mesio-distally, maxillary palatal roots are narrower buccolingually, and the primary molars are characterized by a high incidence of accessory canals.[8,10,213]

Pulpectomy Technique

After administration of local anesthesia and dental dam isolation, the pulp chamber is accessed. The major objectives of the access opening are to remove the roof of the pulp chamber, all coronal pulp tissue, and locate all canals. This requires a thorough knowledge of the location, size, morphology, and variations of the root canals. Studies that analyzed the difficulties encountered with root canal treatment of primary teeth found that most difficulties were attributed to deficiencies in access; small access cavity size impedes access to the root canal system, resulting in missed canals and inadequate instrumentation.[213,214] Primary first molars presented with more difficulties in access and canal location than primary second molars in both arches.[214]

The objective of cleaning and shaping of the root canals is to remove all pulp tissue and microorganisms and their by-products from the root canal system. Traditionally, this has been accomplished with hand files but experimental and clinical evidence shows that NiTi rotaries promote improved preparation quality.[215] Although the use of rotary instruments in primary molars was initially not advised,[213] recent research has found that this instrumentation technique is advantageous and can be utilized in the shaping of primary teeth. Rotary instrumentation of deciduous teeth was first described by Barr et al.[216,217] One of the advantages of using rotary instrumentation when treating primary teeth is a decreased work time that may help to prevent behavior difficulties.[218,219] Due to the built-in taper in the rotary instrument design, their use results in a smooth funnel-form preparation that easily accepts pulpectomy filling material and may increase clinical success.[220] The clinician must also keep in mind the morphology of the primary molar roots that are widely divergent and curved.[8–10] Care must also be taken not to over-instrument as perforations can readily occur in the thin dentinal walls of the root canals.

Correct working length determination is necessary for successful treatment as this ensures a more thorough chemomechanical disinfection of the root canal system without harming the succedaneous tooth (Figure 38-31). Overextension of files can also over-enlarge the apical foramen and result in overfilling of the root filling material. The challenge with establishing working length in primary teeth is that the apical area changes as a result of the physiologic root resorption. Radiographic determination of working length and electronic apex locators are both acceptable methods that are currently used.[221,222] The advantages for the use of electronic apex locator is the avoidance of radiation, bypassing of radiographs in children that cannot tolerate them, and less reliance on subjective interpretation, especially with the complexity of resorption.

When using radiographs, the endodontic working length should ideally be 1–2 mm short of the radiographic apex and, if obvious signs of root resorption are present, it may be necessary to further shorten the working length by an additional 1–2 mm. If hemorrhage is encountered after the pulp tissue has been removed, this is an indication that root resorption has likely occurred and the working length should be shortened 2–3 mm from the radiographic apex.[213] Interestingly, an in-vivo study by Kielbassa et al.[223] concluded that the Root ZX apex locator can be recommended for clinical implementation of endodontics in primary teeth, and that the accuracy was higher when there was no resorption, while in the presence of resorption, the working length was slightly underestimated as is intentionally done with radiographs.[223]

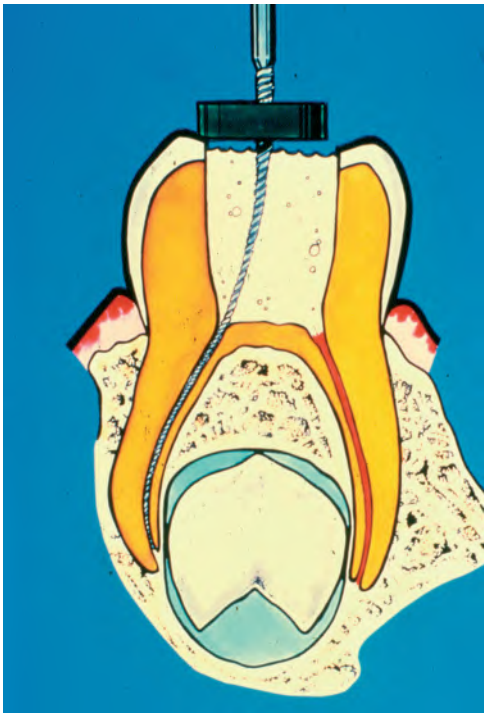


FIGURE 38-31 Pulpectomy treatment. File inserted into the canal. (Courtesy of Dr. Albert C. Goerig, Olympia, WA, U.S.A.)

Root canal treatment in primary molars requires profuse irrigation since it is difficult to completely remove the inflamed and necrotic tissue from the intricate anatomy of the pulp canals by instrumentation alone. As in permanent teeth, the endodontic infection in primary teeth is usually multimicrobial, including aerobic, facultative, and anaerobic microorganisms.[224] Root canal irrigation of a primary tooth with an inert solution cannot adequately reduce the microbial population in the root canal system, and disinfection with irrigants such as sodium hypochlorite or chlorhexidine, significantly increases the possibility of optimal decontamination of the canals.

Sodium hypochlorite is the most commonly used endodontic irrigant. It is safe to use it in primary teeth but since it is a potent tissue irritant it must not be extruded beyond the apex.[18] While Goerig and Camp[213] recommend the use of a 5.25% NaOCl (the percentage of full strength NaOCl in 1983), the AAPD recommends the use of a diluted concentration of 1% NaOCl.[18] Sodium hypochlorie has the capability of dissolving both vital and non-vital tissue. For more details, see Chapter 21.

Chlorhexidine is another antimicrobial that is used for root canal irrigation. It has a broad-spectrum antimicrobial action, lower toxicity than sodium hypochlorite and substantivity (the ability to bind to proteins and thus have prolonged activity). However, chlorhexidine is unable to dissolve pulp tissue.[225]

Ethylenediamine tetraacetic acid (EDTA) has been recommended as an adjuvant in root canal therapy for permanent teeth as it removes the smear layer.[225] However, a study by Tannure et al.[226] did not show any difference in primary teeth pulpectomy outcomes when the smear layer was removed, or allowed to remain.[226]

An ideal root filling material for primary teeth (Figures 38-32A-C) should be antiseptic, resorb concomitantly to the physiologic root resorbing process, harmless to the adjacent tooth germ, radiopaque, easily inserted, easily removed when required, biocompatible, not a cause of gross overextension or under filling, and should not induce pathologic root resorption or furcation/apical radiolucencies.[18,26,59] The root canal filling materials that have been considered resorbable and suitable for primary teeth pulpectomies include $Ca(OH)_2$, non-reinforced ZOE, iodoform paste, and combinations of iodoform with other materials (Table 38-2).[18,26,59] In addition, the use of Ledermix (Lederle Laboratories, Wolfathausen, Germany) that is composed of dimethylchlorotetracycline and triamcinolone has been mentioned as a possible root canal medicament for primary teeth.[59]

The use of ZOE as a root canal obturation material for primary teeth was first described by Sweet in 1930. When in a dense mass, it may resist resorption, irritate the periapical tissues, trigger foreign body reactions, possibly relate to enamel defects in the permanent teeth, and may remain in the alveolar bone for years.[227,228] Several research studies and case reports indicate the success percentages of the use of various root canal filling materials for primary teeth (Table 38-3). Generally; [226,229–246] a) the success rates of primary teeth pulpectomies are between 65% to 100%; b) the degree of pre-operative root resorption has an inverted relationship with the success rate of the treatment; c) since

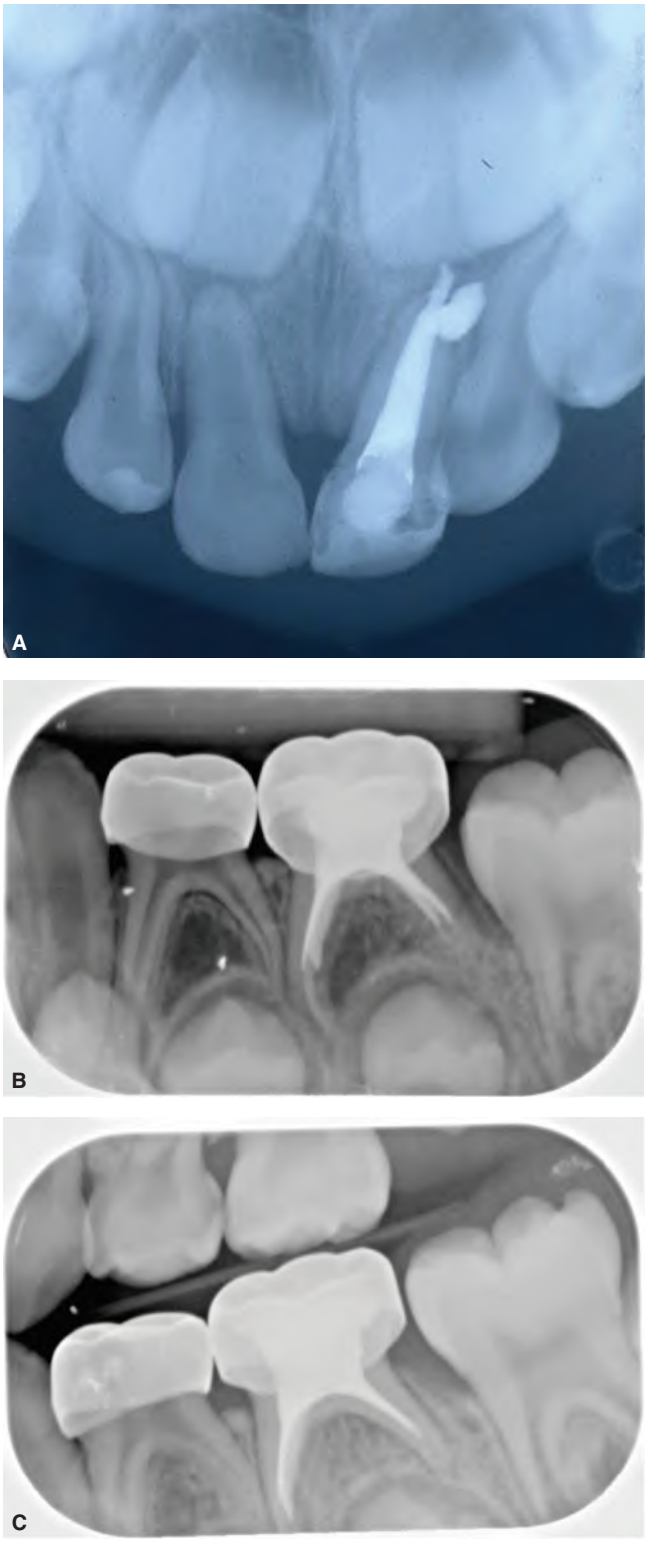


FIGURE 38-32 ***A.*** Successful root canal treatment in a maxillary central incisor. Note the root filing material that has extruded through a sinus track. ***B.*** Postoperative periapical radiograph of a root canal treatment in a primary mandibular left second molar. Note the interadicular radiolucency distal to the mesial root. Also, the end point of the root filling material is at the anatomic apex. ***C.*** Radiograph after 6 months of root canal treatment in the mandibular second molar. Note that the radiolucency distal to the mesial root is no longer present. (38-32A&B, courtesy of Dr. Moti Moskovitz, Jerusalem, Israel.)

TABLE 38-2 Main Components of Root Canal Pastes for Primary Teeth

Paste	Composition
Maisto	Iodoform, camphor, menthol, parachlorophenol, zinc oxide, lanolin and thymol
Kri paste	Iodoform, camphor, menthol, and parachlorophenol
Endoflas	Iodoform, zinc oxide, calcium hydroxide, barium sulfate, eugenol, and parachlorophenol
Vitapex	Iodoform and calcium hydroxide
Metapex	Iodoform and calcium hydroxide

TABLE 38-3 Success Rates for Pulpectomies in the Primary Dentition Using Various Root Canal Filling Materials

Manuscript	Material	Follow up	Type of teeth	Sample size	Success rate (%)
Holan et al. (1993)[235]	Kri	12->48 mos	Molars	44	84.0
	ZOE*			34	65.0
Fuks et al. (2002)[237]	Endoflas	6–52 mos	Incisors and molars	47	70.0
Mortazavi (2004)[233]	ZOE*	10–16 mos	Molars and anterior	32	78.5
	Vitapex			26	100
Moskovitz et al. (2005)[238]	Endoflas	6–77	Molars	139	82.0
Sari et al. (2008)[240]	Sealapex	3 yrs	Incisors, cuspids & molars	52	92.3
Moskovitz et al. (2010)[242]	Endoflas	6–113 mos	Molars	242	90.9
Subramaniam et al. (2011)[241]	Endoflas	18 mos	Molars	45	93.3
	ZOE*				93.3
	Metapex				100.0
Rewal et al. (2014)[232]	Endoflas	9 mos	Molars	50	83.0
	ZOE*				100.0

Abbreviations: mos = months; yrs = years.
*Zinc oxide eugenol.

the radiographic apex does not coincide with the anatomical apex, the operator should intentionally underfill the root canals; d) there is a trend for a higher success rates with under-filled or flush-filled root canal fillings than with overfilled root canals; e) even with an adequate ZOE root canal filling, there is a chance for altering the path of eruption of the succedaneous tooth (e.g., anterior cross-bite or premolar ectopic eruption) since ZOE may resist resorption; f) pulpectomies with ZOE have a similar success rate in molars and anterior teeth; g) iodoform pastes may resorb within the canals but still the clinical and radiographic outcome is excellent; h) the success rate of iodoform pastes is higher than with ZOE; i) pulpectomies after trauma have a reduced success rate compared to pulpectomies related to caries; j) there is no statistically significant difference between the success rates of the different filling materials, but KRI paste, Endoflas or Metapex are considered better than ZOE; k) the success rates are higher in teeth restored with SSCs (with the exception of one study in which a success rate of 100% was reported for teeth restored with GIC against 90.9% of teeth restored with SSC); l) teeth that had a radiolucent area before treatment have a lower success rate than teeth that did not have it (98% and 90.1% respectively); m) there is no general relationship between the root canal therapy and ectopic eruption or premolar hypoplasia; n) periodic follow-up should include radiographic examination of the interadicular and periapical areas of primary teeth with root canal treatment.

Studies on the methodology for placement of the root canal obturation material inside the roots have indicated that, with ZOE pulpectomies, there was no statistically significant difference between using a lentulo spiral mounted on a slow-speed handpiece, or rotated by hand, as evaluated by the quality of the root canal filling or success rate.[234] Vitapex® is provided in a syringe with disposable tips that are introduced into the root canal while filling the canals. If any of the root canal filling material for primary teeth is expressed beyond the apex, it should be resorbable and non-toxic to the periapical tissues and the permanent tooth germ.[227,229]

While ZOE root fillings may resist resorption, iodoform pastes resorb within 1 or 2 weeks, without evidence of causing any disturbance to the permanent successor.[227,229] Holan et al.[235] compared ZOE and an iodoform paste (KRI paste) and found that the success rate for KRI paste was 84% versus 65% for ZOE, ($P < 0.05$). Overfilling with ZOE led to a failure rate of 59% as opposed to 21% for KRI ($P < 0.02$) and underfilling led to similar results, with a failure rate of 17% for ZOE and 14% for KRI.[235] These findings are consistent with previous studies indicating that iodoform-based pastes have a superior clinical efficacy when compared to ZOE. The search for a better root canal obturation paste for primary teeth led to the comparison of the success of primary teeth root canal therapies utilizing a triple antibiotic mix (metronidazole, ciprofloxacin, and minocycline) and Vitapex®, an iodoform paste. The results indicated that both groups showed 100% and 96% clinical success at 6 and

12 months, and a radiographic success at 6 months of 84% and 80% respectively, and 76% and 56% after 12 months respectively.[236]

A two-visit pulpectomy technique has been reported in the literature;[233] in the first visit, the necrotic tissue is removed from the pulp chamber, a formocresol-moistened cotton pellet is placed in the pulp chamber and the tooth is sealed with a temporary restoration; after 1 or 2 weeks the root canals are treated. The clinician however, must keep in mind that due to behavior considerations in pediatric patients, it is desired to diminish the circumstances where there is a need to re-enter a tooth for the same treatment.

LESION STERILIZATION

Lesion Sterilization and Tissue Repair Therapy (LSTR) is a relatively new biologic approach that uses a mixture of three antibacterial drugs, namely, metronidazole, ciprofloxacin, and minocycline (3Mix) for disinfecting root canal systems.[247] This technique is indicated for teeth that show presence of external or internal root resorption and may have guarded prognosis.[248–250] This is a non-instrumentation endodontics treatment, and since this procedure depends on the elimination of microorganisms through chemical and not mechanical means, it is much simpler than the traditional pulpectomy and requires shorter chair time.[248–250] It is also suitable for teeth with physiological resorption as these are more challenging to instrument and fill to the correct length. Repair of damaged tissues can be expected if lesions are disinfected.[250]

To prepare the antibiotic powder, each drug is removed from its enteric coating, pulverized in a porcelain mortar, and stored separately in tightly capped porcelain containers in a refrigerator. On the day of treatment, the antibiotic paste is prepared by combining the three antibiotic powders (3-Mix) with propylene glycol and macrogol immediately, prior to placement into the tooth.[248] Propylene glycol allows for penetration of the antibiotics into dentin.[251] Since minocycline may cause black discoloration of the tooth and gingival, it has been replaced with clindamycin. Some clinicians also add iodoform to the paste in order to make it radiopaque.[249]

The clinical procedure includes proper anesthesia, dental dam isolation, pulp access, and removal of necrotic tissue from the pulp chamber (not from the root canals). The canal orifices are enlarged with a round bur (1 mm diameter and 2 mm depth) to create medicament receptacles. If canal bleeding exists, it is controlled with 10% NaOCl. The walls of the pulp chamber are cleaned with 35% phosphoric acid, rinsed, dried, and the antibiotic mix is then placed in the medicine receptacles created with the round bur and the floor of the pulp chamber. The access opening is sealed with glass ionomer cement and the tooth is restored with a stainless steel crown.[249,250] The success of the technique is manifested by a lack of pain, resolution of swelling/parulis/sinus tract (if present), and bone healing. Burrus et al.[249] recently published three successful cases with LSTR.

PHOTODYNAMIC THERAPY

Root canal sanitizers with a high capacity to eliminate microorganisms are considered a key factor to treatment success of pulpectomies in primary teeth. In this context, antimicrobial photodynamic therapy (PAT) has been considered a very promising approach to disinfect dentinal walls of primary teeth.[252–254] PAT is used as an adjunct to chemo-mechanical methods in an attempt to eliminate persistent microorganisms by using a laser to create free oxygen radicals; these reactive oxygen species damage cellular components, leading to cell death, in this case, microbial disinfection.[252,253] The advantage of PAT is its effectiveness on bacteria resistant to antibiotics and low likelihood of development of bacterial resistance to these treatments.[254] Limited clinical information is currently available on the use and outcome of PAT in root canal disinfection.[255] There have been two case reports on the use of PAT in deciduous teeth,[252,253] but further clinical research is required to establish its efficacy for root canal disinfection, especially when treating teeth with resistant bacterial strains. Once the canals have been obturated, the chamber and access opening are filled with reinforced ZOE, and the tooth is permanently restored. As with permanent teeth, treatment success of endodontically treated primary teeth also depends on the placement of a well-sealed restoration.

TOOTH RESTORATION AFTER PULP THERAPY IN THE PRIMARY DENTITION

The placement of leakage free margins that prevent microbial penetration is of utmost importance. Accordingly, in the case of emergency pulpotomies, a high failure rate was demonstrated for teeth restored with temporary restorations as opposed to those restored with permanent ones.[256] Traditionally, stainless steel crowns (SSCs) have been used for restoring primary teeth that had undergone pulpotomy, or pulpectomy, since SSCs demonstrate greater longevity and reduced need for retreatment, compared to multi-surface amalgam (that would be the case in most primary teeth with carious pulp exposures).[213,257,258] Although a recent Cochrane review could not find any randomized controlled trials to compare the effectiveness of restoring badly broken down primary molars with stainless steel crowns as compared with filling materials, lower levels of evidence have consistently shown stainless steel restorations to be more successful.[259] The authors of the Cochrane systematic review concluded that the absence of evidence for preformed metal crowns should not be misinterpreted as evidence for their lack of efficacy.[259] The clinician may confront situations in which SSC may be esthetically unpleasing to parents, or children. For these cases, GIC that chemically bond to the tooth and release fluoride, and resin restorations, are good options for restoring endodontically treated primary teeth.[260–263]

RELEVANCE OF FOLLOW-UP AFTER PULP THERAPY IN PRIMARY TEETH

Despite the high success rate for most properly selected pulp therapies in the primary dentition, the clinician must not ignore the possibility for complications and failures, some of which may have the potential for serious consequences on the primary and the developing permanent dentitions (Figures 38-18, & 38-33 to 38-35). Potential complications are: malposition, or delay in eruption of the succedaneous tooth, enamel defects, discoloration, and inflammatory or radicular cyst formation.[264] While no significant difference has been found between the life-span of primary teeth with, or without, a formocresol pulpotomy,[265] one study reported that pulpotomized primary teeth may have an early exfoliation.[152] However, this relationship is not clear.

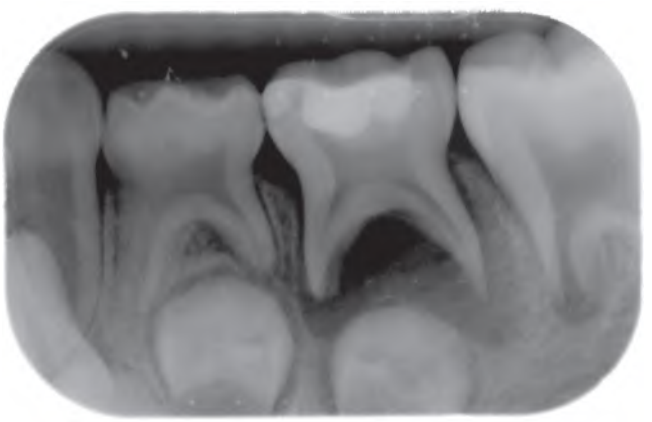


FIGURE 38-33 Periapical radiograph of a primary mandibular left second molar after an unsuccessful indirect pulp capping. Note the large interadicular and periapical area that has already affected the bone compacta surrounding the tooth bud of the permanent successor.

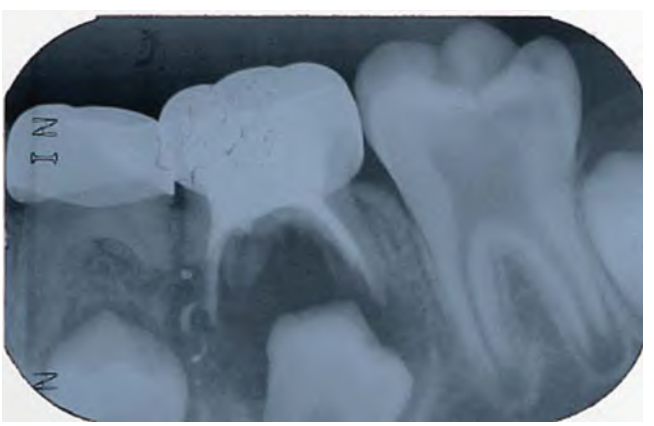


FIGURE 38-34 Follow-up periapical radiograph of an unsuccessful root canal in a primary mandibular left second molar. Note the interadicular and periapical radiolucency, and displacement of the permanent premolar successor. (Courtesy of Dr. Moti Moskovitz, Jerusalem, Israel.)

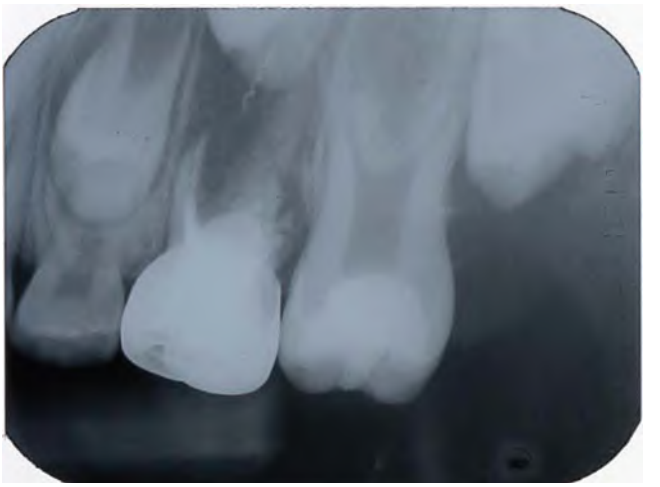


FIGURE 38-35 Follow-up periapical radiograph of an unsuccessful endodontic treatment in the primary maxillary left second molar. Note the separated segment of lentulo spiral associated with a large periapical radiolucency coronally to the permanent successor that shows delayed eruption. (Courtesy of Dr. Moti Moskovitz, Jerusalem, Israel.)

Human and animal reports suggest that primary molar interadicular or periapical pathology may affect the coronal development of the succedaneous premolars, causing hypoplastic areas.[266–268] Another study showed no significant differences in the presence of enamel hypoplasia in premolars under pulpotomized primary molars, even when considering the age at which the pulpotomy was performed.[269]

A serious complication related to pulpotomies is the development of dentigerous cysts that have been related to formocresol pulpotomies.[264,270] Radicular cysts, considered to be associated with pulpotomized primary molars, have been described as large, rapidly growing, causing buccal expansion, facial swelling, pain, pyrexia, pus discharge, and displacement of the succedaneous tooth that in most cases would require adjunctive orthodontics to facilitate their eruption to their adequate position in the arch. Furthermore, the inflammatory exudate may spread into the dental follicle of immature succedaneous teeth arresting their normal development.[271,272] Most of the cysts associated with pulpotomized primary teeth appear in the mandible and under the second primary molar.[264] Cysts under pulpotomized primary teeth may develop at any given time. One study reported a range of five months to three years with an average of 20 months.[271] Another study reported a range of 1.5 to 6 years with an average of 3.5 years.[264] These wide ranges strongly emphasize the need for long-term clinical and radiographic evaluation of pulpotomized primary teeth, and the need to extract as soon as a radiolucent pathological area is discovered.

BLEACHING OF PRIMARY TEETH

Tooth discoloration is common after trauma to the primary dentition. Among other causes, it may be related to calcific metamorphosis, a pulp hematoma, or pulp necrosis.[273,274]

Primary teeth with dark crown discoloration are kept in the mouth without the need of pulp therapy as long as there are no additional clinical and radiographic signs that indicate the need for pulpectomy,[275] or the clinician is requested by children and/or parents who are esthetically concerned to do some treatment that will improve the child's appearance.[276] Available options to treat this problem are facing of the primary teeth with composite materials, strip crowns, prefabricated esthetic crowns, or bleaching.

Dental whitening may be accomplished by using either professional or at-home bleaching modalities with combinations of hydrogen peroxide, carbamide peroxide, and sodium perborate in conjunction with different heat and light sources, or lasers.[277–281] The advantages of in-office whitening or whitening products dispensed and monitored by a dental professional include: a) an initial professional examination to help identify causes of discoloration and clinical concerns with treatment (e.g., existing restorations and side effects); b) professional control and soft-tissue protection; c) rapid results.[281] While internal bleaching requires a high level of patient compliance, dental skill and may be expensive, external bleaching represents an easier and cheaper treatment.[277]

Brantley et al.[277] described a clinical procedure for bleaching traumatized primary teeth that stained after trauma: an alginate impression of the affected teeth is taken for the fabrication of a custom no-reservoir bleaching tray from a soft thermoplastic material (Sof-tray, Ultradent Products Inc., South Jordon, UT). Next, an American Dental Association-approved 10% carbamide peroxide gel that is water-soluble and pleasant-tasting (Platinum Overnight, Colgate Oral Pharmaceuticals, Canton, MA) is placed in the tray. The length and frequency of application of the bleaching material is based on the degree of discoloration and the ongoing results of the treatment. Brantley et al.[277] started with a small amount of bleaching material (10% carbamide peroxide) in a tray for 1 hour per day for two weeks, continued with overnight use every third night for two more weeks, achieving removal of most of the discoloration with no deleterious effects such as sensitivity or abnormal exfoliation.

The most common cause for tooth discoloration is pulpal hemorrhage due to trauma.[282] The process of treatment and irrigation with sodium hypochlorite often resolves the discoloration, however, on occasions the tooth remains discolored. If this is an esthetic concern, the discoloration can be treated effectively with intra-coronal bleaching. Hydrogen peroxide is a popular active ingredient in currently used tooth bleaching materials. It acts on the pigments present in enamel and dentin by transforming complex molecules into simpler (less colored) ones.[283] Sodium perborate is a stable white powder that is also used as a bleaching agent, it was originally used as an interim appointment bleaching agent since it is significantly less caustic than hydrogen peroxide. Current literature and clinical studies support the use of sodium perborate mixed with water for bleaching nonvital teeth.[282,284,285] Studies have shown higher incidences of root resorption when hydrogen peroxide is mixed with sodium perborate,[286] or any mixture of sodium perborate is heated.[284] Therefore, the use of hydrogen peroxide and heating any mixture of sodium perborate are not recommended.[281]

As primary teeth are filled with resorbable pastes, these pastes cannot prevent leakage of the bleaching material into the canal. Leakage of bleaching material into the periradicular area may result in external root resorption.[280] A good sealing plug (of MTA, GIC, RMGI) of at least 2 mm should therefore be placed apically to the cemento-enamel junction to prevent leakage.[278] This plug should allow the sodium perborate to be placed at least 1 mm below the cervical margin. This ensures adequate bleaching of the cervical portion of the crown.[287] Although there are not that many studies of nonvital tooth bleaching in primary teeth it has been shown to be safe and effective.[281] Primary teeth are good candidates for nonvital bleaching as they are highly permeable due to the large diameter of the dentinal tubules. Nonvital bleaching offers a good option to improve esthetics of discolored teeth.

ANTIBIOTICS

Antibiotics are beneficial for the treatment of oral infections when indicated. Drug resistance of common bacteria is prevalent due to over-prescription practices and uninformed demands of patients. To diminish the incidence of drug resistance and the risk of antibiotics overprescription, healthcare providers must exercise caution when prescribing antibiotics to children.[288,289] Antibiotic therapy is usually not indicated when the dental infection is limited to the pulp, or the immediate surrounding tissue. However, when a child presents with systemic signs of infection, a fever or facial swelling, secondary to an odontogenic infection, antibiotics are usually indicated along with emergency dental treatment. Penicillin remains the drug of choice, with the occasional need to add metronidazole for anaerobic coverage.[288,289]

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Chapter 39

Interrelationships of Endodontics and Orthodontics in Treatment Planning and Patient Treatment

James L. Gutmann, Vivian Manjarrés

Only since the turn of the last century has there been a significant focus on the interactive relationships that exist between the disciplines of Endodontics and Orthodontics.[1] These relationships focus heavily on tooth trauma, resorptive defects, and alterations or damage to the dental pulp during orthodontic tooth movement (OTM). Ironically, while the literature provides great support for these relationships during the past 100 years,[2–4] articles are still being published that claim there is no interaction, especially following trauma,[5,6] similar to reports that surfaced in the 1930s.[7] This chapter addresses this interaction, using both literature and clinical experiences and outcomes, along with examples of the types of interactions that may occur, and often do occur, to the tooth and pulpal-periodontal complex during OTM.

Initially, the reader is referred to two extensively documented papers that have addressed the challenges of treatment planning in the presence of endodontic-orthodontic interrelationships[1] and the interactive role of tooth trauma, pulpal status, and orthodontic tooth movement.[8] The information contained within these publications provides a sound literature basis for the claimed relationships that do exist and question those that may not occur. However, unfortunately, there is a paucity of high-level evidence-based studies that have examined the intricacies of the interactions that do occur during OTM.[8,9] Key issues identified in the tooth and pulpal-periodontal complex during OTM that impact treatment planning and clinical outcomes are discussed, and supportive literature identified. These issues have been modified from the literature identified immediately above.[8] Where controversy exists or greater explanations are necessary, an in-depth assessment of current findings and clinical trends are presented.

The prevalence of traumatic tooth injuries in young patients is rapidly being identified globally and the management of these injuries, along with societal concerns is receiving mixed attention based on the surrounding circumstances and knowledge of the treating professionals.[10–44]

There are a significant number of studies being published globally that have addressed the incidence of traumatic injuries in young patients. However, follow-up documentation as to how many of these undergo OTM and the sequelae encountered in this process is not available. This finding demands that in anticipation of any OTM a thorough dental history is mandatory, with leading questions if necessary to secure information regarding a history of tooth trauma, no matter how slight. This concern is especially imperative when child abuse is suspected, as parents would be reluctant to convey this to the treating dentist and the young patient is only too often afraid to share these adverse experiences. The same is true for those subjected to physical spousal abuse, as the potential damage to the teeth may often be overlooked when planning for OTM.[17–19,43,44]

While the prevalence of tooth injuries in young patients is being identified, the incidence and prevalence in adult populations has received little attention.[8]

Studies are essential to ascertain the incidence and prevalence of traumatic tooth injuries in adult patients. Data on this issue is minimal within this population[45] and virtually non-existent relative to how many of these patients seek or ultimately have teeth that are subjected to OTM.

OTM in both the young patient and adult patient has increased substantially in the past 3 to 4 decades. Unfortunately little is known about the prevalence of OTM in adult populations compared to adolescents. There is some documentation that adults seek OTM to alter esthetic appearances, manage tooth crowding or malposition[46] or concomitant isolated periodontal disease, to enhance the quality of life, their self-esteem and self-confidence.[47,48] However, there is also documentation that indicates that some types of OTM in adults may not be as successful as that in younger patients, such as repositioning of impacted canines, retaining crestal bone and preventing open gingival embrasures following OTM.[49] However, these outcomes have not been correlated with tooth trauma, nor have they correlated with previous challenges to the teeth involved, such as extensive restorations, alterations in pulpal viability, previous endodontic procedures, or periodontal disease.[50]

It is common to have a different set of historical and clinical circumstances surrounding OTM with adult teeth due to the fact that most of the teeth involved will have been subjected to multiple insults or challenges to their pulps prior to OTM. Whether or not the impact of these challenges is being addressed prior to OTM is unknown.

Therefore, the incidence of deleterious sequelae over time is unknown.

There is a tremendous void in the literature with regards to this issue. Important questions can be raised in this realm, such as: do all teeth that are planned for OTM subjected to sensibility testing? Are more than just panoramic radiographs taken of teeth, with previous and possibly questionable outcomes, that encompass other treatment modalities? Are other specialists consulted prior to initiating OTM? With specific reference to endodontic concerns, Meeran[51] summarized the issues succinctly: "A complete and detailed history of the dentition should be taken, with specific attention to history of dental trauma. Radiographic examination of the teeth for evidence of pulpal obliteration is highly important, as these teeth are at a higher risk of irreversible pulpal changes during orthodontic treatment. Patients who have risk factors for pulpal necrosis with orthodontic treatment (impacted teeth, teeth with a history of trauma, caries or restorations, teeth with evidence of pulpal obliteration) should be informed about the risk of pulpal damage during treatment and informed consent taken before treatment. Light continuous orthodontic forces must be applied to move teeth, respecting physiologic boundaries. Extreme care should be taken to ensure that the intended orthodontic tooth movement does not challenge the apical blood supply (e.g., compressing the root apex against the cortical plate). Pulpal symptoms that arise during orthodontic treatment should be recognized early and treated appropriately without delay." In line with the previous statement, many authors have addressed the changes that occur in permanent teeth.[52–60]

In teeth that have been traumatized, resorptive activity may be especially enhanced during OTM. Therefore an extensive evaluation of teeth that have been traumatized prior to OTM is essential, as teeth with signs of root resorption before orthodontic movement, whether root treated or not, may be more prone to root resorption during movement.[1,5,61–65]

The importance of securing a good dental history from the patient cannot be over-emphasized. When orthodontic tooth movement is being planned on traumatized teeth there may be an accentuated resorptive response, especially if the trauma was severe.[64,66] Traumatized teeth often display signs of root resorption prior to orthodontic OTM.[67] Resorption of previously avulsed or luxated teeth has also been noted to be greater during OTM.[1] However, if a tooth presents with immature root development and has suffered an intrusive luxation, then it will be difficult to assess the pulpal status; careful orthodontic extrusion over a period of 3 to 4 weeks is advisable as is radiographic monitoring for signs or symptoms of resorption or pulpal demise, respectively. Surgical repositioning can also be considered.[61] It is in this realm of decision making and evaluation that CBCT evaluations may play an additional important role in patient communications and treatment choices, especially in light of potential changes in bone and root density due to OTM.[68]

Patients, both young and adult who have a history of tooth trauma may be at risk for additional trauma. Likewise, OTM may pose an increased risk for resorptive activities, especially should trauma occur during tooth movement.

Very few prospective, or for that matter retrospective, studies exist to provide even best-evidence directives under these circumstances. Clinical experience will usually guide the treatment rendered if any, depending on the signs and symptoms and degree of trauma. However, those patients, particularly the young and those involved in sporting activities who have suffered dental trauma are often at risk for additional trauma[17] and, therefore, during any OTM there may be an increased risk of resorption.

Teeth suffering trauma during orthodontic treatment, especially when periodontal injury occurs, often exhibit a high level of pulpal changes, from vascular alterations and fibrosis and calcifications,[59] to total canal obliteration (radiographically),[60] to a significantly high level of pulpal necrosis.[69–72] This is especially true with teeth that were orthodontically intruded first and then received trauma.[70] Also, teeth that have had trauma with severe periodontal injuries are highly susceptible to pulpal necrosis during orthodontic intrusion and extrusion.[71,72] These circumstances require careful assessment when there is a planned use of temporary anchorage devices, referred to as TADs or miniscrews, for tooth repositioning. Care must be taken not to provoke further trauma during placement of the TADs.

The use of Cone Beam Computerized Tomography (CBCT) scans in patients with a higher risk of root resorption may be of benefit during treatment planning and ongoing OTM.

In this realm of decision making and evaluation, CBCT evaluations may play an additional important role in patient communications and treatment choices, especially in light of the variabilities in bone and root density and the potential changes that may occur in the dental pulp during OTM (Figure 39-1).[63,68,69] However, concern has been expressed about the use of CBCTs in orthodontics, considering it as unacceptable as a substitute for conventional radiography.[73] An alternative may be to use quantitative digital subtraction radiography.[74]

The literature supports both the healing of root fractures and the ability to move these teeth orthodontically, provided there is no obvious evidence of replacement resorption or ankylosis. The use of the CBCT in these cases is essential.

The use of CBCT will be highly effective in determining the exact location and path of the fracture (Figure 39-2).[63,75,76] Significant evidence exists that indicates apical and mid-root fractures can heal, some with and some without endodontic intervention.[77–80] However, whether healed or not, their presence is certainly becoming more common, especially in adult patients for whom teeth can be moved orthodontically. There is a paucity of extensive, detailed evidence to support treatment planning options in this clinical situation. However, based on studies with small sample sizes or case reports,[81] orthodontic movement of teeth with repaired fractures is possible, even if the fractures at the time of the accident were extensive with marked fragment dislocation.[82] Application of light OTM forces would be indicated[83] and case monitoring would be appropriate to ensure positive outcomes.[84–86] In this regard the use of CBCT technology is warranted for both the initial diagnosis and subsequent follow-up evaluation.[68,87] These cases should be evaluated for up to 2 years subsequent to OTM, assessing pulpal responses, and tooth mobility.[1] Should ankylosis occur, movement will not be possible. Issues surrounding the orthodontic movement of teeth that have experienced fractures will be addressed in subsequent sections. Needless to say, the pulps in these teeth will have suffered some challenge to their viability, with some retaining variable responses to sensibility testing, some undergoing complete necrosis or calcification, and a small number experiencing an internal resorptive phenomenon.[88]

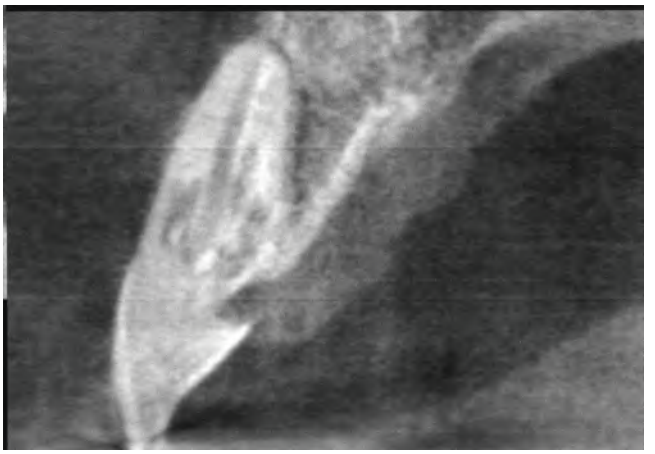

FIGURE 39-1 Details of alterations in root integrity that can be provided using a CBCT image, the full impact of which may not be evident on periapical radiographs. Note the external invasive resorption along the pulpal space coupled with defect on the palatal aspect of the crown at the level of the cingulum.

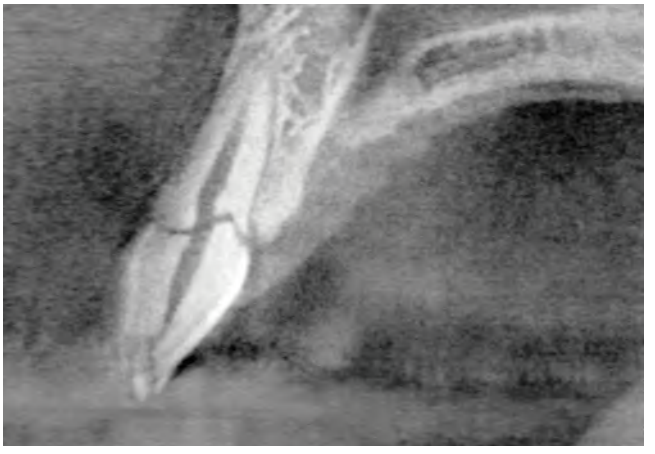

FIGURE 39-2 A coronal third fracture exhibiting a tortuous path through the coronal aspect of the tooth. The ability to determine the true nature of these fractures is enhanced with CBCT.

OTM should be considered in the treatment planning of teeth with subgingival coronal fractures based on the nature of the retained root structure, in particular its length and width, along with esthetic considerations.

Many deep, coronal subgingival fractures cannot be restored adequately and the tooth may develop periodontal complications (Figure 39-2). These cases are the most difficult to treat because of their location and the possibility for extreme mobility of the fractured coronal root segment.[65] Depending on the location of the fracture, immediate stabilization is indicated, and the need for a root canal procedure is necessary for tooth retention.[89] The use of CBCT will be highly effective in determining the exact location and path of the fracture, no matter its location in the root. Furthermore, if the clinician can probe periodontally to the fracture site, then the coronal segment should be removed and a treatment plan for the apical segment determined. Periodontal probings to the level of the fracture indicate that salivary contamination has occurred and the two segments will not heal properly; moreover, the pulp is most likely infected. Once the coronal segment is removed, the apical segment can be orthodontically moved coronally[90] and a post, core, and crown as needed can be fabricated to retain the apical segment.[91–94] Depending on the length and the shape of the root, this is not always feasible, in which case the apical segment must be extracted.

Surgical repositioning can also be considered.[61,95–99] Careful marginal extrusion, along with rotation if necessary, can result in minimal to no root resorption and allows for immediate restoration and stabilization during the healing process.[98] In teeth with immature apical formation, a more conservative approach of orthodontic extrusion is indicated and surgical repositioning is contraindicated.[41,100] Orthodontic intrusion of teeth with immature roots has been shown to have six times higher chance of presenting pulp canal obliteration than mature teeth and a lower risk of developing root resorption, that would tend to favor careful, slow OTM. The most frequent post-injury complications found in these teeth were pulp necrosis (73.3%), marginal bone loss (60%), inflammatory root resorption (40%), pulp canal obliteration (26.7%), and replacement root resorption (20%).[99] Moreover, in the young patient with these types of traumatic injuries, pulp tests cannot distinguish between a

"vital pulp" and an "unhealthy pulp," especially in the absence of signs or symptoms or if OTM is indicated, in the extrusion procedure, or subsequent to the repositioning. Secondly, the restoration of these teeth may not be the same as that planned for the adult patient, especially in the case of pulp space obliteration or other root/crown defects.[101]

The health of the dental pulp, following either traumatic tooth injuries, or a compromised tooth of an adult patient, is difficult to determine. This is especially true because the use of the terms vital and non-vital, along with reversible and irreversible, do not necessarily accurately characterize the status of the pulp.

Just because a tooth is symptom-free, no radiographic changes are evident and the tooth responds to sensibility testing does not mean that it has a healthy dental pulp. Vitality is one issue, while chronic inflammation and/or tissue degeneration lend a whole new perspective on establishing the normality of a dental pulp, before and during tooth movement, especially in the adult patient.[8]

Integrated diagnoses and treatment planning principles must take into account the status of the dental pulp (especially in adults), history of previous dental treatment or trauma, and physical appearance of the teeth. Pulp "sensibility" testing each tooth that will be involved in OTM should be done using both electric pulp testing and thermal tests. However there are no new clinical testing methods and a problem or disconnect actually exists with all presently used tests. While the clinician wishes to know the physiological condition or relative "health" of the pulp tissue, the tests only provide an indirect assessment by stimulation of the nerve tissue or possible blood flow.

The electric pulp test (EPT) provides the clinician with a response or no response that may help to differentiate normal from necrotic pulps. During usage, a medium, such as tooth paste or gel is used to insure the electrical contact between the tooth and the tip of the tester. Ideally the tip of the tester is placed at the incisal edge for the anterior teeth.[102] On posterior teeth, the favored location for placement of the tester is on the buccal cusp tip.[103] Many clinicians tend to favor the mid-facial or buccal surfaces, however false readings may occur.[104] The relative number or reading on the tester is not an indication of the relative health or disease of the pulp. Because EPTs apply an increasing power on a logarithmic, not linear scale, responses at the very highest power levels cannot be reasonably accepted as normal. EPTs can provide false-positives and false negatives up to 20% of the time. Further limitations of EPTs are the lack of response on teeth with immature root development, teeth traumatically injured, and teeth that exhibit significant coronal and/or linear calcification of the pulpal space.[8]

Thermal testing, primarily cold, is more beneficial in determining the responsiveness of any given pulp.[105] This test may be conducted with ice, crystals of ethyl chloride, or carbon dioxide spray or cold water.[106] Ice sticks or ice pencils can conveniently be made by filling and freezing the discarded plastic sheaths from disposable anesthetic needles. Testing is limited to one location on one tooth at time, placing the stimulus on the same position on each tooth. In cases where responses seem uncertain or vague, consider testing all exposed surfaces of each tooth. It will be seen with experience that responses will often vary on the same tooth.[106] An important aspect of using thermal testing actually lies in evaluation of teeth subsequent to OTM, where cold testing is considered much more reliable than EPT.[107,54]

Transillumination is used to determine tooth transparency and presence of crazes or cracks or opacities that would indicate possible previous pulpal changes of concern. All teeth with previous compromises, such as trauma, caries, restorations, etc., should be noted and color photos are recommended. The presence of discoloration,[1] such as a ***yellowish/brownish hue***, may indicate significant amounts of irritational (reparative) dentin may have formed in the crown of the tooth; whereas a ***pinkish hue*** may indicate that there is vascular damage in the pulp and or the presence of internal resorption;[1,108] while a ***grayish hue or gray-blue opacity*** may indicate that the pulp is necrotic.

In light of the compromised, yet symptom-free pulpal status often found in adult patients, the potential impact of heat that may be generated during bracket placement[109,110] and debonding procedures must be addressed.[111–114] Early studies that addressed the impact of heat on the dental pulp indicated that pulp temperatures that rose 5° – 17°C caused progressively severe pulpal necrosis.[115] Initial *in vivo* studies by Baldissara *et al.*[116] on young permanent premolars, demonstrated temperature increases in the pulp from 8.9° to 14.7°C without histologic damage. What appeared to be the determining factor was the rate of heat delivery as opposed to the absolute temperature. Furthermore, the results suggest a low susceptibility of cells to heat, which does not appear to be a major factor of injury, at least in the short term. Additional studies by Baldissara *et al.*[117] indicated that temperature rises from 39.5° to 50.4°C cause pain in patients. Studies that address light curing the brackets to the teeth indicate that temperature increases are below any critical values that might impact irreversibly on the dental pulp. With bracket debonding however, temperature values may rise close to critical values and efforts must be made to minimize these occurrences through the use of water cooling or fine burs. With both scenarios, data obtained does not reflect actual clinical findings that may impact negatively on adult teeth that may already have compromised pulps, due to trauma or to a number of other insults. This is an area that needs research to support clinical procedures.

During the clinical examination, all teeth that have deep composite restorations and/or pulp cappings evident or documented should be tested extensively. Key to this assessment is the depth of the restoration, the remaining dentin thickness, and the tooth's response to thermal testing. Ironically in this day and age of extensive, and sometimes irresponsible, replacement even of good amalgam restorations with composites, failure to understand and consider the fact that deep composite restorations are more prone to cause pulpal

breakdown over time often leads to an extensive challenge to the dental pulp.[118] While empirical, clinical experience tends to support the fact that these teeth often present with accentuated signs and symptoms of pulpal necrosis and acute apical periodontitis. Anticipation of this possibility when planning tooth movement should be a priority.

Histologic and molecular biologic changes in the dental pulp have been identified due to OTM, some of which may result in degenerative changes in the pulp.

Clinicians may not recognize that OTM may adversely impact on the physiology of the dental pulp. Research into this area does support this concept and must be considered during treatment planning.[199] Two factors appear to impact this relationship: (1) the degree of pulpal compromise or challenge before the orthodontic tooth movement; and (2) the degree and type of force being placed on the tooth over time. In the case of the former, this will mostly likely vary from adolescent to adult and from previous episodes of trauma to extent of restorative treatment or long-term presence of periodontal disease.

During rapid tooth movement, pulpal alterations or injury may occur.[59,60,199] This is due primarily to an alteration in the blood vessels in the apical periodontium and those entering the pulp. Clinically the teeth may have altered sensations to stimuli.[120] Effects of this nature may have a direct impact on the metabolism of the pulp tissue, in particular the odontoblasts in fully formed teeth, and Hertwig's Epithelial Root Sheath in incompletely formed teeth (subsequent sections on the impact of trauma on teeth undergoing apexification). A common clinical challenge that has been shown to impact on the dental pulp (and potentially the roots and the periodontium) is movement, both surgical and nonsurgical of impacted canines (Figure 39-3).[121,122] Pulpal changes and their consequences also appear to be proportionally more severe with greater orthodontic forces, particularly during intrusive forces.[59,123]

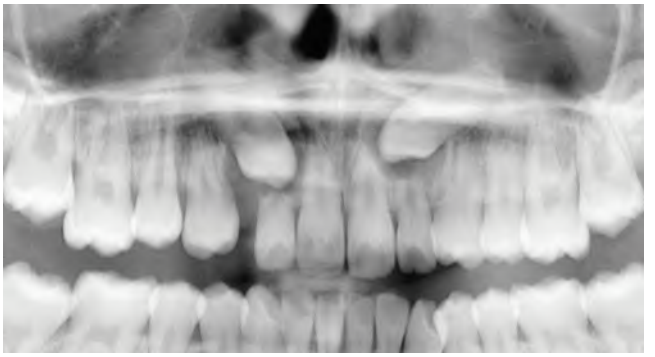

FIGURE 39-3 A panoramic dental radiograph showing extensive root damage to the maxillary lateral incisors due to impacted canines. Furthermore, if the lateral incisors can be maintained, there will be challenges within the scope of root canal procedures. Also, note that there are calcific alterations in the pulp space of the left maxillary incisor indicating pulpal degeneration. (Courtesy of Dr. Kathleen Valencia, Weston, Florida, U.S.A.)

Oppenheim[124,125] showed some signs of severe pulpal degeneration in all human cases using a labiolingual expansion appliance. The movement afforded by this technique resulted in a tipping motion in the apical third of the root. As a result, he recommended the use of light intermittent forces to reduce damage to the dental tissues and provide time for possible repair. Tşchamer noted that some of the odontoblasts will degenerate while other pulpal cells will undergo atrophy during appliance activation in late adolescent patients.[126] Historically, these findings have been supported by numerous studies.[127–131]

Investigative results indicate a relationship between the biologic effect of an orthodontic force and the maturity of the tooth, particularly the dentinogenic activity of the pulp.[132,133] This would imply that a greater dentinogenic activity coupled with a larger apical foramen would result in a reduction of detrimental effects from orthodontic forces. Ooshita noted that the surrounding tissues of bone and periodontal ligament that are linked to the tooth with an open or large foramen demonstrate accelerated activity during tooth movement.[134] Labart et al. reported an increased pulpal respiration as a result of orthodontic forces being applied to the continuously erupting rat incisor.[135] To further clarify these issues, and to address the ability of the pulp to recover following insult, Unterseher et al. assessed the pulpal respiration response after a 7-day rest period following tooth movement.[136] After initial respiratory depression, there was a return to normal respiratory rates within 1 week. Age was negatively correlated with the respiration rate, while apical opening size was positively correlated with the respiration rate.

Historically, specific angiogenic changes in the human dental pulp associated with orthodontic movement have received limited study. Angiogenesis is the formation of new capillary structures ultimately leading to the organization of larger structures by a process of neovascularization.[137] Kvinnsland and associates,[138] using fluorescent microspheres, showed a substantial increase in blood flow in the dental pulp of mesially tipped rat molars. Nixon et al.,[139] using the rat model, also found that there was a significant vascular change with an increased number of functional pulpal vessels as related to the specific forces applied. These findings contradicted those of Anstendig & Kronman who observed fewer blood vessels in tooth pulps subjected to orthodontic forces.[140]

McDonald & Pitt Ford[141] identified that blood flow changes within the pulp during tooth movement are not a mere reduction with no further response, but have dynamic changes that overcome potentially poor perfusion of the tissues. This finding further substantiated the studies by Vandevska-Radunovic et al.[142]

Derringer and co-workers identified a significantly greater number of new microvessels in orthodontically moved teeth within 5 days of treatment commencement.[143] These findings and more current studies[144] would support not only the presence of significant angiogenesis in the pulp, but also the presence of the necessary angiogenic growth factors for enhanced growth of the microvascular system.[145]

Alterations in the pulpal vasculature with subsequent alterations in the metabolism of the pulpal cells will usually

result in an increased deposition of reparative dentin in both the coronal and radicular portions of the pulp, along with a concurrent increase in dystrophic mineralization.[60,119] This response has been reported to result, in some cases, in complete obliteration of the pulpal space.[146] The incidence of this occurrence, however, does not appear to be clinically significant, at least in adolescents,[147] although a resultant pulpal necrosis can occur.[128,148,149] Most of the time related changes that occur in the pulp are considered reversible, unless the pulp has undergone previous insult or challenge. Nixon *et al.*[139] identified a force dependent increase in predentin width that was measured at the peak of the tooth movement cycle in the rat model. Pulps were considered to be normal prior to tooth movement. No differences were found with regard to location in the pulpal space that would indicate that the pulp couldn't distinguish the specific location of the applied force. What is of clinical importance appears to be the history of trauma to the tooth prior to the OTM,[150] and the radiographic observation that the pulpal space may have narrowed prior to or during the active tooth movement. In teeth that have undergone varying degrees of pulp space calcification, in particular those with extensive restorations or evidence of periodontal disease, responses, or lack thereof, to pulp testing with electrical devices may be of no value. Recent studies support the use of thermal testing, as opposed to electric pulp testing[107,54] or pulse oximetry.[151]

Neural responses and evidence for the release of specific neural transmitters have also been assessed during orthodontic tooth movement.[152] Specific intrapulpal axon response to orthodontic movement was explored using human teeth with open apices that were subjected to short-term movement and long-term movement.[153] Subsequently the teeth were extracted and evaluated under both light and electron microscopy. Although unmyelinated axons outnumbered myelinated axons, no significant differences in myelinated or unmyelinated axon numbers were observed between the experimental (orthodontic movement) and the control (no orthodontic movement) teeth. Therefore intrapulpal axon alterations appear minimal and not progressive with conservative tooth movement. This study infers that no irreversible insult is inflicted on healthy teeth undergoing conservative orthodontic treatment. This does not, however, account for the fact that the changes detected generally do not produce symptoms in teeth that already have a symptom-free degenerating pulp. Rather, symptoms of reversible or irreversible pulpitis may still be present and may be masked by the discomfort felt from changes in force that are made during appliance modifications. Furthermore, pulp tests cannot distinguish between a "vital pulp" and an "unhealthy pulp."

Precise metabolic events involved in neural transmission of nociceptive information during tooth movement have focused on the peptidergic pathways, that purportedly inhibit the firing of pain-conducting fibers,[154–157] the presence of calcitonin gene-related peptide immunoreactive nerve fibers (CGRP IR),[158,159] and the presence of substance P (SP).[156,160] These studies indicate that neuropeptides play an important role in the regulation of the blood flow to the pulp and the periodontium. In particular, there is a greater CGRP-IR fiber response in the pulp and periradicular tissues during tooth movement that is accentuated around the vascular system. The resultant increase in the blood flow to these tissues during tooth movement impacts on the availability of cells of hematopoetic origin (osteoclast precursors), that are capable under local stimulatory factors to differentiate into osteoclasts and influence the resorptive remodelling process of the teeth.[142,161]

While radiographic changes of pulp space obliteration during, or following, tooth movement are usually obvious, the lack of further dentin elaboration may not impress the clinician until other clinical or radiographic findings surface. This can occur following the introduction of orthodontic forces that are beyond the physiological tolerance of both the periodontal and pulpal vessels. Subsequent pulpal necrosis may result and may not be detected until clinically there is a darkening of the crown of the tooth. This indicates a liberation of hemoglobin that breaks down into hemosiderin (a dark yellow, iron-containing pigment), that ultimately penetrates the dentinal tubules. Also indicative of a pulp necrosis could be the presence of patient signs or symptoms, appearance of a radiolucency, or the failure of the pulp space to close with irritational dentin in a manner compatible with adjacent teeth.[119] Histological findings in the pulp that may support these possibilities have been described by Mostafa et al.[162] in response to orthodontic movement of teeth or rapid palatal expansion.[163] Specific pulp responses to tooth movement noted included circulatory disturbances with congested and dilated vessels, odontoblastic degeneration, altered gene expression and proliferation of fibroblasts, vacuolization and edema of the tissues and eventually fibrotic changes; to maxillary expansion with heavy orthopedic forces, reversible vascular changes were noted.[163] While some may consider these changes as temporary,[163–165] teeth with complete apical formation and teeth with pulps that have had previous compromises such as trauma, caries, and restorations or periodontal disease may be more susceptible to irreversible pulpal changes or necrosis under this type of orthodontic movement.[166]

During rapid tooth movement, pulpal injury may occur and changes are seen primarily in the apical vascular.[57,167,168] Clinically, patients may describe some altered sensations to various stimuli.[53,131,139,169,170] Depending on the degree and nature of the movement, pulpal degeneration can occur over the time of the orthodontic tooth movement or may be delayed in its appearance. This is often evident in a narrowing of the canal because of increased dentinogenesis.[53,149,158,162] Furthermore, the release of chemical mediators of inflammation from the pulp (especially if undergoing an inflammatory response or is in a degenerative state) during rapid tooth movement may enhance apical resorption.[171]

This potential for adverse responses should alert the clinician to do the following to prevent problems or identify problems that may have occurred with the dental pulp

during orthodontic tooth movement. Initially, baseline pulpal responses on all teeth that will be involved in orthodontic treatment should be obtained. This concept is not readily apparent to the clinician as, only too often, if the teeth look fine "clinically" there is a perceived need that there are no problems. The same is true if there do not appear to be any tooth problems on a panoramic-type radiograph. In this light, a full set of periapical and bitewing radiographs are essential prior to any treatment. In some cases if particular areas are in question, possibly a CBCT would be appropriate.[63,172–176] Moreover, a detailed history of what may have happened to the patient's dentition over time, especially with adults is also essential, *vis a vis* tooth trauma, whether severe or incidental.

During tooth movement, forces should be carefully monitored. Subsequent to orthodontic movement, all teeth should again be evaluated for any signs or symptoms of adverse sequelae, both clinically and radiographically, using the data gathered prior to treatment for comparison. This latter mandate should also be considered at least 1 year following the removal of all orthodontic appliances.

Due to the significant remodeling changes that occur during OTM that may alter the vasculature, as discussed, multiple other changes occur on a molecular biological level that alter the cellular and extracellular matrix reorganization leading to the synthesis and release of various neurotransmitters, cytokines, growth factors, colony-stimulating factors and metabolites, in addition to genetically driven or altered factors that are influenced by the mechanical forces being applied.[177–179] While the impact on the dentition of the child or adolescent patient may be minimal with reference to the teeth and bone, the impact on the adult, who has undergone changes in the dental pulp and supporting periodontium, may be significant. While there is a proliferation of isolated studies in this realm that have been identified, many of these factors during or subsequent to tooth movement,[143,144,180–186] little is known of the implications when integrating these findings in the clinical setting from a pulpal standpoint. Relative to the periodontium, however, matrix metalloproteinase-1 (MMP-1) and cathepsin K are important in root resorption during tooth movement because they degrade the collagenous bone matrix in a mode similar to bone resorption.[187,188]

The presence of root resorptive changes may be difficult to ascertain on two-dimensional radiographs. Sufficient mineral per unit volume must be lost from the tooth to make a judgment of a radiographic appearance of resorption. Hence the value of the CBCT is most important in these cases.

As previously discussed, the use of the CBCT, while potentially exposing the patient to greater amounts of radiation,[73] may be necessary in those cases where the patient is at greater risk for tooth resorption during OTM based on an overall assessment of case factors.[176] Severe incisor root resorption has been associated with impacted maxillary canines, especially in female patients, severely mesiodistally displaced and vertically positioned canines adjacent to the middle third of the adjacent incisor roots, dental follicles wider than 2 mm and normal lateral incisors.[189] The implications are obvious in this scenario as often endodontists are challenged with the management of severe incisor resorption in teeth both symptomatic and symptom-free (Figure 39-4). Moreover, and as discussed previously, the presence of root resorption before OTM, in addition to extractions, reduced root length, decreased crown/root ratio and thin alveolar bone represent risk factors for severe root resorption in maxillary incisors during orthodontic treatment.[190] Hence, the use of the CBCT may be warranted in cases suspected of having teeth with resorptive processes during treatment planning for OTM.

Both apical and lateral root resorption may be identified with OTM, especially in maxillary lateral incisors and premolars.

This outcome can easily present a problem for the clinician, during diagnosis and treatment planning (Figures 39- 5 to 39-7). Often the resorptive defect may not be limited to the mesial or distal surfaces and if present on the buccal or lingual/palatal tooth surfaces may not be discernable using periapical radiographs until >10% or more of the mineral per unit volume is lost.[171,191]

If a pulp is non-vital or degenerating, following trauma in the young patient or as may be found in the adult patient due to multiple long-term insults, and the tooth is undergoing OTM, there may be a greater chance for an active resorptive process.

If a pulp is non-vital or degenerating and undergoing tooth movement, then a greater chance for an active resorptive process exists.[171] ***Hence the dictate to evaluate all teeth prior to orthodontic movement, especially in the adult patient is imperative***. This is especially necessary in cases of planned intrusion[123] vs. extrusion,[192,193] where intrusive forces appear to have a more deleterious impact on the pulp. However, this complication may be minimized with the use of non-steroidal anti-inflammatory drugs.[193]

If necessary, all suspect teeth or teeth with a definite diagnosis that requires a root canal procedure should be treated before orthodontic tooth movement; or if the movement has commenced, it should be stopped and root treatment should be done before movement begins again. Additionally, the placement of a permanent restoration is advisable to ensure a coronal seal.[84]

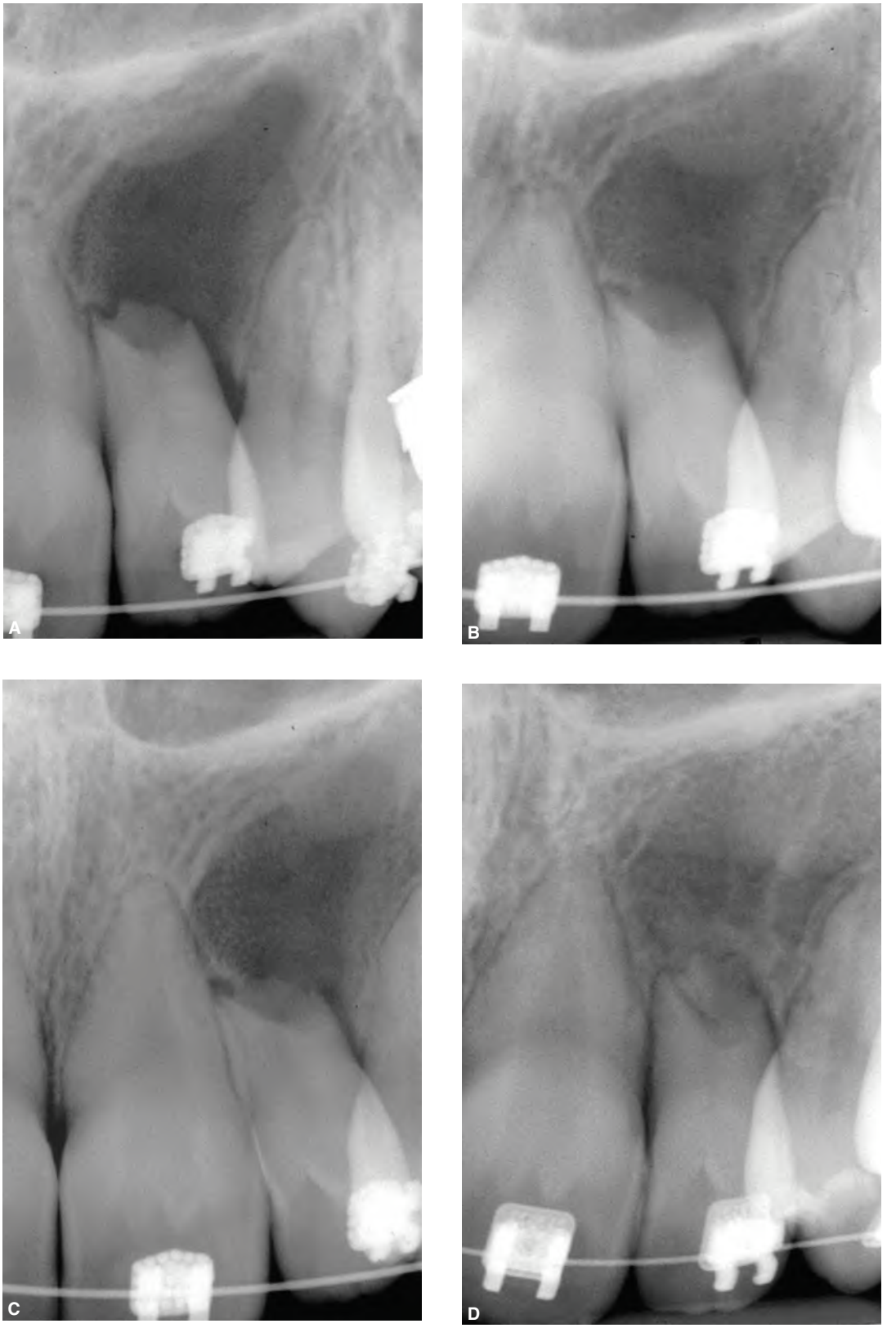

FIGURE 39-4 ***A.*** While resorbed lateral incisors can pose challenges to the endodontist, following the removal of impacted canines, often a "wait and watch" endodontic approach is indicated. ***B & C.*** represent a 3- and 6-month evaluation of the site; the patient is symptom-free and tooth discoloration is minimal. ***D.*** A one-year evaluation shows almost complete osseous repair with the only endodontic intervention being diagnosis, treatment plan and periodic evaluation. (Courtesy of Dr. Sonia Ferreyra, Cordobá, Argentina.)

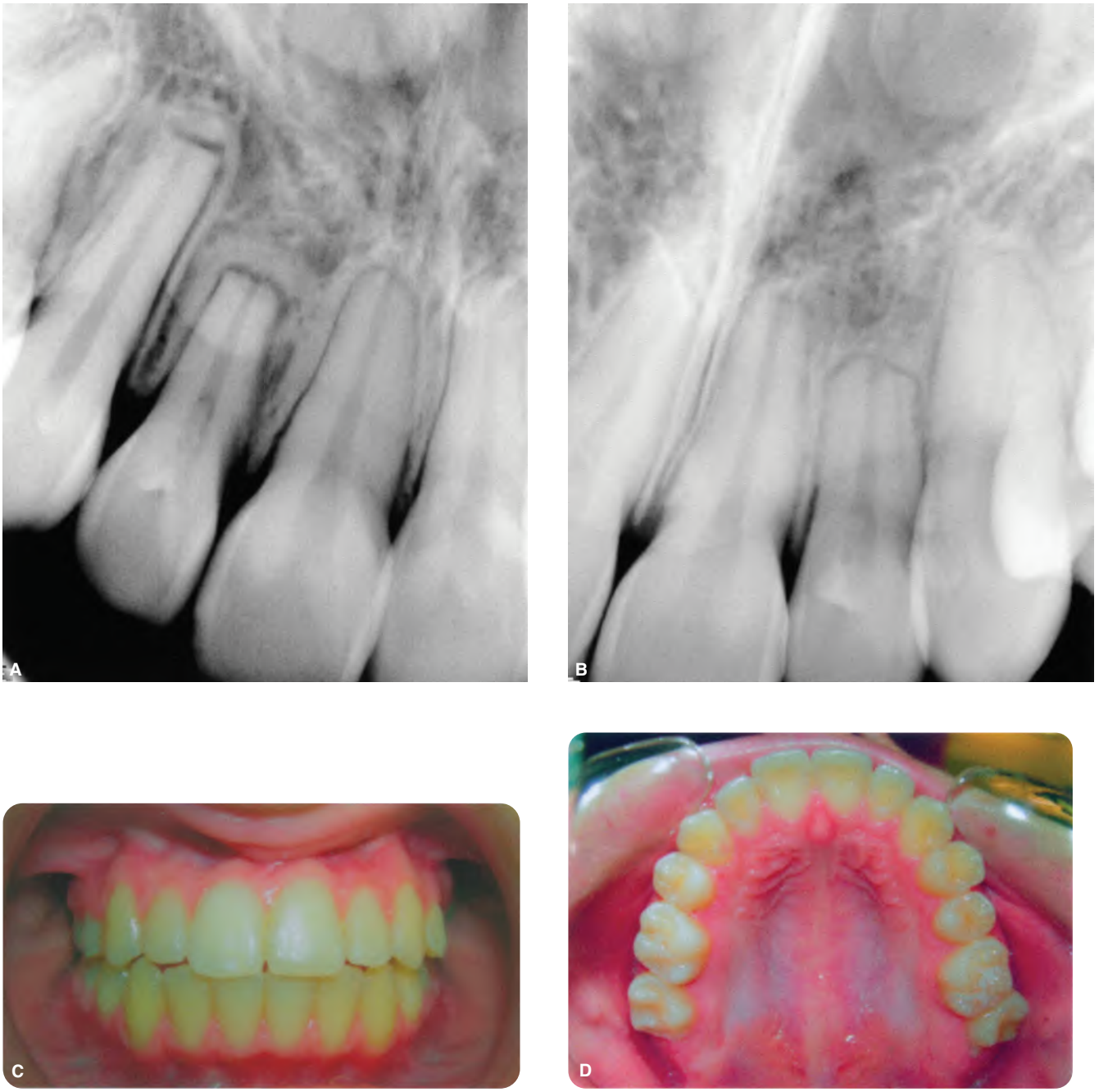

FIGURE 39-5 ***A & B.*** Right and left radiographic views of a 15-year-old female who had undergone OTM for 3 years. Significant resorption is evident on all maxillary incisors. Also of note is the increased bone density and width of the lamina dura around the blunted root apices along with the build of calcification in the coronal aspect of the two lateral incisors that may indicate pulpal degenerative changes. ***C & D.*** No discoloration of the crowns is present. (Courtesy of Dr. Kathleen M. Valencia, Weston, Florida, U.S.A. [Radiographs and photographs].)

Dental pulp tissue plays an important role in the inflammatory process involved with apical root resorption. Pulp extirpation, may be effective to alter progressive severe root resorption that may occur during OTM. The same is true in cases of a necrotic pulp or when there is failure of previous root canal procedures.

Studies indicate that orthodontic force affects dental pulps via the rupture of blood vessels and vacuolization of pulp tissues.[141,194] Further studies have hypothesized that injured pulp cells may express macrophage colony-stimulating factor (M-CSF) and the receptor activator of nuclear factor kappa-B ligand (RANKL), thereby stimulating the appearance of odontoclasts and their destructive activity.[195] Pulp removal

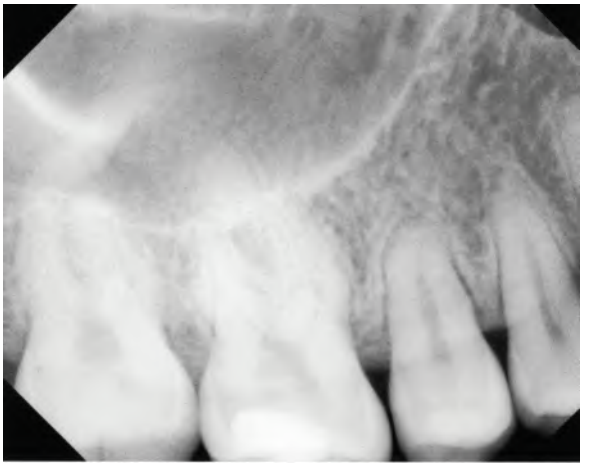

FIGURE 39-6 The maxillary premolars are often affected by apical root resorption during OTM.

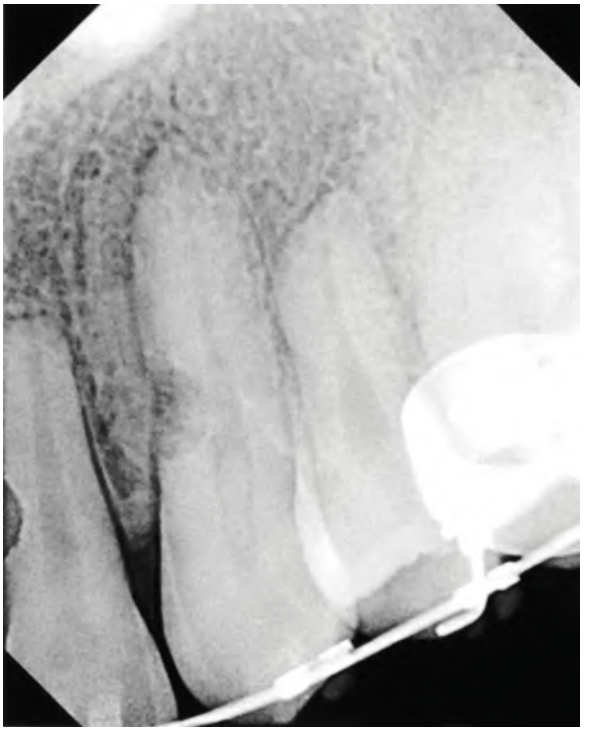

FIGURE 39-7 Lateral root resorption may also occur during OTM.

in the presence of this type of resorptive activity during OTM is suggested as a primary treatment modality for the management of this process.[195] The same situation presents itself in the case of teeth requiring revision of previous root canal procedures prior to OTM (Figure 39-8)

Teeth with previous root canal procedures, without a history of trauma, can be moved the similar distances as teeth with presumably vital pulps; but what about teeth requiring root canal procedures during active OTM?

Root-treated teeth can be readily moved the same distances as teeth with vital pulps.[196,197] This presumes that there would be no other factors that may prevent tooth movement, such as the presence of replacement resorption (or ankylosis) that may occur after certain traumatic incidences, or be the result of injury to the apical periodontal ligament by the root canal filling material.[198] However, the risk of external apical root resorption exists during an OTM.

The clinical challenge is how to manage teeth requiring root canal procedures during OTM. Initially the root canal or canals can be enlarged, shaped, cleaned, and calcium hydroxide ($Ca[OH]_2$) placed as an interim dressing.[199] The coronal access opening must be sealed properly, preferably with a bonded composite material. Orthodontic movement can be resumed immediately. Canal obturation can occur subsequent to the termination of orthodontic treatment; or, the entire root canal treatment can be completed, and the coronal access opening sealed. Orthodontic treatment can resume in 10 days to 2 weeks, after any periradicular inflammation has resolved. A short-term use of non-steroidal anti-inflammatory drugs can be considered.[193]

If a failing root canal treatment is identified (signs, symptoms) during orthodontic tooth movement, then revision of treatment is indicated using $Ca(OH)_2$ and chlorhexidine[200,201] to disinfectant the root canal or to minimize microbial populations found commonly in root canals requiring treatment revision.[202–204]

In an early literature review by Steadman,[205] root canal treatment was criticized in that it was claimed that the devitalized root acts as a foreign body causing chronic irritation and root resorption. (This was more of a carry-over from the Focal Infection Era.) Histological sections of such resorptions showed cellular pictures typical of a foreign-body reaction. Steadman considered that the resorption could not be controlled and therefore the prognosis for these teeth was unfavorable. He even went to the point of suggesting, based on the literature, that because of the resorptions, the roots of these teeth would become ankylosed, thereby eliminating the possibility of OTM. Huettner & Young[206] challenged Steadman's theory and evaluated the root structure of monkey teeth with both vital and non-vital pulps (root canal treatment) following OTM. Maxillary teeth were treated and obturated with gutta-percha and Kerr's root canal sealer. The mandibular teeth were treated and obturated with silver cones and sealer. All teeth initially had vital pulps, were treated in an aseptic environment, and were allowed to "rest" for 3 weeks following the root canal procedures to give the apical periodontal ligament time to heal (not a contemporary dictate). The edgewise fixed appliance technique of orthodontic tooth movement was used and the experiment was performed 6 to 8 weeks prior to animal sacrifice. Histological examination showed no foreign-body reactions and the root resorption that was observed was similar in both teeth with vital pulps and no pulps. Similar findings of no difference in the amount of resorption

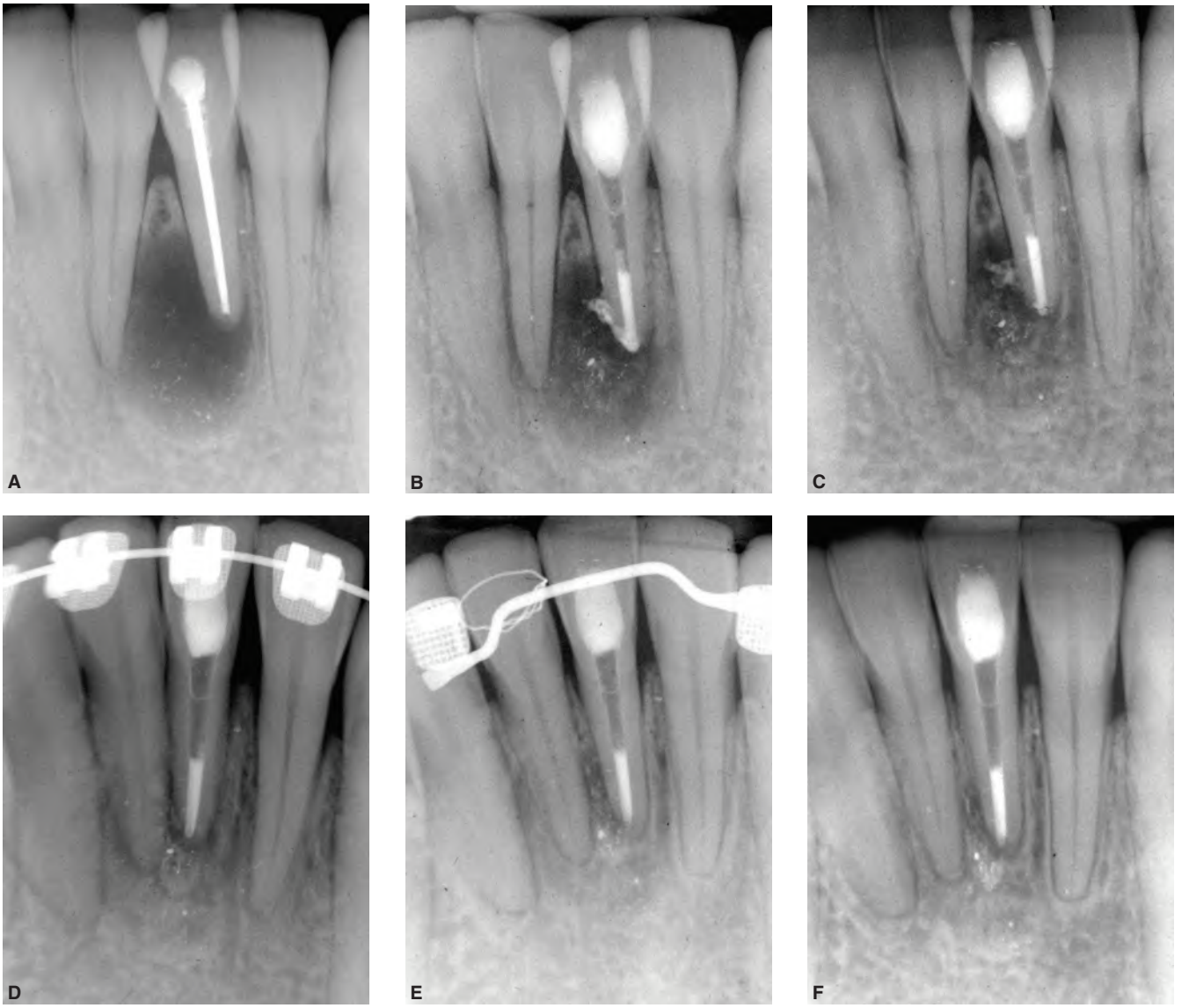

FIGURE 39-8 ***A.*** Mandibular incisor of a 26-year-old male scheduled for extraction prior to OTM due to misalignment and the presence of what was called an "apical cyst" following a root canal procedure. The patient preferred to retain the tooth, if possible. ***B.*** Revision of the previous root canal procedure. ***C.*** Six-month evaluation showing osseous repair. ***D.*** Nine-month evaluation showing repositioning of the tooth along with further osseous healing. ***E.*** Twelve-month evaluation with arch bar to stabilize the repositioned teeth. ***F.*** Eighteen-month evaluation; tooth is stable and patient is symptom-free, however, the tooth has not been restored properly.

with the two experimental groups was also reported by Weiss.[207] Wickwire et al. [208] reviewed 45 orthodontic patient case histories that contained 53 root-treated teeth from six practices that included the following orthodontic techniques: edgewise, Begg, and partial banding mechanotherapy. Historical data, lateral cephalograms, and appropriate radiographs were used to evaluate the teeth. Data revealed those teeth with root canal treatment moved as readily as teeth with vital pulps, but there *"appeared"* to be greater radiographic evidence of root resorption in the root-treated teeth when compared to the controls.

In an in vivo study on cats, Mattison et al. showed no significant difference between external root resorption of endodontically treated and teeth with vital pulps when both were subjected to orthodontic forces;[196] the same outcome was determined by Kindelan.[5]

The severity of external apical root resorption on teeth with vital pulps versus root treated teeth was determined by Spurrier and co-workers.[209] Forty-three patients who had one or more endodontically treated teeth before OTM that exhibited signs of apical root resorption after treatment were studied. Vital contralateral incisors served as controls. Incisors with vital pulps resorbed to

a significantly greater degree than incisors that had been root treated. Control teeth in males exhibited a statistically significant increase in resorption over control teeth in females. On the other hand, no differences were noted between genders with the treated teeth. Similar findings were recorded by Remington et al.,[210] although differences were not found in the incidence of resorption between teeth with vital pulps and root treated teeth, in a targeted population of British school children.[211]

Further studies evaluated the effectiveness of orthodontic forces in moving root-filled teeth and the degree of external apical root resorption that may occur in the ferret animal model.[197] Three months after root canal procedures and OTM with an orthodontic spring, tooth movement was assessed from pre- and post-treatment mandibular casts, and by fluorescence microscopy from labelled (procion red dye) bone deposition. Root-filled teeth and those with vital pulps moved similar distances when subjected to the same forces. Root-filled teeth showed greater loss of cementum after tooth movement than teeth with pulps, but without significant differences in radiographic root length. The root-filled teeth also showed more resorption lacunae than teeth with vital pulps, but the small difference in incidence between active (orthodontically) root-filled teeth and inactive root-filled teeth was not statistically significant. This suggests that the incidence of resorption lacunae may be related to non-vitality, and probably the presence of periradicular lesions, rather than orthodontic forces. These findings are in agreement with other studies.[196]

To the contrary, Bender and associates suggested that the loss of the release of neuropeptides from a pulp that has been removed would result in a decrease of the CGRP-IR fibers and a reduction in the amount of resorption seen in root treated teeth.[171] These suggestions supported the findings of other investigations,[212,213] especially when using attractive magnets for controlled tooth extrusion following deep coronal fractures.[214] In the former studies, the periodontal vascular network and axonal characteristics were within normal limits with no significant changes in blood flow or neuronal densities. In the latter study, no resorption was noted.

In a sample of 39 pairs of contralateral teeth with and without root canal treatment in 36 patients, Mirabella and Årtun found that there was significantly less resorption in root-treated teeth.[215] Here again, to the contrary, other studies found that there was no significant difference in the amount or severity of resorption during OTM between root-filled teeth and their contralateral teeth with vital pulps.[216] The study was done on adult patients (n = 77) with a mean age of 32.7 ± 10.7 years, using multiband/bracket orthodontic movement for at least 1 year. Further studies in this realm appear in conflict and quality evidence-based research is indicated.

Clinically, many important questions relative to this issue should be addressed during treatment planning.

Potential problem: Does the root canal treatment that has been provided appear to be at the highest level of technical excellence, that is, are there any concerns radiographically or clinically (signs or symptoms) about the quality of the treatment?
Outcome-solution: A thorough examination is essential of each tooth that has been root treated. If necessary, referral to a specialist should be considered. There should be no evidence of discomfort to function and no evidence of radiolucencies, apically or laterally on the specific teeth, or for that matter active resorption. The root canal filling should be dense (no voids especially in the apical 1/3) and positioned at the end of the root canal. Excessive extrusion of root filling materials may predispose to a greater inflammatory response during tooth movement.

Potential problem: Is there any reason to question the integrity of the coronal restoration *vis a vis* the potential for coronal leakage that might be contaminating the root canal system?
Outcome-solution: Good periapical and bitewing radiographs must be made and evaluated. If still in doubt, a CBCT may be indicated. Restorative–tooth margins must be evaluated as well as determining the presence or absence of periodontal disease. Restorations must be stable and exhibit no obvious clinical defects.

Potential problem: If root-filled teeth are subject to the resorptive phenomenon during tooth movement, what will happen to the root canal filing material?
Outcome-solution: The tooth may resorb, exfoliate, and the filling material may be removed with the tooth. Likewise, the tooth may resorb, exfoliate, and the filling material may be left in the bone. In these cases, if the material is gutta-percha, then a fibrous capsule will probably surround it. It is also possible that a sinus tract may form, and the material will require removal. With gutta-percha present, the extended material may undergo resorption after the tooth has undergone resorption and exfoliation. In some cases the root may begin to resorb, exposing the filling material; subsequently the resorption ceases with the filling material protruding beyond the new apical foramen. In this situation the root is often seen to develop a new periodontal ligament space and lamina dura around the root apex in close approximation to the filling material. In other cases, once the apical resorption begins a radiolucency develops around the root apex and the filling material. A sinus tract may be developing, or there may be incidences of localized swelling. Likewise, the tooth may remain symptom free and function normally.

Potential problem: Will the apical seal in the root canal system be altered, resulting in failure of the root canal treatment if resorption should occur?
Outcome-solution: If a root canal has been properly enlarged, shaped, cleaned, disinfected, and three-dimensionally obturated, then the apical seal would be sustained no matter what the extent of the apical resorption. Here too, however, multiple possibilities exist. Although the apical seal remains intact during the resorptive process, the complete seal of the canal may be challenged by coronal leakage. Exposure of the dentinal tubules that may harbor necrotic tissue, bacterial endotoxins, biofilms and bacteria may serve as sources that provide sufficient apical irritation to stimulate an extended or deleterious inflammatory resorptive process. Clinical evidence exists for apical root resorption on teeth with previous root canal treatment in which a portion of the root is missing, the filling material is visible in the surrounding periradicular tissues, no radiographic evidence of pathosis exists, and a normal periodontal ligament space and lamina dura are present.

While contemporarily popular for intruding or extruding teeth, mini-screws or temporary anchorage devices may result in pulpal problems or root resorption. Damage to the dental pulp or root structure could occur with the aberrant placement of these devices.

Orthodontic miniscrews have become quite popular due to their simplicity of both placement and removal, along with the need for minimal patient compliance.[217,218] Once placed, they generally remain stationary in the bone and provide increased anchorage capacity for tooth movement, with particularly good outcomes with palatal placement.[218] Details on their advantages on research activities evaluating their outcomes have been identified in the literature, along with several of the complications encountered in their use.[219,220] These complications include injury or damage to adjacent structures, such as periodontal ligament, tooth root, nerves, blood vessels, sinus, and mandibular canal. Animal studies have indicated that pulp and root damage from miniscrew placement rarely affects the prognosis of the tooth,[221] although inflammation and infection can occur around the miniscrew implant. When the damage from the placement of the anchorage device is limited to the cemental surface, healing occurred in time, with no adverse effects on the tooth.[222,223] However, when the miniscrews were place into the roots, close to 80% failed, but ultimately the defects healed with an osteoid or osteocementum tissue.[224,225] The actual failure of miniscrews is primarily due to their inability to remain anchored in the bone under the applied forces or when in advertently placed into a root.[220] With respect to the former, the thickness of the cortical plate appears to play a major role in retention and force distribution.[226]

From an endodontic standpoint, necrotic pulps have been identified due to damage caused by incorrect miniscrew placement[227] as well as extensive apical resorption during extensive intrusive movements using miniscrew anchorage;[228] to both pulpal damage and root damage requiring both nonsurgical and surgical root canal procedures;[229] and to root damage requiring surgical repair.[230]

Teeth undergoing normal apical root closure, or those that have been treated to allow for apexogenesis, can be moved orthodontically.

In teeth undergoing apexogenesis, there is minimal information at best as to the impact of orthodontic tooth movement. Apexogenesis is the continued and normal formation of the root and its apex[231] and is not really a treatment procedure. This term applies to teeth in which the coronal pulp has been compromised through trauma or caries, but clinically it is determined that the apical to middle portions of the pulp are sufficiently viable to allow root development to continue. Often these pulps, when exposed, have been capped with $Ca(OH)_2$ or MTA (either as a pulp cap or the material has been placed on the radicular pulp stump during a pulpotomy).[232–235] With the open apex and enhanced blood flow, normal root formation usually occurs; however, during movement, Hertwig's Sheath may be displaced resulting in an anatomically abnormal root shape, stunted root growth, or possibly even death of the sheath's cells. Clinical experience would tend to support the safe movement of these teeth, although each case will present with different circumstances and needs.

Teeth with non-vital pulps and open apices may also be considered for OTM provided there is no apparent infection present. If infection is present, root treatment using apexification or regenerative procedures must be considered.

With a significant amount of trauma occurring in younger patients who may have teeth in various stages of root development, this issue is of great importance; however, little detailed evidence is available regarding outcomes. Case reports[236] and anecdotal evidence[237] have indicated that teeth undergoing apexification may actually display the formation of a hard tissue barrier during orthodontic tooth movement as opposed to apical resorption.[238]These clinical findings would tend to support the theories of Ooshita[132] and Hamersky et al.[133] in which the accelerated biological activity of the periradicular tissues during tooth movement may have a beneficial effect on a tooth with an open or large apical foramen. In this regard the use of $Ca(OH)_2$ for the apexification procedure would tend to provide an environment conducive for a positive outcome. However, when considering the amount of apexification that is being done contemporarily in one visit using

mineral trioxide aggregate (MTA), no information is available on the outcome of this technique relative to hard tissue formation or apical resorption.[239,240] What is known however is that MTA stimulates the formation of calcium hydroxide, with subsequent formations of calcium phosphates and hydroxyapatite, that are conducive to hard tissue formation (Figure 39-9).[241–243] Moreover, the long-term use of calcium hydroxide may impact on the strength of the dentin.[244] Recent case reports have focused on the use of platelet-rich plasma as an internal matrix followed by the placement of MTA[245,246] or Biodentine (a calcium sulfate bioceramic).[247,248]

Often teeth with open apices are subjected to multiple, and at times, different types of trauma. Each particular set of circumstances must be carefully assessed and integrated treatment plans developed to achieve success.

REVITALIZATION/REGENERATION

The initiative of revitalization/regeneration of the dental pulp or mesenchymal tissues in the case of a seemingly necrotic pulp and open, immature apical formation has developed some of its own nomenclature in its claims, such as bioroot engineering, pulp revascularization, regenerative endodontic treatment or regenerative therapies, regenerative scaffolds and stem cells from the apical papilla (SCAP).[249–252]

While only supported by numerous diverse case reports or case series at this moment, there is a significant research initiative to pursue this model, calling it "the hidden treasure in apical papilla."[251,253] The treasure refers to the uniqueness of the SCAP relative to cellular types and their potential for differentiation and expression of their genetic potential, for example, cells that have the phenotypic capability of becoming new odontoblasts. If possible, this combination, along with the genetic capabilities of cells from the periodontal ligament, or periodontal ligament stem cells (PDLSCs) would be able to truly regenerate the lost or damaged tissues.[251] While the future of this approach to pulpal regeneration appears promising, not all studies have been positive. Chen and co-workers found, in a case series, that the outcome of continued root development was not as predictable as increased thickening of the canal walls in human immature permanent teeth with infected necrotic pulp tissue and apical periodontitis/abscess after revascularization procedures. Continued root development of revitalized immature permanent necrotic teeth depends on whether the Hertwig's epithelial root sheath survives in case of an apical periodontitis/abscess. Severe pulp canal calcification (obliteration) by hard tissue formation might be a complication of internal replacement resorption or union between the intracanal hard tissue and the apical bone (ankylosis) in revitalized immature permanent teeth with necrotic pulps.[254] Even in light of the variable outcomes of this proposed treatment modality, there is no information available relative to the OTM of any of the teeth that have undergone these procedures.

The potential impact of sequelae following OTM on non-surgical root canal procedures or surgical endodontics has received mixed attention and more definitive studies are indicated.

The major focus of any endodontic intervention appears to be in the prevention or management of post OTM root resorption. Many publications have addressed clinical perspectives and empirical approaches to this challenge and the reader is referred to them for a more complete assessment of the issues at hand.[255] However, some recent perspectives have been gleaned on the determination of root canal working length following root resorption as a sequelae to OTM.[256] Using electronic apex locators, the accuracy of working length determination was not affected by resorptive defects in a model that simulated apical root resorption.

The earliest report dealing with the orthodontic movement of teeth with previous endodontic nonsurgical and surgical treatment is attributed to Baranowskyj.[257] In this canine study the rate of healing of the hard and soft alveolar tissues was assessed on teeth that had root fillings and previous periapical surgeries, and that were subject to an early application of orthodontic intrusive forces. Histological assessment of 6-week specimens indicated that healing was completely delayed in the teeth with root fillings and root-end resections. There was no visible attempt at bone regeneration in the surgical defect or at formation of a new periodontal ligament or cementum. The surgical defects were filled with degenerating blood clots and there was evidence of attempts at organization and infiltration with endothelial buds (angiogenesis). The control group showed almost complete healing of all tissues. Histological assessment of both groups at 12 weeks indicated regeneration of bone and periodontal ligament was complete in the control and approximately two-thirds of the experimental group. The apical cementum in the experimental group was also only one-third as complete and a mild resorptive response was evident. The early application of orthodontic forces after surgical endodontic treatment markedly delayed the healing process and the specific cause was identified as tooth mobility and its impact on the ossifying media of the supporting apical tissues.

While not definitive, another report on the orthodontic movement of root-end resected teeth is attributed to Wickwire et al.[208] In their root-treated population of 53 teeth that were moved orthodontically, four had received root-end resections. Although there was a greater incidence of root resorption with the movement of root-treated teeth, no mention was made as to the status of the root-end resected teeth.

While successful movement of teeth following surgical endodontic procedures has been observed clinically, very little has been written addressing the ramifications of the approach to treatment. Considerations would include the propensity for a greater amount of apical resorption due to

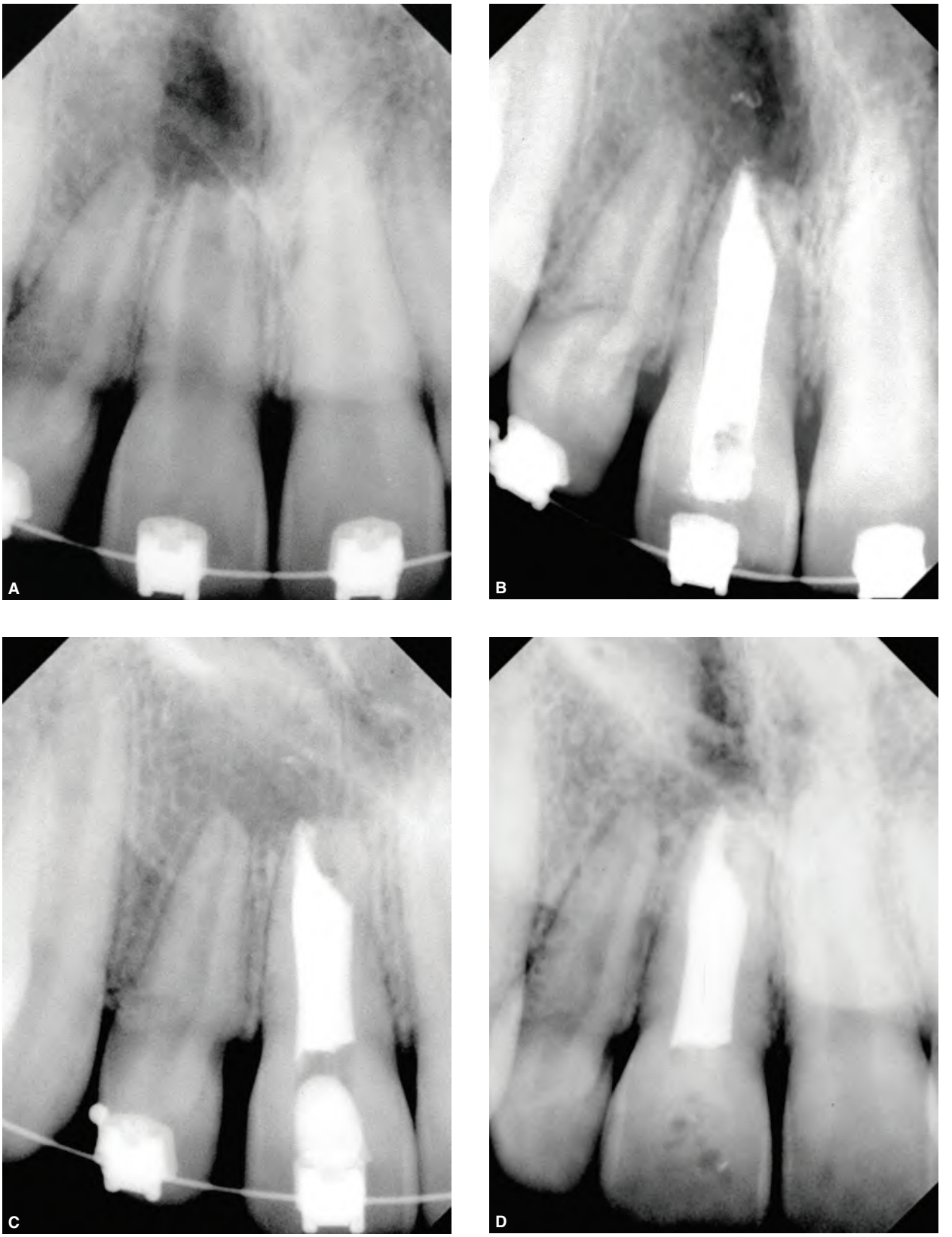

FIGURE 39-9 ***A.*** Radiograph of a maxillary right central incisor taken after OTM has begun. The 18- year-old female patient had suffered trauma at an earlier age, however no periapical radiograph was taken prior to OTM nor were the teeth tested. ***B.*** The tooth was accessed (pulp was necrotic), the canal shaped, cleaned and disinfected and filled entirely with MTA. Note, there was only a small opening apically, even though the original radiograph depicted immature root development. ***C.*** Six-month evaluation showing some osseous repair. ***D.*** Eighteen-month evaluation showing good healing. The patient is symptom-free.

the exposed dentin on the resected root face, irritation and persistent inflammation that may be caused by the root-end filling material, and the adequacy of the seal achieved with the root-end filling material. Other factors to consider are the quality of the nonsurgical root canal obturation at the level of resection, the potential for exposed, contaminated dentinal tubules at the point of resection with potential for harboring residual bacteria, endotoxins and biofilms,[202,203] and the potential for localized marginal periodontitis in those cases where a dehiscence or fenestration may be present. In this context the major causes for failure following surgical endodontic procedures have been identified as failure to debride and obturate thoroughly the root canal system, and the superimposition of periodontal disease in the surgical site.[258,259] Low levels of success with surgical endodontics have been reported also in the absence of a sound buccal cortical plate of bone.[260,261] Recently this scenario has prompted the use of guided tissue regenerative procedures to enhance surgical outcomes.[262–268] These issues must be considered in the treatment planning phase prior to tooth movement.

While there is a paucity of studies, a recent case report by Stefopoulos[269] identified a positive long-term outcome of root-end surgery combined with OTM. A 5-year recall indicated healing that could be classified as incomplete healing or scar tissue. Therefore, if surgical endodontic intervention is absolutely necessary during OTM, then the orthodontic component should be delayed for possibly up to 6 months to allow for tooth stability and significant healing of the periradicular tissues. Another consideration would be to use bone grafting material concomitant with the surgical procedure to stabilize the extracellular matrix and ossifying media forming in the surgical site.

In summary, the complete assessment of all teeth, whether in the young patient or adult, in which OTM is being considered, is essential. Thorough records, including radiographs and photographs before or during orthodontic diagnosis and treatment planning are indicated. Reassessment during the OTM and documentation of any new findings compared to the baseline information are necessary to determine if the dental pulp is undergoing adverse changes or if root resorption is present. This is especially important during OTM of previous traumatized teeth. Prevention in the endodontic-orthodontic relationships that exist, when possible, is paramount to tooth retention and distress-free function.

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Chapter 40

Tooth Discoloration and Bleaching of Non-Vital Teeth

Ilan Rotstein

Tooth discoloration is a clinical esthetic problem. Often, patients seek corrective treatment modalities to improve the appearance of their teeth, mainly in the esthetic zone. In vital teeth, bleaching is usually done extracoronally. In non-vital teeth, it is often done intracoronally; variations may exist. This chapter focuses on bleaching of non-vital teeth.

ETIOLOGY OF TOOTH DISCOLORATION

Tooth discoloration can be induced by intrinsic stains incorporated in tooth structures and extrinsic stains deposited on tooth surfaces. This can be due to patient- or dentist-related causes[1] (Table 40-1). Correct diagnosis of the etiology of discoloration is essential for treatment decision-making and outcome assessment.

Patient-Related Causes

Pulp Necrosis

Microbial, mechanical, or chemical irritation to the pulp may result in tissue necrosis. Disintegration by-products penetrate the dentinal tubules and discolor the surrounding dentin. The degree of discoloration is directly related to how long the pulp has been necrotic. The longer the discoloring compounds are present in the pulp chamber, the greater is the discoloration. This type of discoloration can often be corrected successfully with intracoronal bleaching (Figures 40-1 and 40-2).

Intrapulpal Hemorrhage

Intrapulpal hemorrhage and lysis of erythrocytes are common results of traumatic injury to a tooth. Blood disintegration products, mainly iron sulfides, flow to the pulp lumen and permeate into the dentin tubules to discolor the surrounding tissue. If the pulp becomes necrotic, the discoloration persists and usually intesifies with time. If the pulp recovers, the discoloration may be reversed, with the tooth regaining its original shade. Intracoronal bleaching is usually effective for treating this type of discoloration with high level of patient satisfaction[2-5] (Figures 40-3 and 40-4)

TABLE 40-1 Etiology of Tooth Discoloration

Patient-related causes	Dentist-related causes
Pulp necrosis	Pulp tissue remnants
Intrapulpal hemorrhage	Intracanal medicaments
Calcific metamorphosis	Endodontic obturation materials
Age	Restorative materials
Developmental defects	
Medications	

Calcific Metamorphosis

Calcific metamorphosis is seen most frequently in the anterior teeth. It is a pulpal response to trauma that is characterized by rapid deposition of hard tissue within the root canal

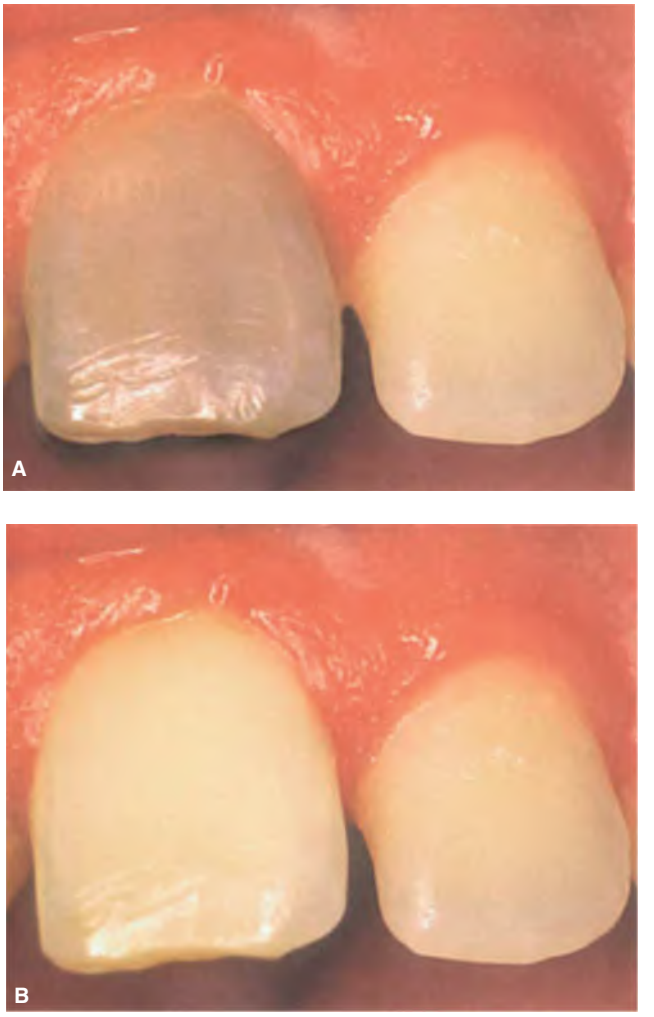
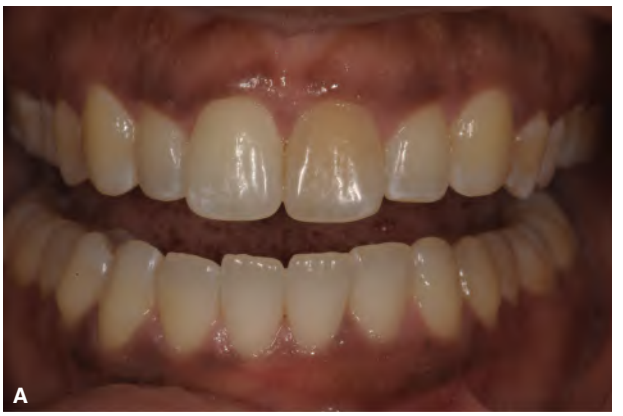
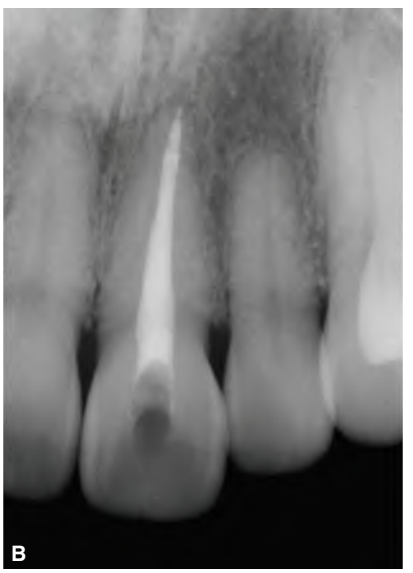


FIGURE 40-1 Discoloration of a maxillary central incisor caused by pulpal necrosis. ***A.*** Tooth shade prior to bleaching. ***B.*** A mixture of sodium perborate and distilled water, placed in the chamber two times for 3 weeks, achieved lightening of the tooth to its natural shade. (Reproduced with permission from Claisse-Crinquette A, Endo Contact, Paris, France, 1999;2:16-20.)





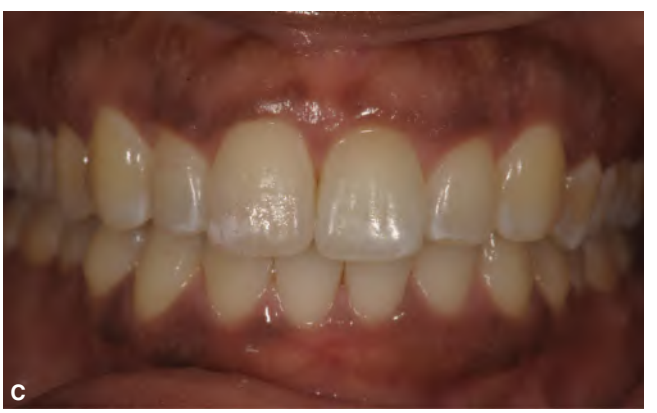
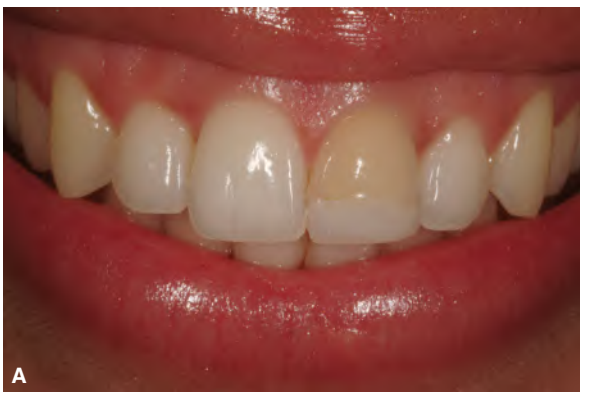
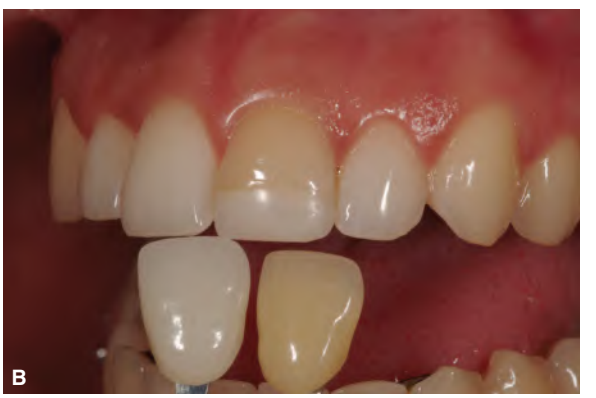


FIGURE 40-2 Intracoronal bleaching of tooth discoloration due to pulp necrosis in a maxillary left central incisor of a 40-year-old female. ***A.*** Pre-treatment photograph. ***B.*** Post endodontic treatment radiograph of the discolored tooth. ***C.*** After 4 weeks of *walking bleach* with sodium perborate, the tooth regained its original shade. (Courtesy of Dr. Abdi Sameni, Los Angeles, CA, U.S.A.)





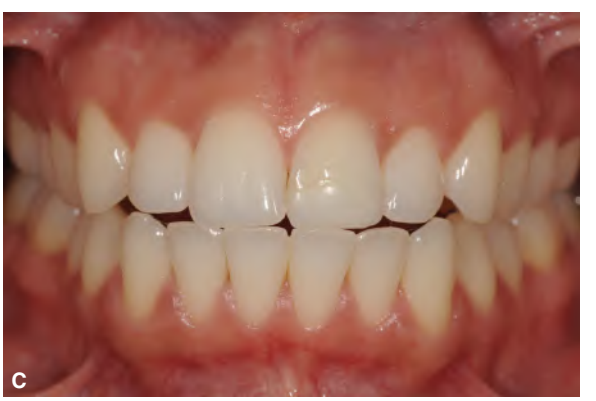


FIGURE 40-3 Intracoronal bleaching of tooth discoloration following a traumatic injury in a maxillary left central incisor of a 25-year-old female. Part of the crown was fractured 13 years previously and was restored with a bonded composite restoration. Endodontic treatment was also done. ***A.*** Pre-treatment photograph showing discolored shade of the crown in contrast to the color of the bonded restoration. ***B.*** A shade guide is used for recording purposes and treatment planning. ***C.*** After 4 weeks of *walking bleach* with sodium perborate, the discolored part of the tooth has regained its original shade and is now matching the bonded restoration. In the long term, a bonded porcelain veneer will be beneficial. (Courtesy of Dr. Abdi Sameni, Los Angeles, CA, U.S.A.)

space. In certain traumatic injuries, a temporary disruption of blood supply occurs, followed by the destruction of odontoblasts. These are replaced by undifferentiated mesenchymal cells that rapidly form reparative dentin. As a result, the translucency of the crowns of such teeth gradually decreases, giving rise to a yellowish or a yellow-brown discoloration. Root canal therapy is often required, followed by intracoronal bleaching.

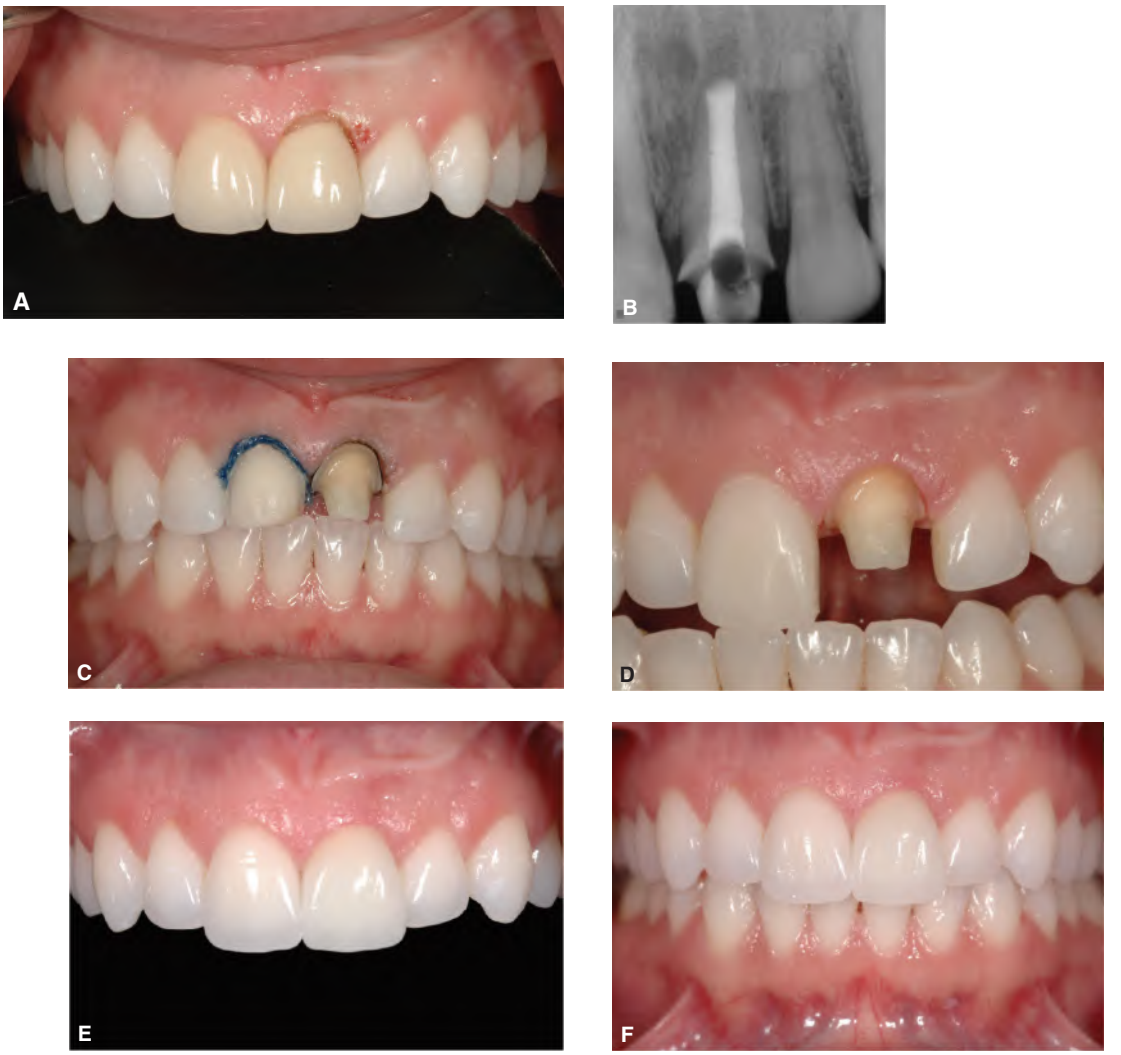


FIGURE 40-4 Intracoronal bleaching in a traumatized maxillary left central incisor of a 23-year-old female. ***A.*** Tooth discoloration is reflected through the porcelain veneer restoration. ***B.*** Periapical radiograph of the affected tooth. The patient suffered a traumatic injury 9 years previously. Endodontic treatment was done that included apexification. ***C.*** The affected tooth is prepared for a porcelain crown and the adjacent tooth for a veneer restoration. Note, dentin discoloration in the maxillary left central incisor as compared to the adjacent right central incisor. ***D.*** Intracoronal bleaching with sodium perborate significantly improved the discoloration. ***E.*** The tooth was restored to esthetics and function. ***F.*** Clinical view of the esthetic zone showing harmonious tooth shades. (Courtesy of Dr. Abdi Sameni, Los Angeles, CA, U.S.A.)

Age

Tooth color tends to become progressively darker with age, which is a physiological change resulting from dentin apposition, thinning of the enamel, and changes of optical properties in tooth structure. Cumulative extrinsic stains from food and beverages also contribute to this type of tooth discoloration that becomes more pronounced in the elderly, owing to the inevitable cracking, crazing, and incisal wear of the enamel and the underlying dentin. Unless pulp pathosis is noted, intracoronal bleaching is not indicated. In such cases, extracoronal approach should be the treatment of choice.

Developmental Defects in Tooth Structure

Discoloration may result from developmental defects occurring during enamel and dentin formation, either hypocalcific or hypoplastic. Enamel *hypocalcification*, a distinct brownish or whitish area, is commonly found on the buccal aspect of affected crowns. The enamel is well formed with an intact surface.

Enamel *hypoplasia* differs from hypocalcification in that the enamel is defective and porous. This condition may be hereditary, as in *amelogenesis imperfecta*, or *hereditary hypophosphatemia*, or a result of environmental factors such as infections, tumors, or trauma. Presumably, during enamel formation, the matrix is altered and does not mineralize properly. The defective enamel is porous and readily discolored by materials present in the oral cavity. In such cases, bleaching effect may not be permanent, depending on the severity and extent of hypoplasia and the nature of discoloration.

Dental fluorosis, or mottled tooth, is a type of enamel hypoplasia. It is caused by an excessive exposure to fluoride during tooth formation, resulting in defects in mineralized structures, particularly in the enamel matrix. The severity of subsequent staining generally depends on the degree of hypoplasia that is directly related to the amount of fluoride exposure during odontogenesis.[6] The affected teeth are not discolored on eruption, but their porous surface attracts extrinsic stains present in the oral cavity. Discoloration is usually bilaterally symmetric. It presents as various degrees of intermittent white spotting, chalky or opaque areas, yellow or brown discoloration, and, in severe cases, surface pitting of the enamel. Discoloration of mild to moderate dental fluorosis is usually bleached externally, however, restorative approaches should be considered for the cases of severe dental fluorosis.

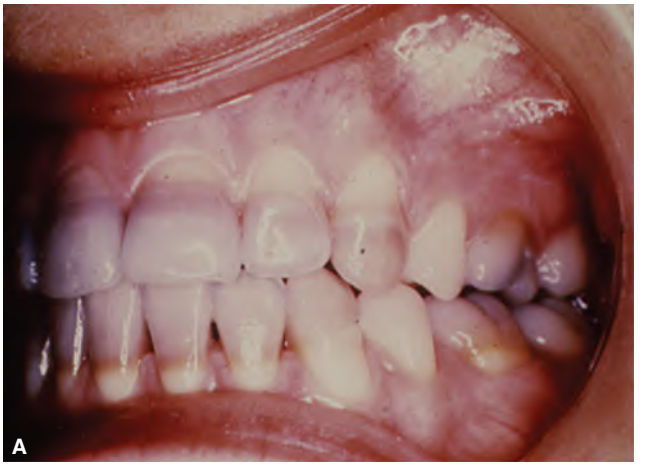
Several systemic conditions may also cause tooth discoloration. *Erythroblastosis fetalis*, for example, may occur in the fetus or in a newborn because of Rh incompatibility factors, resulting in the lysis of erythrocytes and incorporation of hemosiderin pigment in the forming dentin.[7] However, such discoloration is now uncommon. High fever during tooth formation may result in chronologic enamel hypoplasia that gives rise to banding-type surface discoloration. *Porphyria*, a metabolic disease, may also cause reddish or brownish discoloration of deciduous and permanent teeth. *Thalassemia* and sickle cell anemia may cause intrinsic blue, brown, or green discolorations. *Amelogenesis imperfecta* may result in yellow or brown discolorations. *Dentinogenesis imperfecta* causes brownish violet, yellowish, or gray discoloration. These conditions are usually not amenable to bleaching and correction by restorative means is preferred.

Discoloration Caused by Medications

Administration or ingestion of certain medications during tooth formation may cause severe discoloration both in enamel and dentin.[8-10] Tetracycline is an antibiotic that was used extensively during the 1950s and 1960s for prophylactic protection and for the treatment of chronic obstructive pulmonary disease, mycoplasma, and rickettsial infections. It was sometimes prescribed for long periods of time, years in some cases, and therefore was a common cause of tooth discoloration in children. While tetracycline is no longer prescribed for prolonged periods, dentists still face the residue of damage to teeth appearance from such medications.

Tooth shades from discoloration can be yellow, yellow-brown, brown, dark gray, or blue depending on the type of tetracycline, dosage, duration of intake, and patient's age at the time of administration. Discoloration is usually bilateral, affecting multiple teeth in both arches. Deposition of tetracycline may be continuous or in stripes depending on whether the ingestion was continuous or interrupted (Figure 40-5).

The mechanism of tetracycline discoloration is not fully understood. Tetracycline bound to calcium is thought to be incorporated into the hydroxyapatite crystal of both enamel



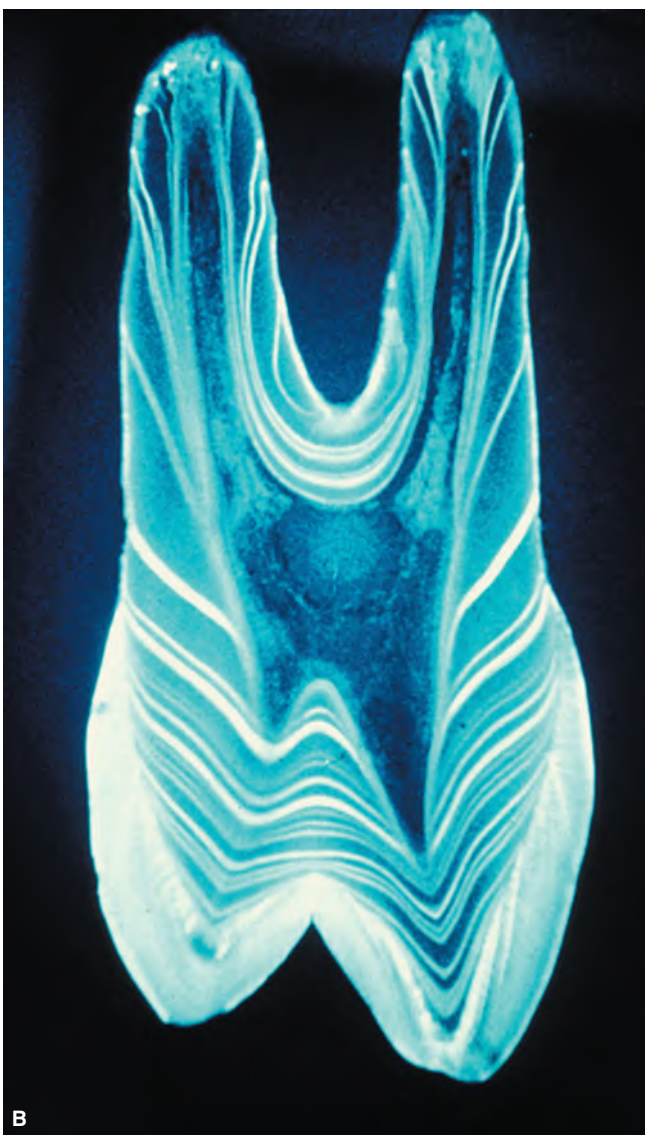


FIGURE 40-5 A AND B: Tetracycline-induced tooth discoloration. ***A.*** Clinical appearance. ***B.*** Fluorescent photomicrograph of an extracted tooth showing stripes of tetracycline deposition caused by start and stop of drug ingestion. (Image 5B, courtesy of Dr. D.L. Meyers., U.S.A.)

and dentin, but mostly found in dentin. The exposure of tetracycline-stained teeth to ultraviolet radiation leads to the formation of a reddish-purple oxidation by-product that discolors the teeth. In children, the anterior teeth often discolor first, whereas the less-exposed posterior teeth are discolored more slowly. In adults, natural photobleaching of the anterior teeth has been observed, particularly in individuals whose teeth are excessively exposed to sunlight owing to maxillary lip insufficiency.

Tetracycline discoloration has been classified into three groups according to severity.[11] First-degree discoloration is light yellow, light brown, or light gray and occurs uniformly throughout the crown, without banding. Second-degree discoloration is more intense and also without banding. Third-degree discoloration is very intense, and the clinical crown exhibits horizontal color banding. This type of discoloration usually predominates in the cervical regions. Correction of tetracycline-stained teeth may pose a clinical challenge. For mild to certain moderate cases, repeated external bleaching for an extended period can be effective.[12-14] However, severe cases, especially those of dark gray color, should be corrected by restorative means.

Dentist-Related Causes

Tooth discolorations caused by certain dental materials or inappropriate operating techniques can occur; such dentist-related discolorations are usually preventable and efforts should be made to avoid them.

Pulp Tissue Remnants

The tissue remaining in the pulp chamber disintegrates gradually and may cause discoloration. Therefore, pulp horns must always be included in the access cavity to ensure removal of pulpal remnants and to prevent retention of sealers at a later stage. Intracoronal bleaching in these cases is usually successful.

Intracanal Medicaments

Several intracanal medicaments can cause internal staining of dentin. Phenolics or iodoform-based medicaments sealed in the root canal and chamber are in direct contact with dentin, sometimes for long periods, allowing penetration and oxidation. These compounds have a tendency to discolor the dentin gradually. Intracoronal bleaching is often used to correct this problem.

Endodontic Obturation Materials

This is a frequent and severe cause of single tooth discoloration. Incomplete removal of obturating materials and sealer remnants from the pulp chamber, mainly those containing metallic components, often results in dark discoloration.[15-19] This can be easily prevented by removing all materials to a level just below the gingival margin (Figure 40-6). Intracoronal bleaching is the treatment of choice in addition to removing the obturating material. The prognosis, however, depends on the type of sealer and the duration of discoloration.

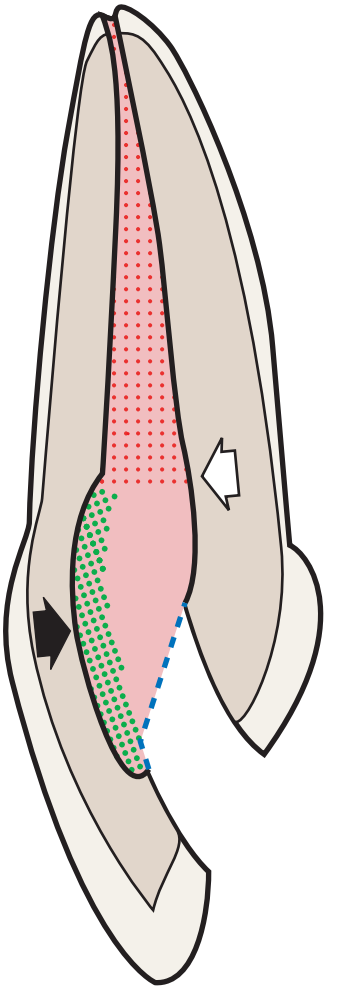


FIGURE 40-6 Gutta-percha, sealer, and dentin removal prior to bleaching. Green-dotted area represents gutta-percha filling that is removed to a level below the attached gingival (white arrow) along with the removal of dentin to eliminate heavy stain concentration and pulp horn material (black arrow).

Restorative Materials

Amalgams

Amalgams were frequently used in the past to restore lingual access preparations or developmental grooves in anterior teeth. In addition to the inherent dark, metallic color of an amalgam restoration, amalgams degrade over time and corrosion products can cause tooth discoloration. Such discolorations are difficult to bleach and tend to re-discolor with time. Amalgam restorations in premolar teeth may also be an esthetic concern in patients with wide smile line. Sometimes the dark appearance of the crown is caused by an amalgam restoration that can be seen through the tooth structure. In such cases, replacing the amalgam with an esthetic restoration usually corrects the problem.

Pins and Posts

Metal pins and prefabricated posts have sometimes been used to reinforce a composite restoration in the anterior dentition. Discoloration from inappropriately placed pins and

posts is caused by the metal seen through the composite or tooth structure. In such cases, coverage of the pins with a white cement or removal of the metal and replacement of the composite restoration is indicated.

Resin Composites

Microleakage around resin composite restorations attracts extrinsic stains. Open margins allow stains to enter the interface between the restoration and the tooth structure and discolor the underlying dentin. Resin composites may also become discolored with time, affecting the shade of the crown. These conditions are generally corrected by replacing the old composite restoration with a new, well-sealed one.

TOOTH BLEACHING

Bleaching is a treatment modality involving an oxidative chemical that alters the light-absorbing and/or light-reflecting nature of a material structure, thereby increasing its perception of whiteness. In-office tooth bleaching using peroxide compounds has been practiced in dentistry for more than a century.

Bleaching Materials

The active ingredients in tooth bleaching materials are peroxide compounds. While currently a variety of bleaching materials are available, the most commonly used peroxide compounds are sodium perborate, hydrogen peroxide, and carbamide peroxide. Bleaching materials for extracoronal bleaching mainly contain hydrogen peroxide and carbamide peroxide, whereas sodium perborate is primarily used for intracoronal bleaching.

Sodium Perborate

Sodium perborate ($NaBO_3$) is available in powdered form or as various commercial preparations. When fresh, it contains about 95% perborate, corresponding to 9.9% of the available oxygen. Sodium perborate is stable when dry. In the presence of acid, warm air, or water, however, it decomposes to form sodium metaborate, H_2O_2, and nascent oxygen. Three types of sodium perborate preparations are available: monohydrate, trihydrate, and tetrahydrate. They differ in oxygen content that determines their bleaching efficacy.[20] Commonly used sodium perborate preparations are alkaline, and their pH depends on the amount of H_2O_2 released and the residual sodium metaborate.[21] Sodium perborate is more easily controlled and is safer than concentrated H_2O_2. Therefore, it should be the material of choice in most intracoronal bleaching procedures (Figure 40-7).

Hydrogen Peroxide

Hydrogen peroxide (H_2O_2) has been used at various concentrations ranging from 3% to35%. H_2O_2 at high concentration is caustic and burns tissues on contact. These materials must be handled with care to avoid their contact with tissues during the handling and bleaching treatment. H_2O_2 is not recommended for routine intracoronal use in non-vital teeth.

FIGURE 40-7 Sodium perborate powder and water are mixed to a thick consistency of wet sand.

Carbamide Peroxide

Carbamide peroxide ($CH_6N_2O_3$), also known as urea hydrogen peroxide, exists in the form of white crystals or as a crystallized powder containing approximately 35% H_2O_2. It forms H_2O_2 and urea in aqueous solution. Concentrations ranging from 10 to 30% (equivalent to approximately 3.5% to 8.6% H_2O_2) are often used. Occasionally, higher concentrations were used in selected cases.

Bleaching preparations containing carbamide peroxide usually also include glycerine or propylene glycol, sodium stannate, phosphoric or citric acid, and flavor additives. In some preparations, carbopol, a water-soluble polyacrylic acid polymer, is added as a thickening agent. Carbopol also prolongs the release of active peroxide and improves shelf life. Carbamide peroxide is mainly used for extracoronal bleaching. Nevertheless, it has been used successfully, either alone or in combination with another agent, for intracoronal bleaching of non-vital teeth.[19,22,23]

Bleaching Mechanisms

The exact mechanisms of tooth bleaching have not yet been fully elucidated; however, it is generally believed that free radicals produced by H_2O_2 may be responsible for bleaching effects, and they are similar to that in textile and paper bleaching. H_2O_2 diffuses through the enamel and dentin, producing free radicals that react with pigment molecules breaking

their double bonds. The change in pigment molecule configuration and/or size may result in changes in their optical properties, and consequently, the perception of a lighter color by human eyes. This assumption also helps to explain the common observation of shade rebounding shortly after the bleaching treatment, probably due to the reformation of double bonds. In addition to the chemical effect, other possible mechanisms include cleansing of tooth surface, temporary dehydration of enamel during the bleaching process, and change of enamel surface.

A number of factors, relating to both the patient (e.g., age, gender, and initial tooth color), the bleaching material used (e.g., type of peroxide compound, peroxide concentration, other ingredients), and application method (e.g., contact time, application frequency) may contribute to the bleaching efficacy and the subsequent stability of the esthetic results.

Intracoronal Bleaching of Endodontically Treated Teeth

Intracoronal bleaching of endodontically treated teeth may be successfully carried out with satisfying long-term esthetic results (Figure 40-8). A successful outcome depends mainly on the etiology, correct diagnosis, and proper selection of bleaching technique[1,24,25] (Table 40-2). The methods most commonly employed to bleach endodontically treated teeth are the "walking bleach" and the thermocatalytic techniques. *Walking bleach* is preferred since it is safer, more comfortable for the patient, and requires less chair time.[26-29]

Walking Bleach Technique

The term *walking bleach* was first coined by Nutting and Poe[30] referring to the bleaching action occurring between patients' visits. Since that time, the technique evolved and underwent few modifications, mainly by eliminating the use of Superoxol (30% H_2O_2) making it a very popular and safe technique.[29,31,32] The *walking bleach* technique should be attempted first in all cases requiring intracoronal bleaching. It involves the following steps:

1. Familiarize the patient with the possible causes of discoloration, the procedure to be followed, the expected outcome, and the possibility of future rediscoloration.

TABLE 40-2 Indications and Contraindications for Bleaching Endodontically Treated Teeth

Indications	Contraindications
• Discolorations of pulp chamber origin • Dentin discolorations • Discolorations not amenable to extracoronal bleaching	• Superficial enamel discolorations • Defective enamel formation • Severe dentin loss • Presence of caries • Discolored composites

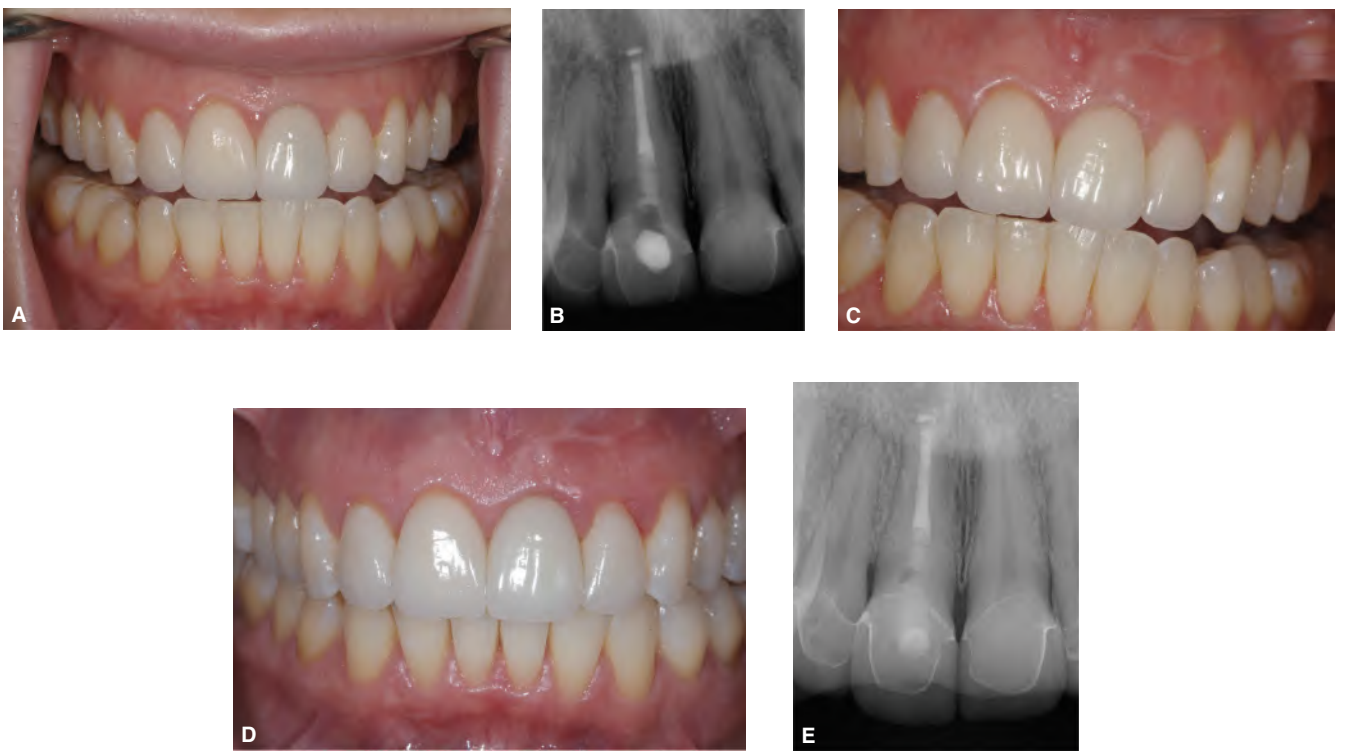


FIGURE 40-8 Intracoronal bleaching of an endodontically treated maxillary left central incisor in a 30-year-old female. ***A.*** Discoloration is reflected through the porcelain veneer restoration. ***B.*** Periapical radiograph of the affected tooth. Intracoronal bleaching was done after completion of the endodontic treatment. A paste of sodium perborate mixed with water was sealed in the pulp chamber. ***C.*** *Walking bleach* improved the shade of the porcelain veneer. ***D.*** Three-year follow up photograph showing esthetic results. ***E.*** Three-year follow up radiograph. (Courtesy of Dr. Abdi. Sameni, Los Angeles, CA, U.S.A.)

2. Radiographically assess the status of the periapical tissues and the quality of endodontic obturation. Endodontic failure or questionable obturation should always be retreated prior to bleaching.
3. Assess the quality and shade of any restoration present and replace it if defective. Tooth discoloration is frequently the result of leaking or discolored restorations. In such cases, cleaning the pulp chamber and replacing the defective restorations will usually suffice.
4. Evaluate tooth color with a shade guide and, if possible, take clinical photographs at the beginning of and throughout the procedure. These provide a point of reference for future comparison.
5. Isolate the tooth with a dental dam. The dam must fit tightly at the cervical margin of the tooth to prevent possible leakage of the bleaching agent onto the gingival tissue. Interproximal wedges and ligatures may also be used for better isolation.
6. Remove all restorative materials from the access cavity, expose the dentin, and refine the access. Verify that the pulp horns and other areas containing the pulp tissue are clean.
7. Remove all materials to a level just below the labial–gingival margin. Orange solvent, chloroform, or xylene on a cotton pellet may be used to completely dissolve sealer remnants. Etching the dentin with phosphoric acid is unnecessary and may not improve the prognosis.[33]
8. To cover the endodontic obturation, apply a sufficiently thick layer, at least 2 mm, of a protective white cement barrier, such as polycarboxylate cement, zinc phosphate cement, glass ionomer, intermediate restorative material (IRM) (Dentsply/ Caulk, York, PA, U.S.A.), white colored MTA (Dentsply/Tulsa Dental, Tulsa, OK, U.S.A.), or Cavit (3M ESPE, St. Paul, MN, U.S.A.). The coronal height of the barrier should protect the dentinal tubules and conform to the external epithelial attachment.[34]
9. Prepare the *walking bleach* paste by mixing sodium perborate and an inert liquid, such as water, saline, or anesthetic solution, to a thick consistency of wet sand. Although sodium perborate plus 30% H_2O_2 mixture may bleach faster, in most cases, long-term results are similar to those with sodium perborate and water alone and therefore need not be used routinely.[3,4,31,32] With a plastic instrument, pack the pulp chamber with the paste. Remove excess liquid by tamping with a cotton pellet. This also compresses and pushes the paste into all areas of the pulp chamber.
10. Remove the excess bleaching paste from undercuts in the pulp horn and gingival area and apply a thick well-sealed temporary filling (preferably IRM) directly against the paste and into the undercuts. To ensure a good seal, carefully pack the temporary filling, at least 3 mm thick.
11. Remove the dental dam and inform the patient that bleaching agents work slowly and that significant lightening may not be evident for several days.
12. Evaluate the patient 2 weeks later and, if necessary, repeat the procedure several times.[29,31] Repeat treatments are similar to the first one.
13. As an optional procedure, if initial bleaching is not satisfactory, strengthen the walking bleach paste by mixing sodium perborate with gradually increasing concentrations of H_2O_2 (3% to 30%) instead of water. The more potent oxidizers may have an enhanced bleaching effect but are not used routinely because of the possibility of permeation into the tubules and damage to the cervical periodontium by these more caustic agents. In such cases, a protective cream, such as Orabase or Vaseline, must be applied to the surrounding gingival tissues prior to dam placement.
14. In most cases, discoloration will improve after one to two treatments. If after three attempts there is no significant improvement, reassess the case for correct diagnosis of the etiology of discoloration and treatment plan.

Thermocatalytic

This technique involves placement of the oxidizing chemical, generally H_2O_2, into the pulp chamber followed by heat application either by electric heating devices or specially designed lamps.[35] Care must be taken when using these heating devices to avoid overheating the teeth and the surrounding tissues. Intermittent treatment with cooling breaks is preferred over a continuous session. In addition, surrounding soft tissues should be protected with Vaseline, Orabase, or cocoa butter during treatment to avoid heat damage.

Potential damage by the thermocatalytic approach is external cervical root resorption caused by irritation to the cementum and the periodontal ligament (Figure 40-9). This is possibly attributable to the oxidizing agent combined with heating.[36,37] Therefore, application of highly concentrated H_2O_2 and heat during intracoronal bleaching is questionable and should not be carried out routinely.

Ultraviolet Photooxidation

This technique applies ultraviolet light (instead of a bleaching lamp) to the labial surface of the tooth to be bleached. A H_2O_2 solution is placed in the pulp chamber on a cotton pellet followed by a 2-minute exposure to ultraviolet light. Supposedly, this causes oxygen release, like the thermocatalytic bleaching technique.[38]

Potential Adverse Effects from Intracoronal Bleaching

Chemical Burns

High concentrations of H_2O_2 are caustic and cause chemical burns and sloughing of the gingiva. When using such solutions, the H_2O_2 concentration should be maintained as low as practical and the soft tissues should always be protected with Vaseline, Orabase, or cocoa butter. H_2O_2 at high concentration is not the agent of choice for routine intracoronal bleaching.

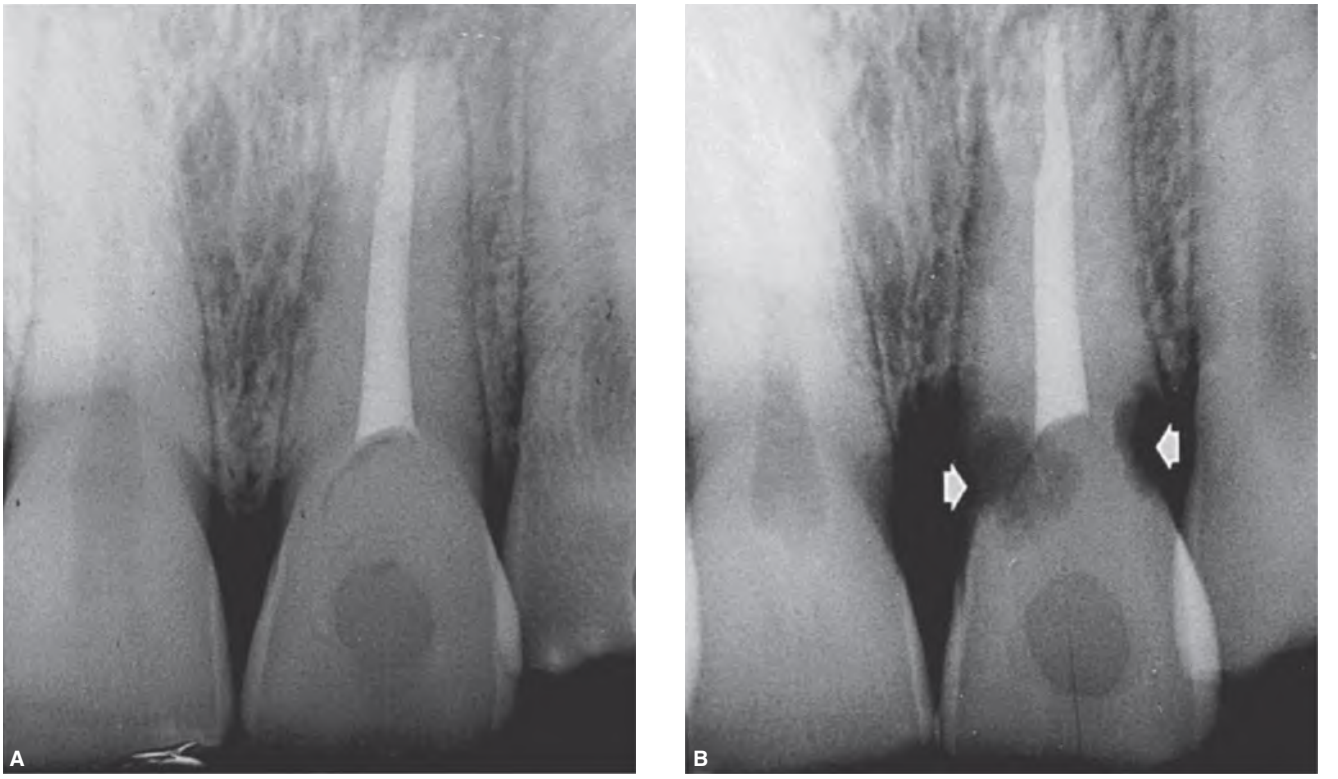


FIGURE 40-9 External root resorption following thermocatalytic bleaching. ***A.*** Nine-year recall radiograph of a maxillary central incisor devitalized by trauma and treated endodontically shortly thereafter. ***B.*** Radiograph taken 2 years following bleaching showing evidence of root resorption (arrows). Highly concentrated H_2O_2 and heat were used as part of the treatment. (Reproduced with permission from Harrington GW, Natkin E. *J Endod*. 1979;5:344-348.[44])

Inhibition on Resin Polymerization and Bonding Strength

Oxygen inhibits resin polymerization; consequently, residual H_2O_2 in tooth structure after bleaching adversely affects the bonding strength of resin composites to enamel and dentin.[39,40] Scanning electron microscopy (SEM) examination has shown an increase in resin porosity.[41] This presents a clinical problem when immediate esthetic restoration of the bleached tooth is required. It is therefore recommended that residual H_2O_2 be totally eliminated prior to composite placement. One study reported that 3 minutes of catalase treatment effectively removed all of the residual H_2O_2 from the pulp chamber of human teeth.[42]

External Root Resorption

Clinical reports[43-53] and histological studies[36,37,54] have shown that intracoronal bleaching may, under certain conditions, induce external cervical root resorption. This is probably caused by the highly concentrated oxidizing agent used in those cases, particularly 30 to 35% H_2O_2. The mechanism of bleaching-induced damage to the periodontium or the cementum is not fully clear. Presumably, the irritating chemical diffuses via unprotected dentinal tubules and cementum defects.[55-57] This causes necrosis of the cementum, inflammation of the periodontal ligament, and, subsequently, root resorption. The process may be enhanced if heat is applied[58] or in the presence of bacteria.[59] Previous traumatic injury and age may act as predisposing factors.[43] (Figure 40-9).

Potential complications can be prevented if safety precautions are carried out during bleaching treatment. Also, routine monitoring of bleaching cases allows early detection of any unwarranted results and reduces the risk of potential complications.

Suggestions for Safe Bleaching of Endodontically Treated Teeth

- Isolate the tooth effectively. Intracoronal bleaching should always be carried out under dental dam isolation. Interproximal wedges and ligatures may also be used for better protection.
- Protect the oral mucosa. Protective creams, such as Orabase, Vaseline, or cocoa butter, must be applied to the surrounding oral mucosa to prevent chemical burns by caustic oxidizers. Animal studies suggested that catalase applied to oral tissues prior to bleaching treatment totally prevents the associated tissue damage.[60]
- Verify adequate endodontic obturation. The quality of root canal obturation should always be assessed clinically and radiographically prior to bleaching. Adequate obturation ensures a better overall prognosis of the treated tooth. It also provides an additional barrier against damage by oxidizers to the periodontal ligament and periapical tissues.

- Use protective barriers over the coronal extent of the root canal filling. This is essential to prevent leakage of bleaching agents that may infiltrate between gutta-percha and root canal walls, reaching the periodontal ligament via dentinal tubules, lateral canals, or the root apex. In none of the clinical reports of postbleaching root resorption was a protective barrier used. Various materials can be used for this purpose.[61,62] Barrier thickness and its relationship to the cementoenamel junction are most important.[34,61] The ideal barrier should protect the dentinal tubules and conform to the external epithelial attachment (Figure 40-10).
- Avoid acid etching. It has been suggested that acid etching of dentin in the chamber to remove the smear layer and open the tubules would allow better penetration of the oxidizer. This procedure has not proved beneficial.[33] The use of caustic chemicals in the pulp chamber is undesirable as they may result in irritation to the periodontal ligament.

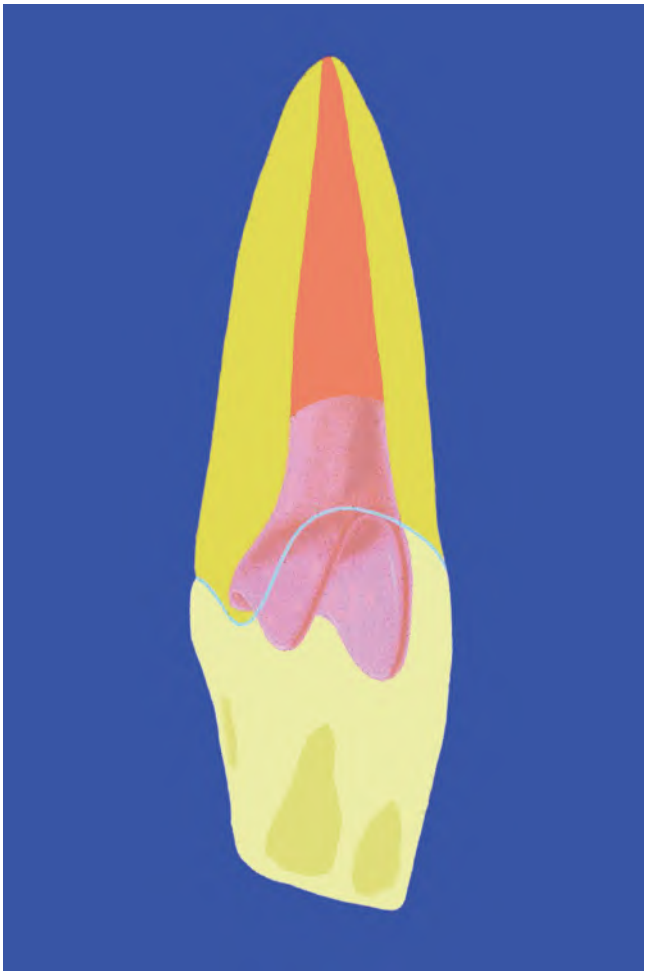


FIGURE 40-10 Schematic illustration of the intracoronal protective bleach barrier. The shape of the barrier matches the contour of the external epithelial attachment. (Reproduced with permission from Steiner DR, West JD. *J Endod*. 1994; 20:304–306.[35])

- Avoid strong oxidizers. Procedures and techniques applying strong oxidizers should be avoided if they are not essential for bleaching. Solutions of 30% to 35% H_2O_2, either alone or in combination with other agents, should not be used routinely for intracoronal bleaching. Sodium perborate is mild and quite safe, and usually no additional protection of the soft tissues is required.[63,64] Generally, however, oxidizing agents should not be exposed to more of the pulp space and dentin than absolutely necessary to obtain a satisfactory clinical result.
- Avoid heat. Excessive heat may damage the cementum and the periodontal ligament as well as dentin and enamel, especially when combined with strong oxidizers.[36,37] Although no direct correlation has been established between heat application and external cervical root resorption, it should be limited during bleaching procedures.
- Recall periodically. Bleached teeth should be frequently examined both clinically and radiographically. Early detection of an unusual occurrence can improve the prognosis since corrective therapy may still be applied.

Restoration of Intracoronally Bleached Teeth

Proper tooth restoration is essential for long-term successful bleaching results. Microleakage of lingual access restorations is a problem,[65] and a leaky restoration may again lead to contamination and tooth discoloration.

The pulp chamber and the access cavity should be carefully restored with a light shade, light-cured, acid-etched composite resin. The composite material should be placed at a depth that seals the cavity and provides some incisal support. Light curing from the labial surface, rather than the lingual surface, is recommended since this results in the shrinkage of the composite resin toward the axial walls, reducing the rate of microleakage.[66] Placing white cement beneath the composite access restoration is recommended. Filling the chamber completely with composite may cause loss of translucency and difficulty in distinguishing between composite and tooth structure during rebleaching.[67]

Discolored tooth structure may reflect through porcelain crowns and veneers thereby compromising the final esthetic result. Intracoronal bleaching, prior to placement of bonded porcelain crowns or veneers, is advantageous (Figure 40-4).

As stated previously, residual H_2O_2 from bleaching treatment may adversely affect the bonding strength of composites.[39] Therefore, waiting for at least 7 days after bleaching, prior to restoring the tooth with resin composites, has been recommended. Catalase treatment at the final visit may enhance the removal of residual peroxides from the access cavity; however, this requires further clinical investigation.[42] Packing calcium hydroxide paste in the pulp chamber for a few weeks prior to the placement of final restoration, to counteract acidity caused by bleaching agents and to prevent root resorption, has also been suggested; this procedure, however, is unnecessary with walking bleach.[21]

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