MICHAEL G. NEWMAN | SATHEESH ELANGOVAN
IRINA F. DRAGAN | ARCHANA K. KARAN

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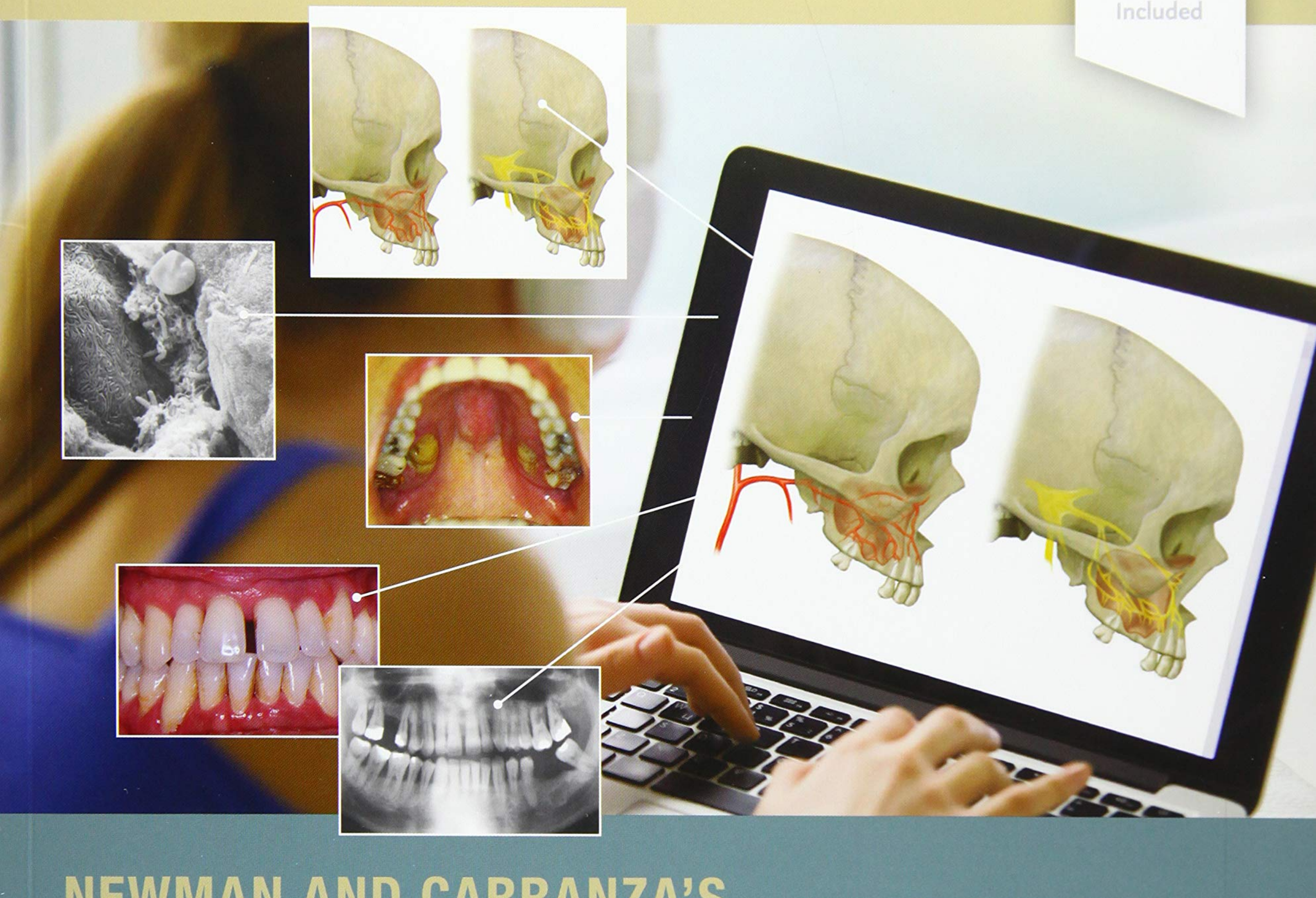

NEWMAN AND CARRANZA'S

Essentials of Clinical Periodontology

AN INTEGRATED STUDY COMPANION

ELSEVIER

Newman and Carranza's Essentials of Clinical Periodontology

An Integrated Study Companion

Michael G. Newman, DDS, FACD

Professor Emeritus
Section of Periodontics, School of Dentistry, University of California, Los Angeles, California

Satheesh Elangovan, BDS, DSc, DMSc

Professor
Department of Periodontics, The University of Iowa College of Dentistry, Iowa City, Iowa

Irina F. Dragan, DDS, DMD, MS

Assistant Professor and Director of Faculty Education & Instructional Development
Department of Periodontology, Tufts University School of Dental Medicine, Boston, Massachusetts

Archana K. Karan, MDS

Periodontist
Private Practice, Chennai, India

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Preface

With the help of Elsevier's advanced technology and high standards of quality, an international team of editors and contributors have developed *Newman and Carranza's Essentials of Clinical Periodontology*, the first edition companion guide for *Newman and Carranza's Clinical Periodontology* 13th Edition (NC13) textbook. The main objective of this endeavor is to develop an exam-centric factual companion guide that complements and also supplements the corresponding content in NC13 textbook. Keeping the text content minimal (restricting only to essential facts) and delivering the core information using easy understandable visual aids in the form of illustrations, tables, figures and infographics are the hallmarks of this companion guide.

There are five major features in each chapter of this guide:

- **Relevant terminology** and **fast facts** in each chapter offer students important terminologies, key facts and take-home messages
- **Core knowledge** feature delivers the central and fundamental information from the chapters of NC13 textbook in a succinct manner using visual aids such as tables, illustrations, figures or infographics.
- Interspersed within core knowledge are **'basic or clinical correlate' callout boxes** to underscore the clinical relevance of information in basic science chapters and vice versa.
- **Case-based learning exercises** to allow students to apply the knowledge gained from other features in a relevant clinical scenario.

The multifaceted, complex task of producing NC13, the main source for this companion guide required the collaboration of numerous experts from various fields, and their contributions are invaluable. We know that this new companion guide for NC13 will be a valuable source of both students and practitioners in dental and allied fields around the world.

Michael G. Newman
Satheesh Elangovan
Archana K. Karan
Irina F. Dragan

Acknowledgments

First and foremost, the editors of this companion guide thank all the editors and contributors of *Newman and Carranza's Clinical Periodontology*, 13th Edition (NC13), the textbook that is the primary source of information for this companion guide. It is certain that the task of researching, preparing, and assembling the enormous amount of periodontology-related content necessary for creating NC13 had to be borne by many experts who shared their experience and knowledge. We express our deep gratitude to all those contributors whose expertise, ideas, and efforts built that valuable resource, which this companion guide supplements and complements.

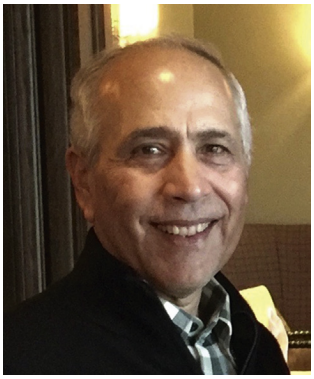
NC13 has been a trusted and valuable periodontics resource for students, residents, academicians, scientists, and clinicians since the early 1950s. Dr. Michael G. Newman, one of the senior editors of NC13, is also one of the editors of this guide. We would like to thank all the other senior editors affiliated with NC13, including Drs. Fermin A. Carranza, Henry H. Takei, and Perry R. Klokkevold.

The level of understanding and the practice of clinical periodontics have evolved tremendously since the mid-20th century. Advances in basic science and clinical techniques have increased the knowledge base so dramatically that it is virtually impossible for individuals to master and retain all the information. The main objective of producing *Essentials of Clinical Periodontology* was to develop an exam-centric factual companion guide that complements and supplements the corresponding content in NC13.

Drs. Newman and Elangovan express their appreciation to their coeditors, Drs. Irina Dragan and Archana Karan, for their constant involvement and significant contributions to this project since its conceptualization stage; our special thanks to Dr. Karan for spending countless hours in drafting infographics for the core knowledge feature. Special thanks also to the following contributors from Tufts University School of Dental Medicine: Drs. Noshir Mehta, Samar Shaikh, Kai Lei, Pooyan Refahi, Gayathri Shenoy, Sarah Almeshred, Lauren Marzouca, Jared Wirth, and Charles Hawley.

Our appreciation is also given to Elsevier and particularly to Alexandra Mortimer, Joslyn Dumas, and Erika Ninsin. Their expertise and detailed attention to every word and every concept contributed greatly to producing a quality book and a truly useful website. The online version of the book continues to assume greater importance to our readers. Elsevier's electronic capabilities provide a rich, useful, and complete resource.

We express gratitude to our parents, our family members, colleagues, friends, and mentors, who have always been so tolerant, encouraging, and understanding and who guided our first steps in our profession and helped us develop our ideas in the field.



Michael G. Newman

Satheesh Elangovan

Archana Karan

Irina F. Dragan

1 Evidence-Based Clinical Practice

Relevant Terminology

Terminology / Abbreviation	Explanation
blinding	The process by which allocation of intervention(s) is concealed to one or many individuals involved in a clinical study. If it is concealed only to the study participant, it is called a single blinded study, whereas in double and triple blinded studies, the allocation of intervention is concealed to two and three individuals in the research team, respectively
case-control study	Individuals with the primary endpoint of interest (cases) are compared with individuals without the primary endpoint of interest (control), to identify the exposure. Conducting case-control studies is highly challenging due to the inherent bias involved in selecting cases and controls
cohort study	Individuals subjected to a specific exposure are monitored longitudinally and compared with nonexposed individuals for the occurrence of the primary endpoint of interest
confounders	In studies exploring the association between an exposure and an endpoint, it is important to take into consideration the variable(s) related to the exposure (i.e., not necessarily causal) and causally associated with the endpoint. These variables are called confounders, for they can mask the real effect of the exposure on the endpoint. Example: smoking is a confounder in the association between periodontitis and cardiovascular disease outcomes
evidence	Synthesis of all valid research conducted earlier that answers a specific PICO question
exposure and endpoint	Exposure is a specific etiologic factor or intervention (e.g., treatment). Endpoint is an outcome of a disease or an intervention
external versus internal validity	External validity refers to how well the findings from a study can be applied outside the context of that study. Internal validity refers to how well a study is carried out (especially in avoiding confounders). The better the confounders are controlled in a study, the higher its internal validity
PICO format	The question that is formulated (the first step in evidence-based dentistry) should be simple and specific to the clinical scenario. It should contain information on the following key components: problem or population (P), intervention (I), comparison group (C), and outcomes (O), and hence is termed a PICO question
randomization methods	Study participants are randomized in RCTs using a variety of methods, including coin toss and computerized programs
randomized clinical trial (RCT)	A clinical study design for testing the efficacy of interventions, in which the research participants are randomized (by established methods) into two or more arms, in an effort to minimize bias[1]
temporality	In studies looking into causality, it is extremely important to establish that the cause preceded the effect; this criterion is called temporality
true versus surrogate endpoints	True or tangible endpoints directly reflect how a patient feels, functions, or survives. Surrogate or intangible endpoints are substitutes for true endpoints. Tooth loss and changes in probing depth measure are examples of true and surrogate endpoints, respectively

Fast Facts

Components of evidence-based dentistry	Patient values/preferences, clinical experience/judgment and scientific evidence
Evidence-based clinical decision-making	Decision-making performed in a clinical setting for a given clinical scenario that takes into consideration patient values/preferences, clinical experience/judgment, and scientific evidence[2]
Steps in evidence-based clinical decision-making	1. Formulating a clinical question to be answered 2. Searching for and acquiring the evidence 3. Appraising (assessing the quality) the evidence 4. Applying the evidence in a given clinical scenario 5. Evaluating the outcomes[3]
Advantages of evidence-based dentistry	Efficient way for clinicians to stay current Maximizes potential for successful clinical outcomes
Evidence quality	Depending on the design and the inherent bias in a study or a group of studies from which the evidence is derived, the evidence quality/level can range from low to high
Randomized controlled trial	For clinical studies testing an intervention, properly designed and conducted randomized controlled trials will yield high-quality evidence with minimal bias
Research design types	Randomized controlled trials, case-control, cohort, preclinical (animal), case series, and case reports
Sources of evidence	Primary: evidence derived from original research studies and publications Secondary: evidence derived from combination of multiple original studies
High levels of clinical evidence	Clinical practice guidelines represent the highest level of clinical evidence. Meta-analysis and systematic reviews that combine evidence from multiple individual clinical studies come second in the hierarchy of levels of clinical evidence, and are examples of secondary sources of evidence
Low levels of clinical evidence	Evidence derived from case reports, case series, or expert opinions
Systematic review versus meta-analysis	Systematic reviews are predominantly qualitative, whereas meta-analysis is quantitative in nature. Both identify and combine carefully selected studies to answer a specific research question. Meta-analysis is usually presented as a component of a systematic review[4]
Key advantage of systematic reviews and meta-analysis	They combine multiple previously published individual studies and include data from all the subjects of these studies, thus the effective sample size (power of study) increases significantly

Core Knowledge

Introduction

Numerous resources exist for clinicians to access information relevant to everyday clinical practice. Care providers must hence possess the skills necessary for cultivating an ability to evaluate information they read or hear about. These evaluative skills:

- Are as important as learning the clinical procedures themselves
- Must help in a lifelong learning process that allows the busy clinician to find and filter relevant, credible, and updated information for quick integration into treatment plans

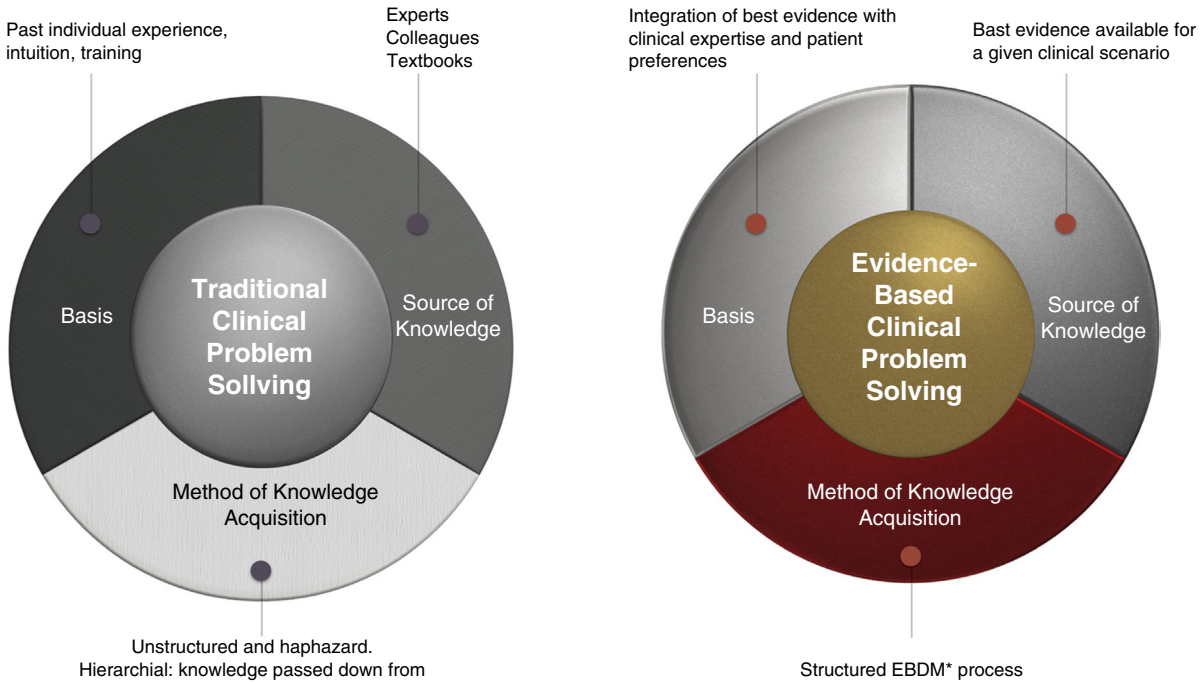
Principles of Evidence-Based Decision-Making

There is a difference between traditional clinical problem-solving and problem-solving based on best evidence. The clinical reasoning process varies in the two approaches. While traditionally one makes clinical decisions mainly using intuition, individual experience, and knowledge from colleagues and textbooks, evidence-based decision-making (EBDM) is a formalized process that allows a clinician to search for the best current scientific evidence that can be integrated quickly into practice (Fig. 1.1).

Evidence alone is insufficient to make correct clinical decisions. Without due consideration for a clinician's individual expertise and patients' inputs or circumstances, it would be unwise to blindly follow search results of best evidence. The process of EBDM is based on a few main principles (Fig. 1.2) or components that are well integrated in its flow, allowing for the successful addition of best scientific evidence as an important dimension to traditional clinical decision-making.

Sources and Levels of Evidence

Special core competencies need to be developed for critical thinking, problem-solving, and lifelong learning. The EBDM process is conceived in a structured manner to allow for developing these skills. Before the actual process of EBDM is learned, one must be aware of the sources of evidence (Table 1.1).



• **Fig 1.1** Traditional Versus Evidence-Based Clinical Problem-Solving. The difference in the two approaches for clinical problem-solving lies in the reasoning process. Traditionally, solving clinical problems relied heavily on subjective reasoning based mostly on experience, intuition, and expert opinion. In evidence-based clinical problem-solving, the approach is more objective due to a structured, formal process of asking the right questions that filter search results and help obtain relevant, updated evidence. *EBDM, evidence-based decision-making.

CLINICAL CORRELATION

Why is it important for a clinician to practice evidence-based decision-making?

While there are many ways to manage a particular clinical problem, it is important for a clinician to be aware of the best possible treatment modality for that particular scenario. Being informed involves certain skill in having the ability to search for, filter, obtain and apply good scientific evidence in a clinical scenario. The process of EBDM is important to achieve this level of clinical competence.

Hierarchies exist among types of experimental and observational studies and their quality, to guide clinical decision-making. The quality/level of evidence is directly related to the type of clinical question asked. For example, clinical questions on *therapy* would consider clinical practice guidelines (CPGs) based on meta-analyses and systematic reviews of RCT studies as the highest levels of evidence, while a clinical question on *prognosis* would give a higher ranking to CPGs based on meta-analyses and systematic reviews of cohort studies.

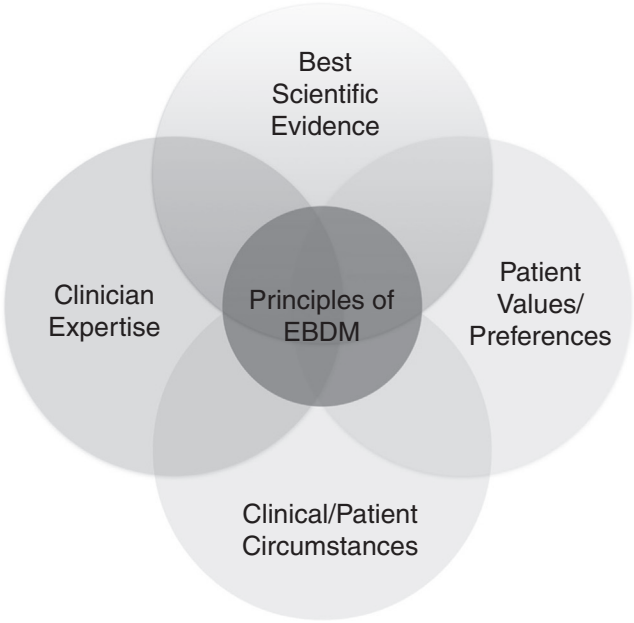
One must know the types of studies that constitute the highest levels of evidence in order to be able to apply filters for efficient searching and retrieval of best evidence (Fig. 1.3).

EBDM Process and Skills

Due to the rapid advances made, today's clinicians must develop critical appraisal skills to identify valid and useful information that can help with treatment planning and patient management. The formalized EBDM process is structured to undertake this daunting task with maximum efficiency.

The EBDM process involves five steps (Fig. 1.4):

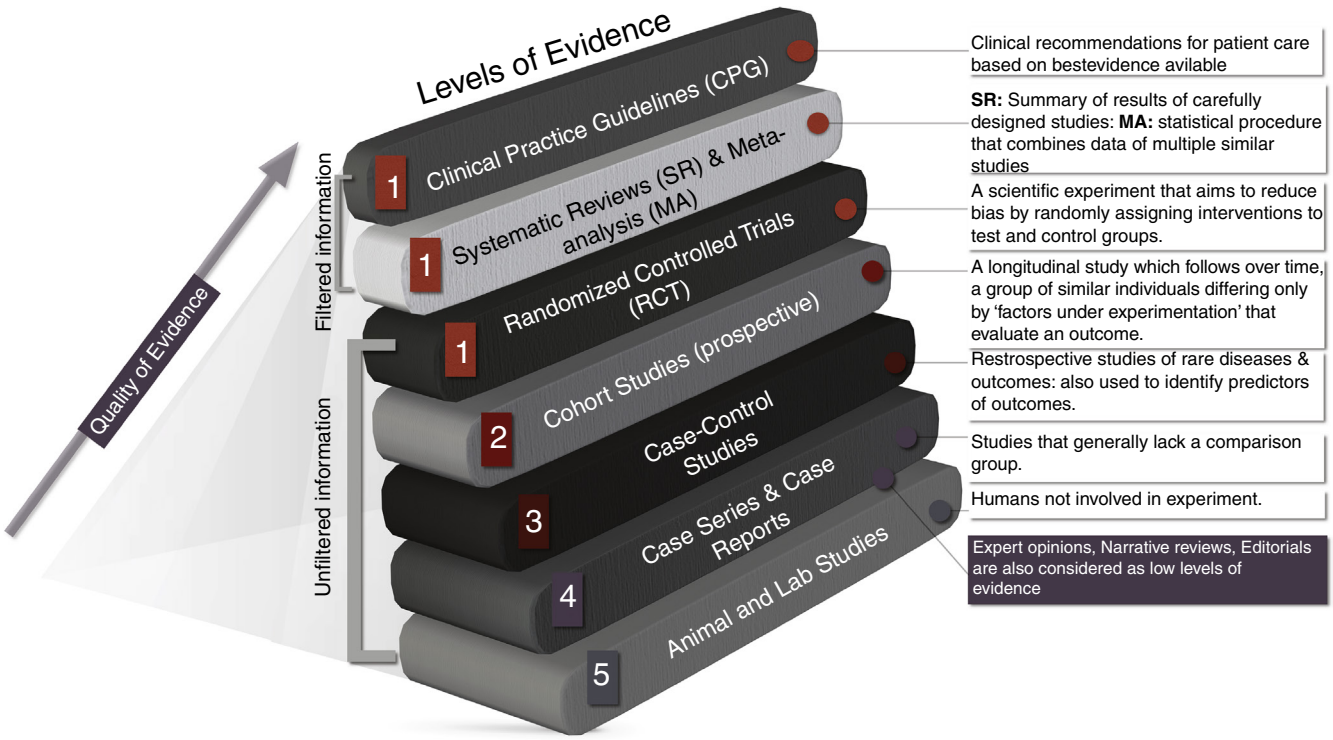
1. **Ask**: Asking the right question follows PICO format, which requires defining four components to a clinical problem (Problem/Population, Intervention, Comparison, and Outcome). This is important for:
   - Forcing the clinician to identify the single most important outcome the search should be focused on
   - Identifying the keywords required for step 2 of the process
2. **Acquire**: Filtered and unfiltered information can be found in biomedical databases like PubMed, EMBASE, DARE, and NCG. For example, using PICO terms typed into PubMed's MeSH (Medical Subject Heading) database combined with Boolean operators like AND and OR, one can search efficiently for relevant literature. PubMed's "Clinical Queries" feature also helps to quickly pinpoint relevant citations for the question posed.



• **Fig 1.2** Principles of Evidence-Based Decision-Making. Evidence-based decision-making involves incorporating all the following principles for a holistic approach to solving clinical problems: best scientific evidence, clinician experience and judgment, patient values and preferences, and clinical/ patient circumstances (American Dental Association Center for Evidence-Based Dentistry)[2].

TABLE 1.1 Sources of Evidence

Primary Sources	Secondary Sources
Original peer-reviewed research studies and publications	Valid studies and publications put together to synthesize and generate clinically applicable information
Test of efficacy	Test of effectiveness
Randomized controlled trials (RCT), cohort studies	Clinical practice guidelines (CPG), systematic reviews (SR), meta-analysis (MA)
Exercise caution in relying solely on primary sources for clinical decisions	These are more reliable sources on which to base treatment plans because they stand for higher levels of evidence



• **Fig 1.3** Levels of Evidence. The figure represents the different types of study designs and their levels of evidence that guide clinical decisions. Each level contributes to the total body of knowledge. As we progress up the pyramid, the amount of literature and the risk of bias decrease significantly, while the relevance increases tremendously. Filtered information: these levels represent secondary sources such as critical summaries/analyses and practice recommendations based on primary sources of evidence. Unfiltered information: these levels represent primary sources, such as articles in peer-reviewed journals, that show evidence regarding a topic under investigation.[5]

3. **Appraise**: Critically appraising all the evidence collected is a skill learned with time. Checklists and forms exist to help with this step of EBDM, guiding users through a structured series of Yes/No questions. Some common appraisal tools used are:
   - Consolidated Standards of Reporting Trials (CONSORT) statements for reviewing RCTs
   - Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) for reviewing SRs
   - Critical Appraisal Skills Program (CASP) for reviewing other types of studies, including RCTs and SRs

CLINICAL CORRELATION

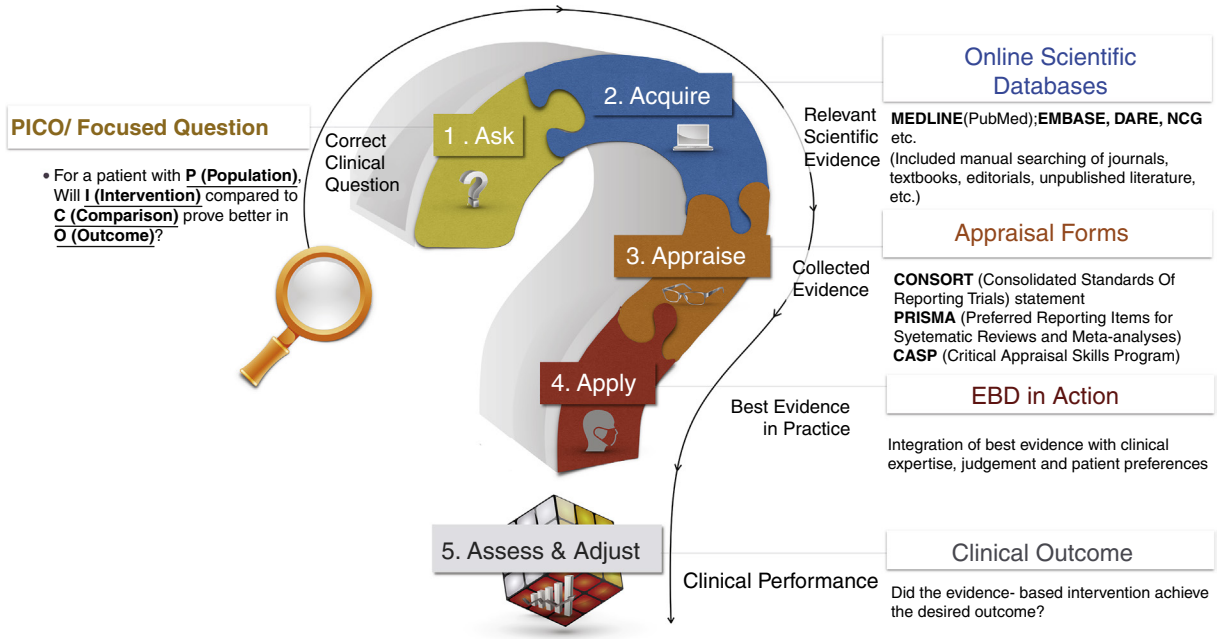
What are the advantages of a formal process of evidence-based decision-making?

EBDM takes time and practice to learn to use. Nevertheless, when followed correctly and consistently as a structured process, it brings about an understanding of:
- What constitutes 'good' evidence
- Benefits versus risk quantification of any new intervention
- What fits well with individual clinical expertise and patient values/preferences

4. **Apply**: In this step, the clinician integrates the results of best scientific evidence obtained in the first three steps with good clinical judgment and patient preferences, and applies it to the clinical scenario. This takes clinical decision-making to a whole new level of competence compared with traditional methods of problem-solving.
5. **Assess & Adjust**: The final step in the EBDM process is to evaluate how effectively the intervention identified in the above four steps brings about a good clinical outcome. Depending on whether the solution works or not, the results are shared with other care providers through various means, or adjustments are made in interventions, to provide better patient care.

Conclusions

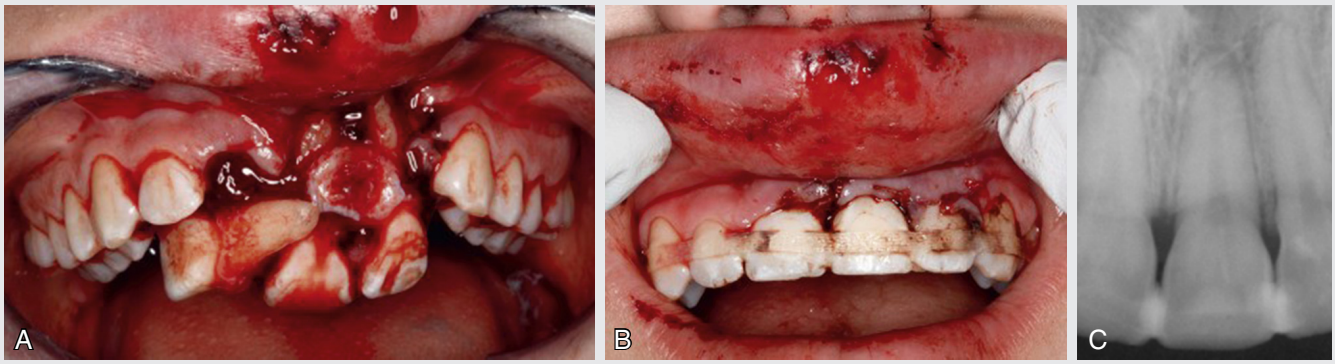
As EBDM integrates into the clinical problem-solving process and becomes standard practice, it becomes vital for clinicians to understand the importance of critical thinking, rigorous methodology in research, and what constitutes credible evidence for clinical use. The EBDM process takes time to learn and practice. However, once learned well, it helps to effectively translate the findings from best available scientific evidence into clinical practice by providing the skill sets required for health care providers to make competent clinical decisions.



• **Fig 1.4** Evidence-Based Decision-Making Process. The process is structured into five steps that can be thought of as the five A's (ask, acquire, appraise, apply, and assess).[6] EMBASE, Excerpta Medica dataBASE; DARE, Database of Abstracts of Reviews of Effects; NCG, National Guideline Clearinghouse.

CASE-BASED LEARNING EXERCISE

Scenario: A 13-year-old female patient was struck in the face with a softball. She was later cleared by paramedics for any medical conditions, and dental trauma was identified as the primary injury. She presented to the dental office 45 minutes after the trauma. The teeth remained in her mouth, and the preference of the patient and her parents was to "do anything to keep the teeth." Upon clinical examination, there was complete avulsion of the maxillary right central incisor from the socket and lateral luxation of the maxillary left central and lateral incisors (A). In addition, there was an alveolar bone fracture partially encasing the roots of the maxillary left central and lateral incisors. The clinician replanted the teeth and reapproximated the gingival tissue with sutures. A stable and accurate Ribbond and flowable composite splint were used to stabilize teeth (B) and a radiograph was taken (C).



Clinical images are from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier

Questions

1. Which of the following is NOT a possible Outcome in the following compiled PICO question related to this patient**?** For a patient with replanted teeth (P), will long-term splinting (2–4 weeks) (I) compared **with** short-term splinting (7–14 days) (C) increase:
- **a.** Patient satisfaction.
- **b.** Functional periodontal healing
- **c.** The risk of tooth resorption
- **d.** Successful tooth integration

2. Before treating this patient, the clinician reads a clinical practice guideline (CPG) in order to make a clinical decision. CPG are _____ resources:
- **a.** Primary
- **b.** Secondary
- **c.** Tertiary

3. From the type of study designs mentioned below, identify the one with the highest level of evidence:
- **a.** Case-control study
- **b.** Cohort study
- **c.** Randomized controlled trial
- **d.** Systematic review

4. The clinician evaluated the outcome of the rendered treatment during the follow-up visits. Is post-treatment evaluation of outcomes a part of evidence-based dentistry process?
- **a** Yes
- **b.** No

This chapter was developed from chapters 1 and 2 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: c

Explanation: Long-term splinting will facilitate the successful tooth integration and functional periodontal healing that will assure patient satisfaction. The risk for tooth resorption will decrease, not increase, with long-term splinting.

2. Answer: b

Explanation: Secondary resources are synthesized studies and publications of primary research that has already been conducted. CPGs are based on the previous studies performed.

3. Answer: d

Explanation: The study designs mentioned guide clinical decisions and contribute to the body of knowledge. Of the listed choices, systematic reviews represent the highest level of evidence (see Fig. 1.3).

4. Answer: b

Explanation: Evidence-based dentistry not only involves applying the best evidence in a given clinical situation but also includes assessment of post-treatment outcomes and adjusting the clinical process based on the outcome assessment.

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2

Anatomy, Structure, and Function of the Periodontium

Relevant Terminology

Terminology/Abbreviation	Explanation
alveolar bone proper	The inner socket wall of thin, compact bone with the cribriform plate.

ankylosis	• Fusion of the cementum and the alveolar bone with obliteration of the periodontal ligament (PDL)
	• May develop in teeth with cemental resorption (considered abnormal cemental repair where bone fills resorption cavity instead of reparative cementum), chronic inflammation, tooth replantation, occlusal trauma, and in embedded teeth
	• Neither definitive cause nor treatment is available
	• Osseointegration of titanium implants is considered a form of ankylosis
	• Characterized by:
	  • Metallic sound on percussion
	  • Lack of physiologic tooth mobility and proprioception (due to lack of PDL tissue)
	  • Inability of tooth to adapt to altered forces as physiologic drifting and eruption cannot happen

bone cells	Cells seen within bone are mainly of four types: • osteogenic cells—precursors that develop into osteoblasts • osteoblasts—bone-forming cells • osteocytes—maintain bone tissue • osteoclasts—bone resorbing cells
bone marrow	• The red hematopoietic marrow of the newborn becomes fatty or yellow inactive marrow with aging • Foci of red marrow can be seen as radiolucent areas in maxillary tuberosity, maxillary and mandibular molar and premolar areas, and the mandibular symphysis and ramus angle
bundle bone	• Bone adjacent to the PDL that contains a great number of Sharpey fibers • Resorbed after tooth extraction • Can be seen throughout the skeletal system wherever ligaments and muscles are attached
cancellous bone	• Trabeculae enclosing marrow spaces • Predominantly found in interdental and interradicular spaces • More in maxilla than in mandible
cell adhesion proteins	Osteopontin and sialoproteins, important for the adhesion of both osteoblasts and osteoclasts.
cemental aplasia	Absence of cementum.
cemental hyperplasia/ hypercementosis	Excessive deposition of cementum. • Hypercementosis of the entire dentition may occur in Paget's disease • Usually it may be localized to teeth undergoing supraeruption or low-grade periapical irritation from pulpal disease
cemental hypoplasia	Paucity of cementum.
cemental resting lines	• Incremental lines parallel to the long axis of the root viewed in microscopic sections, separating lamellae of cementum • Indicate "rest lines" that are more mineralized than adjacent cementum and represent appositional growth pattern of cementum
cemental reversal line	A deeply staining irregular line, viewed in microscopic sections, that demarcates newly formed (reparative) cementum from the root, delineating the border of a previous cemental resorption

Relevant Terminology—cont'd

Terminology/Abbreviation	Explanation
cemental spike	Spike-like excrescence created by either coalescence of cementicles, or calcification of PDL fibers at the point of insertion into cementum on root surface.
cemental tear	Detachment of cementum fragment from root surface (may occur in response to a severe blow to the tooth).
cementodentinal junction	The terminal apical area of the cementum where it joins the internal root canal dentin.
cementoenamel junction (CEJ)	The location where the enamel and the cementum meet.
coupling	Interdependency of osteoblasts and osteoclasts during bone remodeling.
cribriform plate	A structure pierced by numerous small holes.
dehiscence	Denuded areas of alveolar bone covering tooth roots that extend through the marginal bone.
dental follicle	Consists of undifferentiated fibroblasts; the zone that is immediately in contact with the dental organ continues with the dental papilla.
desmosome	Adhesive junction involved in cell-cell attachment. Consists of: • Intracellular component—two dense attachment plaques into which tonofibrils insert • Extracellular component—an intermediate electron-dense line in the extracellular compartment
disuse atrophy/afunctional atrophy	Decreased occlusal function results in reduced number and thickness of trabeculae as well as atrophied PDL.
endosteum	Tissue that lines the internal bone cavities. Composed of a single layer of osteoblasts (osteogenic layer) and a small amount of connective tissue (fibrous layer).
epithelial cell rests of Malassez	Remnants of Hertwig root sheath, forming clusters of cells within the PDL.
fenestration	Isolated area in which the root is denuded of bone and the root surface is covered by periosteum and overlying gingiva.
gingival zenith	The most apical part of the marginal gingival scallop.
hemidesmosomes	Structural proteins that play a role in the adhesion of basal epithelial cells to the underlying basement membrane.
Hertwig epithelial root sheath	• Apical portion of REE (reduced enamel epithelium), determines root shape and forms cementum • Disappears during the development of periodontium, but remains as the epithelial cell rests of Malassez • Secretes proteins (e.g., bone sialoprotein, osteopontin, and amelogenin)
Howship lacunae	Eroded bone surfaces containing osteoclasts; occur in bone undergoing resorption.
junctional epithelium (JE)	The reduced-enamel epithelium unites with the oral epithelium and forms JE, a continually self-renewing structure. A collar-like band of stratified squamous nonkeratinizing epithelium, it tapers from the coronal end (10–29 cells wide) to 1–2 cells wide at its apical termination. In healthy periodontium, JE terminates at the CEJ.
lamina dura	Radiographic appearance of compact bone that lies adjacent to PDL.
lamina lucida and lamina densa	Two layers of basal lamina visible under the electron microscope. Under the light microscope, they together form the structure referred to as basement membrane.
lamina propria	Gingival connective tissue core underlying gingival epithelium.
Langerhans cells	Dendritic cells derived from monocyte precursors in the bone marrow, located among suprabasal layers of epithelium. Serve as antigen-presenting cells in the innate immune response. They contain Birbeck granules.
melanocytes	Dendritic cells located in the basal and spinous layers; synthesize melanin.
melanosome	Organelle found in melanocytes that is a site for synthesis, storage, and transport of melanin. Melanosomes are responsible for color and photoprotection in animal cells and tissues.
Merkel cells	Tactile receptors, connected to adjacent cells via desmosomes.
orthokeratinization	Represents complete keratinization. No nuclei are seen in the stratum corneum where a horny layer is present over a well-defined stratum granulosum.
osteoblasts	Cells that produce the organic matrix of bone, differentiated from pluripotent follicle cells.

Continued

Relevant Terminology—cont'd

Terminology/Abbreviation	Explanation
osteoclasts	Cells of hematopoietic origin, formed by the fusion of mononuclear cells to form large, multinucleate cells. The activity and morphology of their ruffled border can be regulated by parathyroid hormone and calcitonin.
osteocytes	Bone cells formed when osteoblasts that become trapped in lacunae within the bony matrix. Osteocytes extend processes into canaliculi for exchange of oxygen and nutrients.
parakeratinization	Incomplete keratinization process in which pyknotic nuclei are retained in the stratum corneum.
periosteum	The tissue that covers the outer surface of bone. Its inner layer is composed of osteoblasts surrounded by osteoprogenitor cells; the outer layer, composed of collagen fibers and fibroblasts, is rich in blood vessels and nerves. Bundles of periosteal collagen fibers penetrate the bone.
physiologic migration of the tooth	With time and wear, the proximal contact areas of the teeth are flattened, and the teeth tend to move in the mesial direction.
reduced enamel epithelium (REE)	Formed from outer and inner epithelia of the enamel organ. The apical portion of REE becomes the Hertwig epithelial root sheath.
stippling	• Presents on the attached gingiva bound to underlying bone. • Presents as Microscopic elevations and depressions on the surface of the gingiva due to connective tissue projections within the tissue. • Stippling does not necessarily indicate health, and smooth gingival tissue does not necessarily indicate disease.
sulcular epithelium	Thin, nonkeratinized stratified squamous epithelium without rete pegs.
tight junctions	Also called zona occludens. Involved in cell-cell attachment, allowing small molecules to pass from one cell to another.
tonofilaments	Structural filaments of keratin; make up tonofibrils in the epithelial tissue.
trauma from occlusion	Injury to the periodontium caused by forces that exceed the adaptive capacity of the periodontium.

Fast Facts

Three zones of oral mucosa	• Masticatory mucosa (gingiva, hard palate), keratinized • Specialized mucosa (dorsum of tongue), keratinized • Mucous membrane (lining mucosa), not keratinized
Zones of gingiva	• Marginal gingiva • Gingival sulcus • Attached gingiva • Interdental gingiva (pyramidal or "col" shape)
Penetration of the probe	Can be affected by: • Probe diameter • Probing force • Level of inflammation
Width of attached gingiva	• Distance between the mucogingival junction and the projection on the external surface of the bottom of the gingival sulcus • Not same as keratinized gingiva • Greatest in the incisor region and narrower in the posterior segments (narrowest mandibular premolar region)
Functions of gingival epithelium	• Mechanical, chemical, water, and microbial barrier • Signaling functions
Architectural integrity of gingival epithelium	Maintained by: • Cell-cell attachments via desmosomes, adherens junctions, gap junctions, and tight junctions • Cell–basal lamina attachments via hemidesmosomes • Mechanical support by keratin cytoskeleton

Fast Facts—cont'd

Cells comprising gingival epithelium	• Keratinocytes (major type)
	• Non-keratinocytes:
	  • Langerhans cells (phagocytes, antigen-presenting cells)
	  • Melanocytes (melanin-producing cells)
	  • Merkel cells (tactile receptors)

Development of gingival sulcus	• The reduced enamel epithelium unites with the oral epithelium and transforms into the junctional epithelium
Turnover times of oral epithelium	• 5–6 days for palate, tongue, and cheek • 10–12 days for gingiva • 1–6 days for junctional epithelium. Rapid shedding of cells effectively removes bacteria and serves as a part of antimicrobial defense mechanisms
Three types of connective tissue fibers in gingival connective tissue	• Collagen fibers, mainly type I in lamina propria; type IV seen in basement membrane and blood vessel walls • Reticular fibers • Elastic fibers
Cells in the gingival connective tissue	• Fibroblasts (predominant) • Mast cells, releasing histamine • Macrophages (phagocytes) • Histiocytes (phagocytes) • Adipose cells • Small number of inflammatory cells (neutrophils and plasma cells) seen near base of sulcus in clinically healthy gingiva
Blood supply to gingiva	• Supraperiosteal arterioles—extend along facial and lingual aspects of alveolar bone, giving out capillaries that reach up to the sulcular epithelium and between rete pegs • Vessels of the periodontal ligament—extend into the gingiva and anastomose with capillaries in the sulcular area • Arterioles—emerge from interdental bone crest and extend parallel to the crest of the bone to anastomose with vessels of PDL
Physiologic pigmentation	Normal pigmentation of gingiva, oral mucosa, and skin due to the presence of a non-hemoglobin-derived brown pigment, melanin, within epithelium.
Gingival crevicular fluid (GCF)	• Minimal in health, increases during inflammation • Cleanses materials from the sulcus and improves adhesion of the epithelium to the tooth via its plasma protein content • Possesses antimicrobial properties
Formation of PDL	• During tooth eruption, collagen fibrils become activated, gradually acquiring an organized orientation (oblique to the tooth) • Alveolar bone deposition occurs simultaneously with PDL organization • Both developing and mature PDL contains undifferentiated stem cells that retain the potential to differentiate into osteoblasts, cementoblasts, and fibroblasts
Cells in periodontal ligament	• Connective tissue cells (predominantly fibroblasts, cementoblasts, and osteoblasts) • Epithelial cell rests of Malassez • Immune cells • Cells associated with neurovascular elements
Six groups of principal fibers of PDL	• Transseptal: no osseous attachment • Alveolar crest • Horizontal • Oblique: largest group • Apical • Interradicular
Sensory fibers innervating PDL	• Free nerve endings as nociceptors (pain transmission) • Ruffini, Meissner, and spindle-like endings as mechanoreceptors
Ground substance of PDL	• 70% water • Glycosaminoglycans (hyaluronic acid and proteoglycans) and glycoproteins (fibronectin and laminin)

Continued

Fast Facts—cont'd

Physical functions of PDL	• Protects vessels and nerves from mechanical injury • Transmission of occlusal forces to the bone (oblique fibers sustain major part of axial force) • Attachment of teeth to bone • Maintenance of gingival tissues in their proper relationship to the teeth • Resistance to the impact of occlusal forces (shock absorption)
Orthodontic tooth movement and periodontium	• Site-specific bone remodeling in the absence of inflammation • Tensile forces stimulate the formation and activity of osteoblastic cells, whereas compressive forces promote osteoclastic activity
Axis of rotation	• The periodontal ligament is shaped like an hourglass, narrowest in the region of the axis of rotation • Multirooted teeth: axis of rotation is located in the interradicular bone between roots • Single-rooted teeth: axis of rotation is located in the area between the apical third and the middle third of the root
Four types of cementum (Schroeder)	• Acellular afibrillar cementum (most coronal) • Acellular extrinsic-fiber cementum (cervical third) • Cellular mixed stratified cementum (apical third) • Cellular intrinsic-fiber cementum
Organic matrix of cementum	• Type I (90%) collagen and type III (5%) collagens • Sharpey fibers are predominantly type I
Cementum resorption (root resorption): etiology and pathogenesis	• Local factors: trauma from occlusion, orthodontic movement, pressure from malaligned erupting teeth, periapical and periodontal diseases • Systemic conditions: calcium deficiency, hypothyroidism, hereditary fibrous osteodystrophy, Paget disease • Multinucleated giant cells and large macrophages are responsible for cementum resorption
Thickness of cementum	• Unlike all other periodontal tissues (epithelium, connective tissue, bone and periodontal ligament), cementum does not undergo continuous turnover, but increases with age because it can be continuously deposited in an appositional manner • Increases more in the apical regions and furcations than in the cervical regions to compensate for eruption of teeth (which happens to compensate for tooth attrition in order to maintain occlusal contact) • Increases more in the distal than mesial regions to compensate for physiological mesial drifting of teeth
Cementoenamel junction	Three types usually seen: • Cementum overlaps enamel in 60%–65% cases • Edge-to-edge butt joint in 30% • Cementum and enamel do not meet in 5%–10% cases
Non-collagenous molecules common to cementum and bone	• Bone sialoprotein • Osteopontin
Non-collagenous molecules unique to cementum	• Cementum attachment protein: helps with preferential adhesion of osteoblasts and PDL fibroblasts to root surface versus gingival fibroblasts/keratinocytes • Cementum-derived growth factor: enhances proliferation of gingival fibroblasts and PDL cells
Functions of cementum	• Anchorage—primary function; provides the medium for anchoring tooth to alveolar socket via PDL fibers • Adaptation—continuous deposition of cementum (especially in apical portions) occurs to compensate for tooth wear and mesial drifting • Repair—damage to roots (fractures, resorption) can be repaired by new cementum deposition
Alveolar process	• Portion of the maxilla and mandible that forms and supports the tooth sockets • Forms as tooth erupts for the osseous attachment of tooth and disappears after tooth loss

Fast Facts—cont'd

Cancellous bone and cortical bone	These structures have the same cells and intercellular matrix. They differ in the basic arrangement of the components: • Compact bone—bone is tightly packed in concentric sheets/lamellae • Cancellous bone—bone is loosely arranged as a network of bony trabeculae interspersed with marrow cavities
Composition of bone	• ⅔ inorganic matter and ⅓ organic matrix • 99% of the body's calcium ions are from bone • 90% of organic matrix is collagen type I
Bone remodeling	The major pathway responsible for bony changes in shape; allows resistance to forces, repair of wounds, and maintenance of calcium and phosphate homeostasis in the body through the coupling of bone resorption by osteoclasts with bone formation by osteoblasts
Regulation of bone remodeling	• A decrease in blood calcium results in parathyroid hormone (PTH) release • PTH stimulates osteoclastogenesis (production of osteoclasts) • Osteoclasts resorb bone, releasing calcium ions into the blood • Normal blood level of calcium turns off the secretion of PTH via a feedback mechanism
Distance from CEJ to alveolar crest	• Young adults 0.75–1.49 mm • Increases with age to average 2.81 mm (not solely from aging; can also be due to cumulative effect from periodontal disease)
Osseous topography	Height and thickness of the facial and lingual bony plates are affected by: • The alignment of teeth • The angulation of root to the bone • Occlusal force
Alveolar bone formation	• Alveolar bone develops around each tooth follicle during odontogenesis • Formed during fetal growth by intramembranous ossification • During odontogenesis, alveolar bone merges with the separately developing basal bone to become one continuous structure
Effects of aging on gingival dimension	In a healthy periodontium free of trauma, the width of the attached gingiva theoretically increases with age through continuous eruption as a result of tooth surface attrition, while the gingival margin moves with the tooth coronally.
Effects of aging in progression of periodontal diseases and response to periodontal therapy	• Aging provides only clinically insignificant increased risk of loss of periodontium, and is not a true risk factor for periodontal diseases • Aging itself has zero to minimal impact on an individual's response to periodontal treatment
Effects of aging on gingival connective tissue and PDL	Gingival connective tissue and PDL become denser and coarser, attributed to fewer, more irregular fibroblasts present in periodontium.
Mucogingival junction and aging	• Remains stationary throughout adult life, while teeth move in an occlusal direction • As a result, the width of attached gingiva increases with age

Core Knowledge

Introduction

The normal support to retain teeth in their function is provided by the four main tissue components of the periodontium working as a single unit:

- gingiva
- periodontal ligament (PDL)
- cementum
- alveolar process

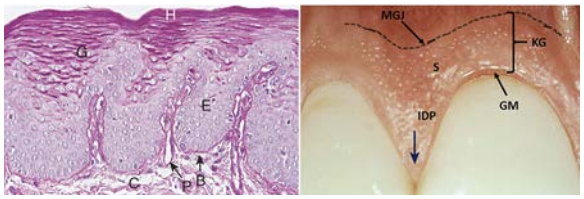
Gingiva

The gingiva is that part of the oral mucosa that covers the alveolar processes of the jaws and surrounds the necks of the teeth. Macroscopically, the gingiva can be divided into four anatomic zones:

1. **Marginal gingiva**—also called "free gingiva," it forms the terminal unattached border of gingiva surrounding the cervical area of a tooth. It is sometimes separated from the attached gingiva by a *free gingival groove.*
2. **Gingival sulcus**—a shallow, v-shaped crevice around every tooth that is bound on the inside by the tooth surface, outside by the sulcular epithelium, and at the apical region by the gingival epithelial attachment (junctional epithelium, JE).
3. **Attached gingiva**—firm and resilient, the attached gingiva continues apically from the marginal gingiva and is tightly bound to the tooth surface and the periosteum of alveolar bone. On the facial surfaces, it continues apically

as the movable alveolar mucosa and is demarcated from it by the *mucogingival line* (or mucogingival junction). On the palatal aspect in the maxilla, it continues imperceptibly as firm palatal mucosa, while on the lingual aspects of the mandible, it continues as the alveolar mucosa that blends into the mucous membrane of the floor of the mouth.

4. **Interdental gingiva/papilla**—occupies the interproximal space/embrasure cervical to the contact points of teeth. The papilla is "pyramidal" in shape (single apex/tip cervical to the contact point) between anterior teeth and "col" shaped (two tips, facial and lingual, just cervical to the contact area with a valley-like depression connecting them) between posterior teeth.



• **Fig. 2.1** Structure of the Gingiva. (Left) Normal human gingiva stained with periodic acid–Schiff staining. Epithelium (E) is separated from the underlying connective tissue (C) by the basement membrane (B). Epithelium consists of superficial hornified (H) and underlying granular layers (G). Note the blood vessel walls in the papillary projections of the connective tissue (P). (Right) Buccal gingiva, indicating the gingival margin (GM), keratinized gingiva (KG) and interdental papilla (IDP) that is separated from the alveolar mucosa by the mucogingival junction (MGJ). Note the stippled (S) appearance of healthy gingiva. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

Microscopically, the gingiva comprises:

- **Epithelial components**—the primary cell type of stratified squamous epithelium is the *keratinocyte.* Three degrees of *keratinization* (the process of forming scales of keratin in the superficial layers) are possible within the gingiva:
  - Orthokeratinization: completely keratinized, with a well-demarcated superficial horny layer (stratum corneum) with no nuclei and a well-defined underlying stratum granulosum
  - Parakeratinization: less differentiated and keratinized, with pyknotic nuclei in the most superficial layers; the stratum granulosum is not well defined. This is most common in the gingiva
  - Non-keratinized: surface cells are nucleated, showing no signs of keratinization
- **Connective tissue components**—made up of cells and collagen fibers within an extracellular matrix that forms the core of the connective tissue, underlying the epithelial components.

See Fig. 2.1 and Table 2.1 for clinical and structural characteristics of gingival epithelium.

The gingiva is attached to the tooth surface by both epithelial and connective tissue components. The JE and underlying supporting gingival fibers within connective tissue function together as one unit called the dentogingival unit (Fig. 2.2).

TABLE 2.1 Structural and Functional Characteristics of Different Areas of Gingival Epithelium

	Oral Epithelium (OE)	Sulcular Epithelium (SE)	Junctional Epithelium (JE)
Function	• Protection	• Protection	• Attachment and host defense
Location	• Covers crest of marginal gingiva • Outer surface of marginal and attached gingiva	• Extends from coronal limit of JE to crest of marginal gingiva	• Cuff/collar-like band of stratified epithelium around necks of teeth
Degree of keratinization	• Mostly parakeratinized; sometimes orthokeratinized	• Nonkeratinized	• Nonkeratinized

Differentiating features	• Rete pegs are present and interdigitate with underlying connective tissue core
	• Though mainly composed of keratinocytes, nonkeratinocytes/clear cells typically found are:
	  • Langerhans cells—antigen-presenting cells helping with host defense
	  • Melanocytes—melanin producing cells
	  • Merkel cells—nerve endings for tactile perception
		• Normally does not contain Merkel cells or rete pegs
		• Has the potential to keratinize if reflected and exposed to oral cavity or if plaque is completely eliminated within the sulcus
		• Semipermeable to bacterial products and tissue fluids (less permeable than JE)
			• No rete pegs; tapers from coronal end (10–29 cells thick) to apical end (1–2 cells thick)
			• Permeable to gingival crevicular fluid (GCF) and inflammatory/ immune cells.
			• Exhibits extremely rapid turnover rate of cells (continuous self-renewal) with mitotic activity in all layers

CLINICAL CORRELATE

After a surgical flap procedure in which the junctional epithelium (JE) is mechanically "separated" from the tooth surface, how is the epithelial attachment reestablished? Is this the same procedure that happens following surgical removal of the entire gingival attachment, for example during gingivectomy?

The two surgical situations described above are hypothesized to heal via different mechanisms. Following mechanical separation of the JE from the tooth surface during flap surgery, some junctional epithelial cells remain in contact with tooth (and hence are called DAT cells or "directly attached to the tooth" cells); these cells can proliferate to regenerate the epithelial attachment in about 7 days. In cases where gingivectomy is performed with complete removal of the JE, there are no DAT cells that can initiate epithelial proliferation. Instead, a new epithelial attachment forms from adjacent oral epithelium. Migration of cells occurs from the cut oral epithelial edge toward the root surface; it takes at least 2 weeks for regeneration of a complete JE that will grow apically over the root surface until it encounters firm collagen fibers attached to cementum.

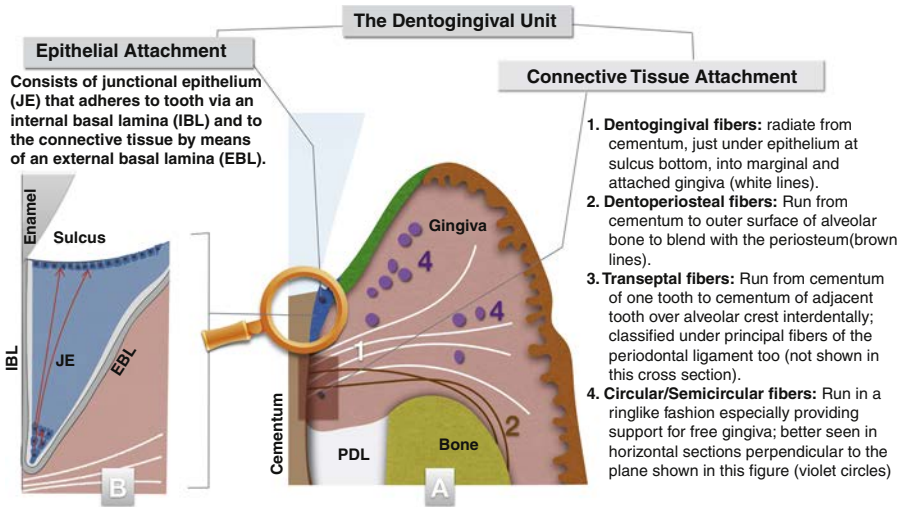
Functions of the Gingiva

- Gingival epithelium:
  - Physical barrier against foreign agents;
  - Host defense coordination;
  - Rapid turnover, especially of JE cells, ensures effective clearance of invading bacteria and their metabolic products from the gingival sulcus.
- Gingival connective tissue:
  - High turnover of cells and collagen matrix ensures good repair and regenerative potential;
  - Abundant blood and nerve supply ensures health, healing after surgery, and very little scarring.

CLINICAL CORRELATE

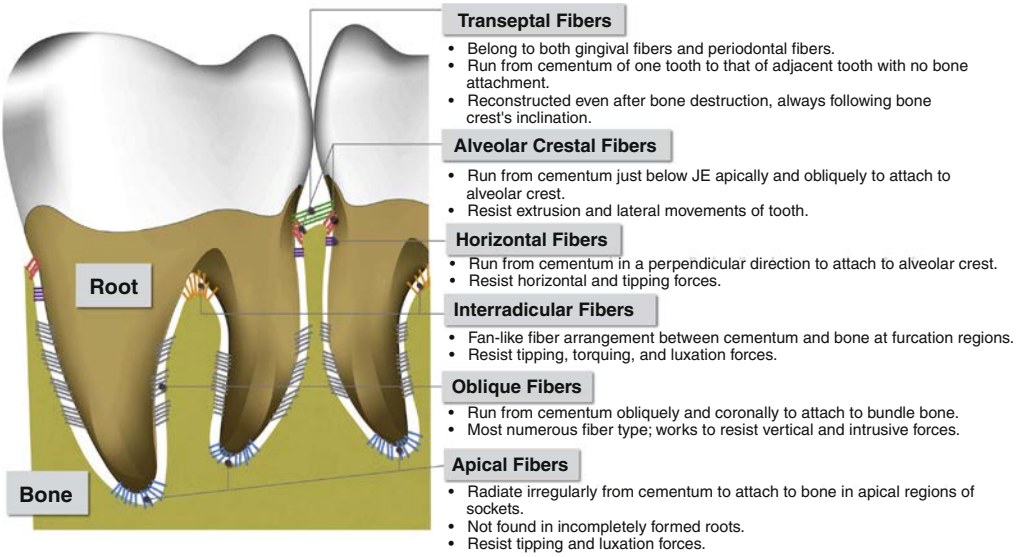
What is the difference between *active* eruption and *passive* eruption?

Active eruption is the movement of the teeth in the direction of the occlusal plane, whereas passive eruption is the exposure of the teeth via apical migration of the gingiva. Active eruption is coordinated with attrition; the teeth erupt to compensate for tooth substance that has been worn away by attrition. Although originally thought to be a normal physiologic process, passive eruption is now considered a pathologic process. It involves gingival recession as the JE retreats apically from its original position near the cementoenamel junction.



• **Fig. 2.2** The Dentogingival Unit. The attachment of gingiva to the tooth surface includes both epithelial and connective tissue components. In this diagram, part A (right) represents the entire dentogingival unit, mainly comprising the junctional epithelium (attachment epithelium seen as blue area) and gingival group of fibers (connective tissue attachment seen as reddish-brown area). The three types of epithelium seen in the gingiva are: oral epithelium (brown), sulcular epithelium (green), and junctional epithelium (blue). Part B (left) shows a magnified view of the epithelial attachment which comprises:

1. Junctional epithelium (JE)—seen as blue area with blue cells, sandwiched between gray areas;
2. Internal basal lamina (IBL)—seen toward tooth surface; comprises lamina lucida and lamina densa; can attach to enamel, cementum, or sometimes even dentin;
3. External basal lamina (EBL)—seen away from tooth surface, toward connective tissue component of gingiva (also contains lamina lucida and lamina densa). The basal lamina connects to JE cells via hemidesmosomes. The JE is wider at the coronal end (10–29 cells thick) than at its apical end (1–2 cells thick). Apical to the epithelial attachment, connective tissue attachment is seen in the form of collagen fibers inserting into the tooth surface. Red arrows represent the direction of movement of JE cells during differentiation and turnover where they travel coronally to the bottom of the gingival sulcus and are shed into the crevice. (All structures in the figures are diagrammatic representations for concept understanding; they are not drawn to scale.)



• **Fig 2.3** Principal Fibers of the Periodontal Ligament. Collagen fibers within the periodontal ligament space, embedded in cementum and alveolar bone at both ends, provide a soft connectivity between the periodontium's mineralized tissues. They are typically grouped into the following types based on their location and orientation: (1) transeptal fibers (green lines), (2) alveolar crestal fibers (red lines), (3) horizontal fibers (purple lines), (4) interradicular fibers (orange lines), (5) oblique fibers (gray lines), and (6) apical fibers (blue lines). In addition to the principal fibers, smaller collagen fibers (the indifferent fiber plexus) run associated with them in various directions. All fibers undergo regular remodeling by periodontal ligament cells to cope with and adapt to variations in stimuli.

Periodontal Ligament

The periodontal ligament (PDL) fills the space between the bony tooth sockets and the roots of the teeth. It:

- Extends coronally to meet the most apical portion of the gingival lamina propria and merges with the dental pulpal tissue at the apical foramen
- Is a highly vascular and cellular connective tissue that contains many fibers, the majority of which are collagen fibers arranged in specific patterns to resist various physical forces encountered by the tooth. These collagen fibers (mainly type I) are called the principal fibers of the periodontal ligament (Fig. 2.3).

Periodontal Ligament Components

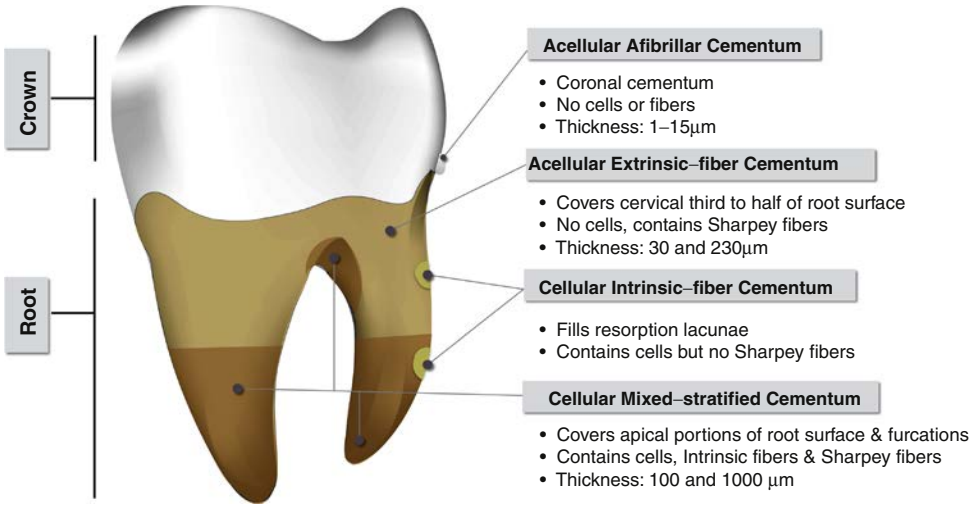
The PDL tissue is composed of:

- **Periodontal fibers:**
  - Principal fibers—collagen fibers arranged in regular bundles with specific orientations (Fig. 2.2)
  - Immature elastin fibers—oxytalan fibers (run parallel to the root surface in a vertical direction to bend and enter the cementum near the cervical portions; thought to regulate blood flow within the PDL space) and elaunin fibers
- **Cellular elements:**
  - Connective tissue cells:
    1. Fibroblasts—most numerous, responsible for collagen turnover, both synthesis and degradation
    2. Cementoblasts—responsible for cementum formation; line the tooth side of the PDL space
    3. Osteoblasts—responsible for bone formation; line the bone side of the PDL space
    4. Osteoclasts—responsible for bone resorption
  - Epithelial cell rests of Malassez—remnants of Hertwig epithelial root sheath found as interlacing strands or cell clusters within the PDL space close to the cementum. They are hypothesized to proliferate when stimulated to form periapical and lateral root cysts and undergo calcification to form cementicles. May be involved in periodontal repair and regeneration
  - Defense cells—neutrophils, macrophages, eosinophils, mast cells, etc. are also found within the PDL space
  - Cells associated with neurovascular elements
- **Ground substance:** This fills the space between fibers and cells and is composed of:

CLINICAL CORRELATE

In the practice of restorative dentistry, why is it important to consider periodontal ligament changes around a tooth?

The thickness of the periodontal ligament (PDL) is regulated by the functional movements of the tooth; in teeth without opposing tooth contacts, the PDL is thin and functionless, whereas the opposite effect is seen (i.e., the PDL is wider) around teeth under excessive occlusal forces. In the case of teeth that have been long out of function, if they are chosen to serve as abutments for removable prostheses or fixed bridge, or will be opposing a new prosthesis, the PDL is poorly adapted to carry the sudden occlusal loads placed by a restoration. The patient may be unable to comfortably use the restoration immediately after placement. An adjustment period must elapse before the supporting PDL tissues become adapted to the new functional demands.



• **Fig 2.4 Types of Cementum.**

- Glycosaminoglycans—hyaluronic acid and proteoglycans
- Glycoproteins—fibronectin, laminin

Functions of Periodontal Ligament

- **Supportive:**
  - Provision of a soft-tissue "casing" around teeth;
  - Transmission of occlusal forces to the bone;
  - Attachment of teeth to the bone;
  - Maintenance of the gingival tissues in their proper relationship to the teeth;
  - Resistance to the impact of occlusal forces (i.e., shock absorption). Two theories attempt to explain this phenomenon:
    1. Tensional theory—the principal fibers of the PDL play the major role in shock absorption. Forces on teeth cause the usually wavy collagen fibers to straighten, and are transmitted to the alveolar bone. When the forces exceed the adaptive capacity of alveolar bone, they are dissipated to the basal bone.
    2. Viscoelastic theory—fluid within the PDL space plays the primary role in shock absorption, with the principal fibers playing a secondary role. Forces on teeth cause outward movement of fluid from within the PDL space into alveolar bone, which leads to tightening of fiber bundles within the PDL space. This in turn puts pressure on blood vessels running between the fibers, causing stenosis and back pressure, thus leading to replenishment of fluid (within PDL space) lost to bone.
- **Formative—**bone, cementum, and connective tissue are formed by cells within the PDL:
  - In response to tooth movement
  - To accommodate or adapt to external forces on the periodontium
  - To repair injured tissues
- **Remodeling—**the breakdown and replacement of old cells and fibers occurs in the PDL space constantly throughout life, with the help of fibroblasts and mesenchymal cells that differentiate into osteoblasts and cementoblasts when the need arises.
- **Nutritional**—blood vessels supply nutrients to cementum, bone, and gingiva from the PDL space. Lymphatic drainage is also present within the PDL.
- **Sensory—**nerve fibers follow the course of blood vessels within the PDL space and end as one of several types of receptors:
  - Free nerve endings—lose their myelin sheath and end in a tree-like configuration; carry pain sensations
  - Ruffini-like receptors—mechanoreceptors found in the apical area
  - Meissner's corpuscles—coiled nerve endings; mechanoreceptors found in midroot regions
  - Spindle-like nerve endings—show fibrous encapsulation; located apically; transmit pressure and vibration sensations
- **Regulation of PDL width (homeostasis)—**the metabolism and spatial locations of cell populations (those responsible for formation of bone, cementum and PDL connective tissue) are tightly regulated and exquisitely controlled to ensure that the width of the PDL spaces around teeth remain fairly constant throughout life.

Cementum

Cementum is an avascular, calcified tissue of mesenchymal origin that covers the surface of the anatomic root. Root cementum is considered to be both part of a tooth and part of the periodontium. It mainly comprises:

- Organic content:
  - Collagen fibrils (extrinsic and intrinsic fibers)
  - Cellular elements (cementoblasts and cementocytes)
  - Calcified matrix.
- Inorganic content (45%–50%)—hydroxyapatite; less than in bone (65%), dentin (70%), or enamel (97%)

TABLE 2.2 Acellular and Cellular Cementum[1]

	Acellular (Primary) Cementum	Cellular (Secondary) Cementum
General features	• Slowly formed before tooth erupts to reach occlusal plane • Devoid of cells • Covers cervical half of root surface • Main function is anchorage	• Rapidly formed after tooth reaches occlusal plane • Contains cementocytes within lacunae that communicate via canaliculi • Covers apical portions of root surface and furcations • Main functions are adaptation and repair

Types	Acellular Afibrillar Cementum	Acellular Extrinsic-fiber Cementum	Cellular Mixed Stratified Cementum	Cellular Intrinsic-fiber Cementum
Cells	• None	• None	• Cementocytes	• Cementocytes
Collagen fibers	• None	• Sharpey fibers	• Sharpey fibers • Intrinsic fibers	• Intrinsic fibers
Fiber origin	–	• PDL fibroblasts	• PDL fibroblasts • Cementoblasts	• Cementoblasts

PDL, periodontal ligament.

Cementum presents as two major forms over the root (Fig. 2.4):

- Acellular (primary) cementum
- Cellular (secondary) cementum

The two types of collagen fibers within cementum are:

- **Extrinsic fibers**—also called Sharpey fibers, they represent the calcified portions of PDL fibers inserting into the cementum. They are laid down mostly perpendicular to the cemental root surface and come from a source external to the cementum, viz., PDL fibroblasts.
- **Intrinsic fibers**—laid down within the cementum mostly parallel to the cemental root surface and come from a source of cemental origin, viz., cementoblasts.

CLINICAL CORRELATE

What would be the ideal cementum type after periodontal regenerative procedures are performed?

The acellular extrinsic-fiber cementum is the type most desired following regenerative periodontal procedures. The cellular mixed stratified cementum is also of importance for the anchorage of the tooth within its alveolus. This is because both of these cemental types contain extrinsic fibers that are actually PDL fibers inserting into cementum.

Table 2.2 discusses the different types of cementum in detail.

Comparison of Cementum and Bone

Cementum and compact bone are very similar tissues; both are specialized connective tissues and share some chemical and structural characteristics. However, cementum is avascular and noninnervated compared with the richly vascularized and innervated bone tissue.

Cementum is more resistant to resorption than bone, and this property is what makes orthodontic movement possible. The forces placed on both cementum and bone during appliance activation is the same. The avascular nature of cementum makes it more resistant to resorption than the richly vascularized bone tissue when *optimal* orthodontic forces are applied carefully.

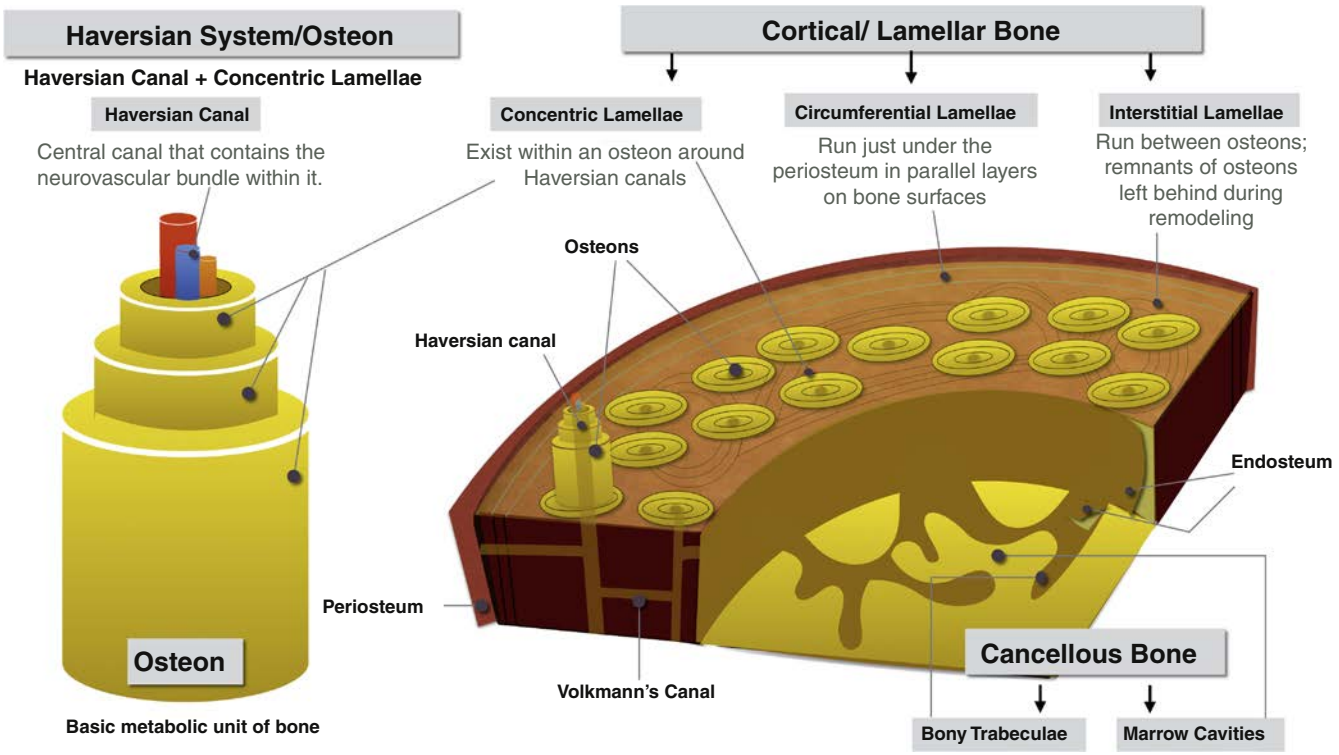
Functions of Cementum

- **Anchorage**—mainly achieved by acellular extrinsic-fiber cementum with some contribution from cellular mixed stratified cementum. In both types, Sharpey fibers allow anchorage of the tooth within the osseous socket.
- **Adaptation**—mainly achieved by cellular cementum. By continuous deposition, especially in apical and furcation areas, cellular cementum compensates for tooth wear that causes tooth eruption, to facilitate contact with the opposing tooth at the existing occlusal plane. Cementum also deposits on the distal root surfaces more than on mesial surfaces to compensate for physiological mesial drifting of teeth.
- **Repair**—mainly achieved by cellular intrinsic-fiber cementum. Reparative cementum formation is seen in cementum resorption bays and fracture lines. Cementum deposits rapidly during repair and does not usually contain any extrinsic fibers that can play a role in anchorage.

CLINICAL CORRELATE

Can cementum repair occur in nonvital teeth? What are the most important criteria for cementum repair?

Cemental repair can occur in both vital and devitalized teeth. The process requires viable connective tissue adjacent to cemental resorption areas/bays. If epithelium is not excluded from resorption areas during healing, it proliferates into the resorption area and cementum repair cannot take place.



• **Fig 2.5** Bone Histology and Structure. Bone is made of outer cortex (lamellar bone) and inner medulla (cancellous bone). The following components make up a complete bone structure.

- **Haversian system/osteon—**this is the basic metabolic unit of bone (found in both cortical and trabecular bone) made of:
  - Central Haversian canal which contains the neurovascular bundles.
  - Concentric layers of lamellar bone that contain osteocytes within lacunae, communicating with nearby cells via canaliculi.
- **Volkmann's canals—**contain blood vessels running between adjacent Haversian canals; responsible for the rich vascular network within compact bone.
- **Bone Linings—**bone is covered both on the outside and inside by soft tissue:
  - Periosteum—bilayered structure (outer fibrous layer, inner cellular [osteogenic] layer) that wraps the outer surface of cortical bone.
  - Endosteum—thin cellular layer that lines the inner portions of cortical and cancellous bone surfaces that face the medullary cavities.
- Cortical bone is made up of osteons and lamellae (circumferential, concentric, and interstitial).
- Cancellous bone is made up of trabecular bone and marrow cavities.

Alveolar Process

A discussion of the alveolar bone that supports and houses teeth within bony sockets will be better understood following a quick recap of certain characteristics common to all bone tissue.

Properties of Bone Tissue

General characteristics of human bones:

- Living tissues that possess toughness and elasticity
- Site of attachment for tendons, ligaments, and muscles
- Storage site for minerals (e.g., calcium, phosphorus)
- Provide the medium (marrow) for development and storage of blood cells

Classification of bones can be based on their developmental characteristics or their microscopic structure:

- *Development-based classification:*
  - Endochondral bones—formed by replacement of cartilage with bony tissue (e.g., trunk, extremities)
  - Intramembranous bones—formed by direct replacement of sheets of connective tissue membranes with bony tissue with no cartilage formation (e.g., mandible, alveolar process)
- *Microscopic structure–based classification:*
  - Mature bone:
    1. Compact/cortical/lamellar—solid bone mass arranged in layers called lamellae
    2. Cancellous/spongy/trabecular—honeycomb appearance with marrow cavities
  - Immature/woven bone: first bone formed; osteocytes trapped within rapidly forming matrix and irregularly oriented collagen fibers.

The main constituent structures of bone are:

- Bone cells (osteogenic cells, osteoblasts, osteocytes, and osteoclasts)
- Bone linings (periosteum and endosteum) Haversian system/osteons (Fig. 2.5).

Bone Composition Bone is a mixture of organic and inorganic substances:

- Inorganic/mineral content (⅔)—mainly calcium and phosphorus in the form of hydroxyapatite with trace amounts of magnesium, potassium, etc.
- Organic matrix (⅓):
  - collagenous proteins (90%)—mostly type I and type V;
  - noncollagenous proteins (10%)—osteocalcin, osteopontin, bone sialoprotein, osteonectin, BMP, etc.

Bone remodeling is a biologic phenomenon: that refers to the coupling of the processes of bone resorption (by osteoclasts) and bone formation (by osteoblasts). This is a lifelong remodeling process. Bone continues to change in order to adapt to forces placed on it, to repair fracture wounds, and to maintain calcium and phosphorus homeostasis. This complex process is regulated by distantly produced hormones (e.g., parathyroid hormone, calcitonin) and locally released factors (e.g., acid phosphatase and cathepsin secreted by osteoclasts at the site of resorption).

Sequence of events in bone remodeling:

1. **Cutting cone**—osteoclasts derived from blood "tunnel" into bone via Haversian canals, resorbing lamellar bone. They are found lining irregularly etched bone concavities called Howship lacunae where they create a sealed acidic environment that demineralizes bone and exposes organic bone matrix for degradation by enzymes. This resorption tunnel created within a Haversian system is called the "cutting cone."

CLINICAL CORRELATE

Why does the alveolar process resorb after tooth extraction?

The alveolar process is highly vascularized and extremely sensitive to tension and pressure stimuli transmitted via PDL fibers from a tooth in its socket. It continuously remodels in response to such stimuli, and maintains its volume around sockets. Once a tooth is extracted, this stimulus no longer exists and the alveolar process undergoes *disuse atrophy*. It resorbs because it is no longer required for its primary functions of tooth support and force absorption.

2. **Filling cone**—after resorption ceases (usually in about 3 weeks), osteoclasts are replaced by osteoblasts that begin to lay down new bone, beginning at the site where resorption ceased. These areas are marked by "reversal lines." The entire area of the Haversian system/osteon where active bone formation occurs is called "filling cone."

Properties of Alveolar Bone

The alveolar process is that portion of the maxilla and mandible that forms the tooth socket and houses the tooth root within it. It forms to allow osseous attachment of the PDL fibers around a root and resorbs when the tooth is lost. It consists of:

- External cortical plate;
- Alveolar bone proper—internal thin cortical plate of bone forming the tooth socket;
- Supporting alveolar bone—cancellous bone sandwiched between the two cortical bone plates.

See Fig. 2.6 for a detailed description of the alveolar bone that surrounds and houses the tooth root.

Functions of Alveolar Bone

Alveolar bone:

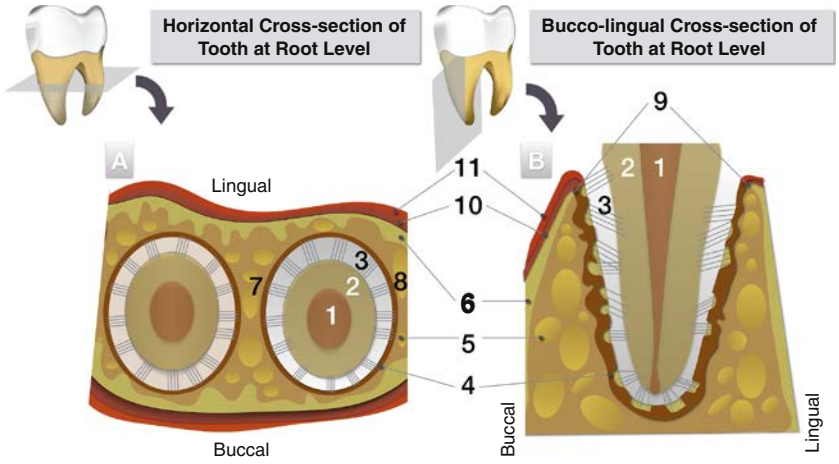
- Houses roots of teeth
- Anchors teeth roots to alveoli via Sharpey fibers
- Helps absorb and distribute occlusal forces generated during tooth contact
- Supplies blood vessels to the PDL
- Organizes eruption of primary and permanent teeth

Aging and the Periodontium

An understanding of the impact of aging on the periodontium is critical, because life expectancies are increasing all over the world. Aging has been associated with all of the following periodontal changes:

- Decreased keratinization and thickness of gingiva
- Increased width of attached gingiva
- Increased collagen content in gingival connective tissue
- Increased fibers and decreased cellularity within the PDL space
- Increased cementum width due to continuous deposition (especially in apical and lingual aspects of roots)
- Decreased osteogenic potential within alveolar bone

The biologic effects of aging actually have either no impact or only a minimal impact on an individual's response to periodontal treatment. Cognitive and motor skills are often affected in the aged population, leading to difficulties in maintaining oral hygiene; this significant aspect must be considered along with biologic changes to understand the periodontal changes that happen with aging.



• **Fig 2.6** Structure of Alveolar Bone. The alveolar bone encases the tooth root and underlies the gingiva. This figure shows two different cross-sections of the alveolar bone at the root level of a molar: (A) Horizontal/transverse cross-section close to the midroot level (where both interdental and interradicular bone are visible) and (B) Buccolingual longitudinal cross-section (where alveolar crest is visible). Numbers indicate the structures found in these sections:

Structures of the tooth:

1. Pulp—contains neurovascular bundle of the tooth
2. Root covered by cementum on the surface.

Structures of the periodontal ligament space:

3. Periodontal ligament (PDL) space with bundles of collagen fibers connecting cementum to bone.

Structures of alveolar bone:

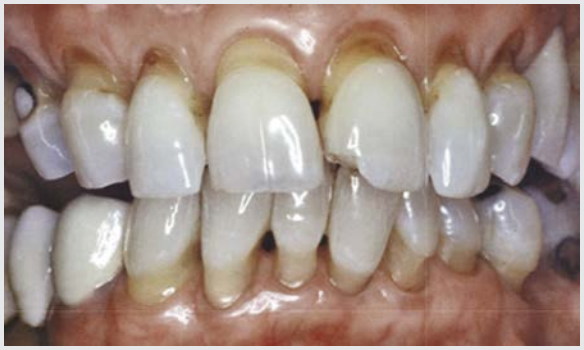
4. Alveolar bone proper—cortical bone plate that immediately lines the periodontal ligament space. Also known as:
   - *Bundle bone*—as it contains bundles of Sharpey fibers inserting into it
   - *Cribriform plate*—a histologic description, due to its porous nature that allows PDL fiber insertion and neurovascular exchange within the PDL space
   - *Lamina dura*—a radiological description denoting the thin radiopaque line that appears around the root in a radiograph
5. Supporting cancellous bone—seen surrounding the bundle bone. This may be absent on the facial aspects of teeth (especially mandibular incisors) leading to just one cortical plate (fused from the alveolar bone proper and external cortical plate) in these regions.
6. External cortical plate—made of compact lamellar bone and Haversian systems.
7. Interradicular bone—more cancellous bone is found between roots of a molar than buccally or lingually.
8. Interdental bone—comprises cancellous bone sandwiched between bundle bone of adjacent teeth; mesial physiological migration of teeth sometimes results in remodeling, and the entire interdental space may then be made up of bundle bone in various stages of formation and resorption, with very little cancellous bone.
9. Alveolar crest—this is where the external cortical plate and the alveolar bone proper meet, at usually 1.5–2 mm below the level of the cementoenamel junction of the tooth.

Structures of periosteum:

10. Inner cellular layer—this osteogenic layer contains osteogenic precursor cells and bone lining cells (flattened osteoblasts that line the bone surface).
11. Outer fibrous layer.
12. All anatomic representations are diagrammatic and meant for concept understanding and not drawn to scale.

CASE-BASED LEARNING EXERCISE

Scenario: A 72-year-old female patient presented with the chief complaint "My gums are receding." She quit smoking 20 years earlier. She did not report any systemic conditions and was not taking any medications apart from iron supplements. Patient reported flossing (but not regularly), and brushing her teeth twice a day. She had been treated for periodontitis in the past, and her current probing depths were in the range of 1–3 mm with bleeding on probing in 15% of her teeth. She also presented with generalized gingival recessions.



Clinical images are from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

Questions

1. Macroscopically and microscopically, all of the anatomic structures are part of the gingiva, EXCEPT:
   a. Gingival margin.
   b. Connective tissue.
   c. Cell rests of Malassez.
   d. Interdental papilla.
2. Which of the following functions is characteristic for gingival connective tissue?
   a. Host defense coordination
   b. Physical barrier against foreign agents
   c. High turnover of cells and collagen matrix
3. The principal fibers of the periodontal ligament are primarily type _____ collagen.
   a. I
   b. II
   c. III
   d. V
4. The percentage of organic content in cementum is:
   a. 30%–35%.
   b. 40%–45%.
   c. 50%–55%.
   d. 60%–65%.
5. Considering the increasing/advanced age of the patient, we are expecting the following periodontal changes, EXCEPT:
   a. Increased width of attached gingiva.
   b. Increased collagen content in gingival connective tissue.
   c. Increased osteogenic potential within alveolar bone.

Case-Based Learning Exercise

Solutions

1. Answer: c

Explanation: Macroscopically, the gingiva can be divided into four anatomic zones: marginal gingiva, gingival sulcus, attached gingiva, and interdental gingiva/papilla. The epithelial cell rests of Malassez exist in the periodontal ligament.

2. Answer: c

Explanation: The first two options are specific for gingival epithelium. The high turnover of cells and collagen matrix ensures good repair and regenerative potential, specific for gingival connective tissue.

3. Answer: a

Explanation: The principal fibers of the periodontal ligament are type I collagen. They are arranged in regular bundles with specific orientations (see Fig. 2.2).

4. Answer: c

Explanation: The organic content is 50%–55% and is composed of collagen fibrils, cellular elements, and calcified matrix. The inorganic content is primarily hydroxyapatite (45%–50%).

5. Answer: c

Explanation: Aging is associated with all of the listed periodontal changes except option c. Aging is, in fact, associated with a reduction in osteogenic potential.

This chapter was developed from Chapters 3 and 4 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Reference

1. Bosshardt, D. D., & Selvig, K. A. (1997). Dental cementum: the dynamic tissue covering of the root. *Periodontology, 2000, 13,* 41–75.

3 Periodontal Disease Classification

Relevant Terminology

Terminology/abbreviation	Explanation
aggressive periodontitis	Term used prior to the "2017 World Workshop of Periodontal and Peri-implant disease classification", to characterize cases with rapid disease progression in otherwise healthy individuals. • Often applied to young adults with severe established periodontal bone destruction • Not to be confused with rapid rate of disease progression due to underlying medical conditions (see *periodontitis as a manifestation of systemic disease*) • In the current classification scheme, most cases of "aggressive periodontitis" would be classified as "Grade C" periodontitis • In the new classification, if disease is localized aggressive, the term "molar-incisor pattern" will be affixed to the stage of the disease to describe the extent that is unique to this condition (e.g., periodontitis with molar-incisor pattern, Stage III, Grade C)
endodontic-periodontal lesion	Pulpal infection resulting in the destruction of the periodontal ligament and the adjacent alveolar bone.
gingivitis	Common inflammatory condition of the gingiva, associated with retained dental plaque (biofilm) without alveolar bone loss.
mucogingival deformities and conditions	Significant departures from the normal shape of the gingiva and alveolar mucosa, which may involve the underlying alveolar bone, e.g., gingival recessions, lack of keratinized gingiva, pseudo (gingival) pockets.
necrotizing periodontal diseases	Characterized by acute manifestation, often accompanied by systemic symptoms (e.g., fever). They are painful lesions that invariably affect the interdental papillary tissues. Due to the resulting necrosis and destruction of junctional epithelium, deepening of pocket is not a characteristic feature (a viable junctional epithelium is required for pocket formation and deepening).
periodontal-endodontic lesions	Bacterial infection from a periodontal pocket leads to loss of attachment and root exposure where inflammation spreads to the pulp via lateral and accessory canals, resulting in pulpal necrosis (retrograde pulpitis).
periodontitis	An inflammatory disease of the supporting tissues of the teeth initiated by specific microorganisms or groups of microorganisms, resulting in host-induced progressive destruction of the periodontal ligament and alveolar bone with increased probing depth recession, or both.
periodontitis as a manifestation of systemic disease	Refers to a distinct group of hematologic and genetic disorders that have been associated with the development of periodontitis in affected individuals. These conditions are not responsive to conventional periodontal therapy unless the underlying medical condition is managed (where possible).
pseudopocket	Gingival excess caused by inflammation resulting in deepening of the sulcus coronal to the cementoenamel junction that may be misinterpreted as a periodontal (true) pocket.

Fast Facts

New classification overview	• In 2017 the American Academy of Periodontology and the European Federation of Periodontology convened periodontal experts from around the world to develop updated definitions for periodontal health, gingival disease, periodontitis, periodontal manifestations of systemic diseases, and peri-implant diseases. This new classification system supersedes the previous 1999 Classification of Periodontal Diseases and Conditions • The current classification of periodontal diseases is based on a multidimensional staging and grading system • Staging (stages I through IV) is predominantly determined by the severity of the disease at time of presentation • Grading (grades A through C) is linked to the risk of progressive periodontitis • Peri-implant conditions are part of the classification and are stratified as peri-implant health, peri-implant mucositis, peri-implantitis, and peri-implant soft and hard tissue deficiencies • A classification of mucogingival recessions by Cairo et al.[1] has been embraced and is based on the assessment of gingival margin level in relation to the interdental tissues adjacent to the recession defects
Key diagnostics for gingival diseases	• Gingival diseases may occur on a periodontium with no attachment loss, or on a periodontium with attachment loss that is stable (i.e., reduced periodontium, like for e.g., in a treated case) and not currently associated with active bone loss • The key elements that lead to a diagnosis of gingivitis are visual changes of the dental gingiva (edema, erythema, bulbous papillae) and signs of bleeding upon sulcular probing with a periodontal probe • Although rarer, non-plaque-induced gingival diseases can manifest in the gingiva with signs of inflammation, usually as a result of autoimmune or idiopathic etiology
Key diagnostics for periodontal diseases	• The clinical feature that distinguishes periodontitis from gingivitis is the presence of clinically detectable attachment loss as a result of inflammatory destruction of the periodontal ligament and alveolar bone • Probing depth measurement alone is inadequate for an assessment of periodontitis, because recession of the marginal gingiva may underestimate attachment loss. Conversely, if the gingival margin is located above the cementoenamel junction as a result of inflammation, measurements of increased probing depth may not reflect true bone loss (see *pseudopocket* in terminology list)
Key diagnostics for peri-implant diseases	• Peri-implant health is characterized by an absence of visual signs of inflammation and bleeding on probing • Health is challenging to define around implants because implant placement and restoration parameters determine the healthy peri-implant tissue dimensions for each unique site. Thus the best predictor of disease is relative change, determined based on comprehensive baseline records of the loaded implant; progressive bone loss in relation to the radiographic bone level assessment at 1 year following the delivery of the definitive restoration is the diagnostic sign that bears most weight for diagnosing peri-implantitis. • In the absence of initial radiographs and probing depths, radiographic evidence of bone loss ≥3 mm and/or probing depths ≥6 mm in conjunction with profuse bleeding represents peri-implantitis.
Periodontitis associated with endodontic lesions	• In most cases, pulpal infection precedes periodontal lesions (i.e., endodontic-periodontal lesions). It is advisable to consider endodontic therapy as a first line of intervention prior to periodontal interventions
Localized tooth-related factors	• Isolated cases of periodontal lesions in an otherwise healthy periodontium may be due to tooth anatomic factors, restorations, or fractures. Prior to initiating periodontal therapy, the possible involvement of factors such as cervical enamel projections, palatal grooves, and enamel pearls must be considered
Medication-related osteonecrosis of the jaw (MRONJ)	• Updated term that has replaced the phrase *bisphosphonate-related osteonecrosis of the jaw (BRONJ)* to include the increasing list of medications that may lead to osteonecrosis. Because there is no effective treatment for MRONJ, a thorough medical history and updated medication list prior to every periodontal intervention is of paramount importance

TABLE 3.1 1999 Diagnostic Categories and Their Incorporation Into the New Classification System[2]

2017 Classification System (4 categories)	Periodontal Diseases and Conditions			
	Periodontal Health, Gingival Diseases and Conditions	Periodontitis	Other Conditions Affecting the Periodontium	Periimplant Diseases and Conditions
1999 International Classification System (8 categories): incorporation under categories in new system	I. Gingival Diseases	II. Chronic Periodontitis III. Aggressive Periodontitis IV. Periodontitis as a Manifestation of Systemic Diseases V. Necrotizing Periodontal Diseases	VI. Abscesses of the Periodontium VII. Periodontitis Associated with Endodontic Lesions VIII. Developmental or Acquired Deformities and Conditions	– (newly introduced category for 2017)

Core Knowledge

Introduction

In order to accommodate new advances in knowledge and paradigm shifts that have come to light since the 1999 International Classification of Periodontal Diseases, the 2017 World Workshop Classification system for periodontal and periimplant diseases and conditions was drawn up as a joint effort of the American Academy of Periodontology (AAP) and European Federation of Periodontology (EFP). The consensus reports from this workshop proposed a new classification system that was officially released and published in 2018. This chapter provides updates, insights, and rationale for the new system compared with the 1999 classification system.

The New Classification Scheme for Periodontal and Periimplant Diseases and Conditions: Objectives and Comparisons with Old System

The objectives of the new classification system were:

- To create a simple classification system that could be implemented in general dental practice;
- To create a system that accounts for both *current* periodontal status (assessed by staging periodontal disease) and *future* susceptibility to periodontal disease (assessed by grading periodontal disease);
- To create a system that takes into account treatment planning customized to individual patient scenarios;
- To create a live/dynamic system that can accommodate regular updates and incorporate future knowledge (for example, biomarkers) emerging from research.

The 1999 international classification system had eight major categories; the 2017 system arranges periodontal and periimplant diseases and conditions into four major categories:

1. periodontal health, gingival diseases and conditions
2. periodontitis
3. other conditions affecting the periodontium
4. periimplant diseases and conditions

Table 3.1 explains how the 1999 classification was incorporated into the new classification system.

The complete 2017 classification of periodontal and periimplant diseases and conditions is discussed in Table 3.2.

Periodontitis: Classification and Diagnosis

The main change from current practice is that a complete diagnosis of a patient with periodontitis will include staging and grading of the disease. Determining a patient's current disease status (by staging) and future disease susceptibility (by grading) represents important steps, especially in patients who have received periodontal therapy in the past. Several considerations are employed in this process (Tables 3.3 and 3.4):

1. **Stages**—disease is categorized into four stages based on:
   - Severity (measured by clinical attachment loss at site with greatest loss or evidence of radiographic bone loss/ tooth loss)
   - Complexity of management (measured by probing depth, pattern of bone loss, furcation lesions, tooth mobility, number of remaining teeth, etc.)
2. **Extent and distribution**:
   - Localized (< 30% teeth)
   - Generalized (> 30% teeth)
   - Molar-incisor pattern
3. **Grades**—categorized into three grades based on risk of rapid progression (using direct measures such as radiographic bone loss or clinical attachment loss, and indirect measures such as bone loss/age ratio).

Establishing a diagnosis for periodontitis involves two steps:

1. Determination of the extent of periodontitis followed by staging and grading (e.g., localized periodontitis, Stage II, Grade B)
2. Risk factor documentation (e.g., Type 2 diabetes [HbA1c 6.9%] and current smoking [8 cigarettes/day])

CLINICAL CORRELATE

What Are the Major Changes in the New Classification System That a Clinician Must Be Aware of While Diagnosing and Managing Periodontal Conditions?

There are four major changes to be kept in mind:

1. For the first time, the new classification system defines periodontal health and gingivitis for patient with[3]:
   - an intact periodontium
   - a reduced periodontium due to causes other than periodontitis
   - a reduced periodontium due to periodontitis
2. "Chronic and aggressive periodontitis" terminologies have been removed because there is very little evidence to support their existence as separate entities. They are now thought to be variations along a spectrum of the same disease process: periodontitis.
   - The exception to this rule is the case of the classical localized juvenile (aggressive) periodontitis. Here a clearly defined clinical phenotype exists; however, it still does not warrant a separate category. It is hence considered under the description of "extent" of periodontitis called "molar-incisor pattern," in addition to "localized" and "generalized" periodontitis.
   - Staging (process designed to assess disease severity at the time of presentation) and grading (process designed to assess disease susceptibility in future; risk profiling included) periodontitis are a vital part of the process of diagnosis and classification, for it provides guidance during treatment planning.
3. A major change has occurred in the classification of mucogingival deformities and conditions. For example, with regard to gingival recessions, the previous classification was more descriptive in nature and involved an assessment of a defect's relation to the mucogingival junction and radiographic assessment of interdental bone. The current classification is evidence-based and classifies recessions based on predictability of recession coverage using contemporary periodontal plastic surgery procedures.
4. A classification category for peri-implant diseases and conditions has been included for the first time in a periodontal classification system.

TABLE 3.2 **2017 Classification of Periodontal and Periimplant Diseases and Conditions[1]**

Periodontal Diseases and Conditions			
Periodontal Health, Gingival Diseases and Conditions	**Periodontitis**	**Other Conditions Affecting the Periodontium**	**Periimplant Diseases and Conditions**
Periodontal and gingival health: • Intact periodontium • Reduced periodontium	Necrotizing periodontal diseases: • Gingivitis • Periodontitis • Stomatitis	Systemic diseases/conditions affecting periodontal tissues	• Periimplant health • Periimplant mucositis • Periimplantitis • Periimplant soft and hard tissue deficiencies
Gingivitis: dental biofilm–induced : • Dental biofilm–induced • Systemic and local risk factors mediated • Drug-influenced gingival enlargement	Periodontitis: • Staging: 1–4 • Extent: localized, generalized, molar-incisor pattern • Grading: A,B,C	Other periodontal conditions: • Periodontal abscesses • Endo-perio lesions	
Gingival diseases: non–dental biofilm–induced: • Genetic/developmental • Infections • Inflammatory/immune conditions • Reactive processes • Neoplasms • Endocrine/metabolic diseases • Traumatic lesions • Gingival pigmentation	Periodontitis as a manifestation of systemic diseases	Mucogingival deformities and conditions: • Gingival phenotype • Gingival recession • Decreased vestibular depth • Aberrant frenum/muscle pull • Gingival excess Traumatic occlusal forces: • Primary • Secondary • Orthodontic forces Tooth- and prosthesis-related factors	

Adapted from Caton, et al.[2] with permission.

TABLE 3.3 Key Periodontitis Staging Elements

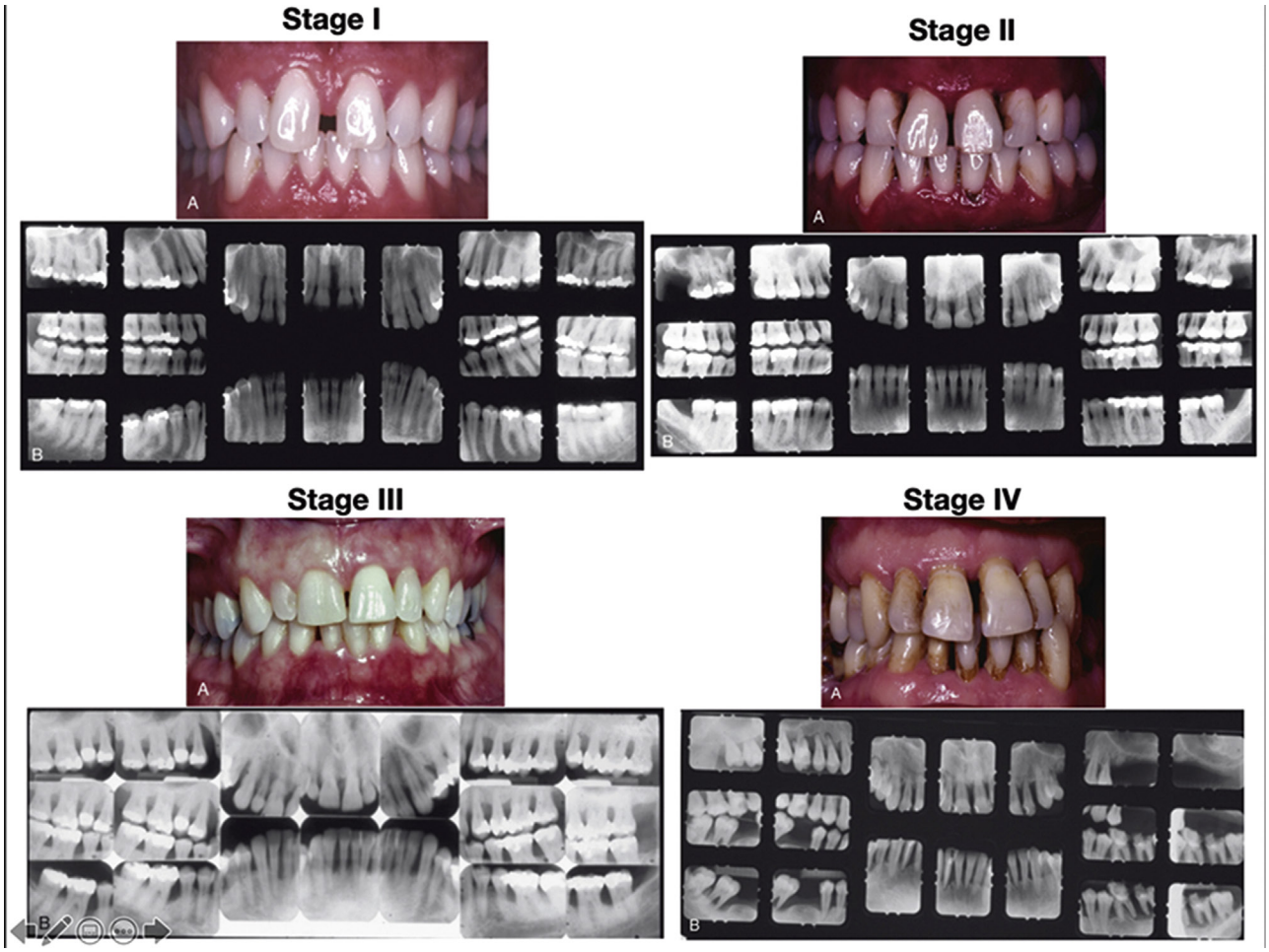
	Periodontitis	Stage I	Stage II	Stage III	Stage IV
Severity	Clinical attachment loss	1–2 mm	3–4 mm	≥5 mm	≥5 mm
	Radiographic bone loss	Coronal third of the root	Coronal third of the root	Middle or apical third of the root	Middle or apical third of the root
	Tooth loss due to periodontitis	No tooth loss	No tooth loss	≤4 teeth	≥5 teeth
Complexity		• PD ≤4 mm • Mostly horizontal bone loss	• PD ≤5 mm • Mostly horizontal bone loss	In addition to Stage II: • PD ≥6 mm • Vertical bone loss ≥3 mm • Class II or III furcation involvement • Moderate ridge defects	In addition to Stage II: • Need for complex rehabilitation due to masticatory dysfunction, tooth mobility, bite collapse, pathologic migration, <20 remaining teeth
Extent and Distribution	• Localized (<30% of the teeth involved) • Generalized (≥30% of the teeth involved) • Molar-incisor pattern				

Adapted from Tonetti, M.S., et.al.[4] with permission.
PD, probing depth.

TABLE 3.4 Key Periodontitis Grading Elements

	Progression		Grade A: Slow Rate	Grade B: Moderate Rate	Grade C: Rapid Rate
Primary criteria (Direct evidence should be used when available)	Direct evidence of progression	Radiographic bone loss or CAL	No loss over 5 years	<2 mm over 5 years	≥2 mm over 5 years
	Indirect evidence of progression	% bone loss/age	<0.25	0.25–1	>1
		Case phenotype	Heavy biofilms with low levels of destruction	Destruction commensurate with biofilm deposits	Destruction inconsistent with biofilm deposits; clinical patterns suggestive of periods of rapid progression and/or early onset
Grade modifiers	Risk factors	Smoking	Non-smoker	<10 cigarettes/day	≥10 cigarettes/day
		Diabetes	Non-diabetic	Diabetic with HbA1c<7%	Diabetic with HbA1c≥7%

Adapted from Tonetti, M.S., et.al.[4] with permission.
CAL,clinical attachment loss.



• **Fig. 3.1** Clinical and Radiographic Images Depicting The Various Stages of Periodontitis Based on The 2017 Disease Classification. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

Hence the diagnostic statement will carry all the required information of classification, current disease extent and severity, future susceptibility, and risk factor assessment; it will look like this:

Diagnosis: Localized periodontitis, Stage II, Grade B modified by Type 2 diabetes and smoking.

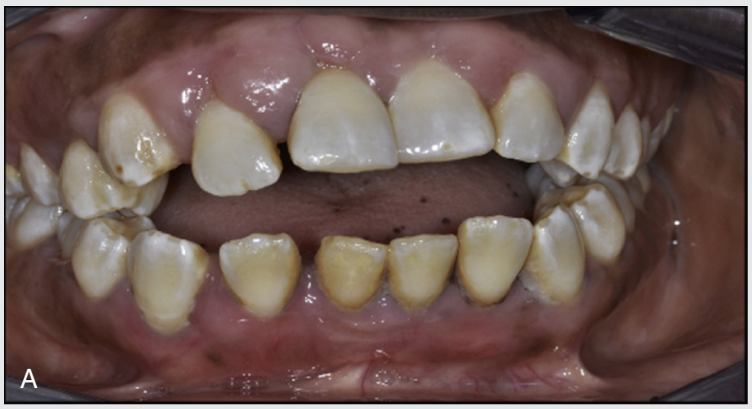
Clinical and radiographic images depicting the various stages of periodontitis are presented in Fig. 3.1.

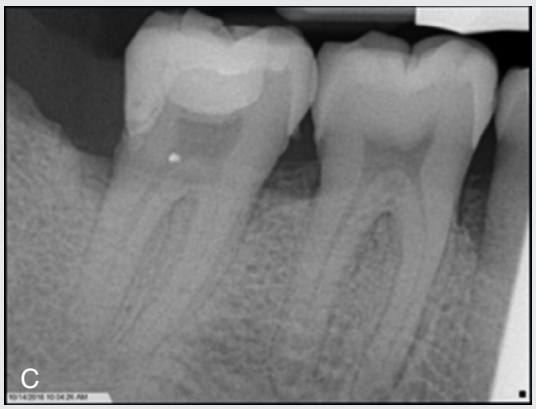
Conclusion

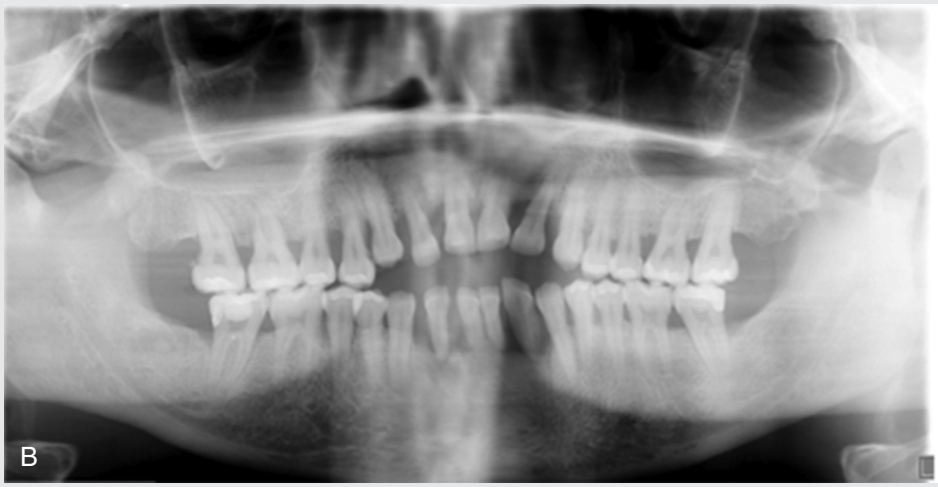
This chapter has provided an overview of the current classification system and the rationale behind the newer categorizations. The reader is referred to the textbook chapter (Chapter 5) as well as the official classification workshop proceedings for detailed reading of the individual entities described under every category.

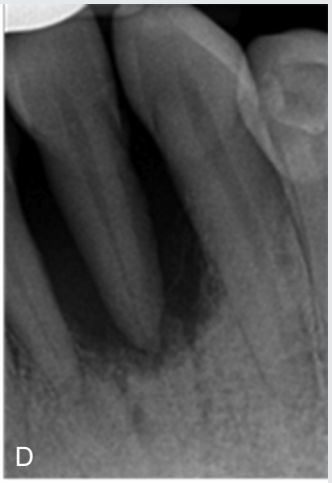
CASE-BASED LEARNING EXERCISE

Scenario: A 37-year-old female presented with the chief complaint: "My gums are bleeding and are tender. My teeth are very loose too." She worked as a nurse and had noticed major changes in her dentition in the past 3 years. She reported a medical history of hypertension that was initially treated with amlodipine and later switched to lisinopril. Clinical findings were (A): generalized gingival enlargement with deep probing depths ranging from 6 to 11 mm, generalized bleeding on probing, generalized mobility, secondary occlusal trauma, furcation involvement and deposits of plaque, and calculus. Radiographic findings were (B, C and D): generalized slight-to-moderate and localized areas of severe horizontal bone loss, especially in the maxillary and mandibular anterior regions.









Questions

1. Identify which is NOT a major category in the 2017 classification.
   a. Periodontal health, gingival diseases and conditions
   b. Periodontitis
   c. Periodontal manifestations of systemic diseases and acquired conditions
   d. Occlusal trauma

2. When assessing the severity of periodontitis, what clinical parameter do we consider?
   a. Mobility
   b. Bleeding on probing
   c. Interproximal attachment loss
   d. Furcation involvement

3. Identify the severity stage of bone loss as seen in the radiograph (Figure C) for tooth #31.
   a. Stage I
   b. Stage II
   c. Stage III
   d. Stage IV

4. Based on the clinical and radiographic presentation (Figures A, B and C), what will be the appropriate grading for this patient?
   a. Grade A
   b. Grade B
   c. Grade C

Case-Based Learning Exercise

Solutions

1. Answer: d

Explanation: The four main categories are A, B, C, and Periimplant Diseases and Conditions. Occlusal trauma used to be a major category in the 1999 classification, but has now been added as Traumatic Occlusal Forces and is part of category C (subcategory: Other Conditions Affecting the Periodontium).

2. Answer: c

Explanation: The main change from the 1999 classification is that a complete diagnosis of periodontitis will include staging and grading of the disease. Severity is measured by interdental clinical attachment loss at the site with greatest radiographic bone loss.

3. Answer: b

Explanation: When reviewing the radiograph, we can notice that the apical portions of the intra-bony defects around tooth #31 extend up to the coronal third (15%–33%) of the root; this is considered Stage II, according to the 2017 classification.

4. Answer: c

Explanation: When evaluating the clinical and radiographic presentation, considering the percentage bone loss in the past 3 years and the relatively young age of the patient, this is considered rapid progression or grade C.

This chapter was developed from Chapter 5 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

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4

Periodontal Disease Pathogenesis

Relevant Terminology

Terminology/abbreviation	Explanation
anaerobic bacteria	Bacteria that thrive in the absence of oxygen.
antigen presentation	T cells recognize only fragmented antigens displayed on cell surfaces; therefore, in order to stimulate adaptive immunity, antigen-presenting cells (APC) must digest, process, and present these antigens in conjunction with MHC-II molecules on their surface.
antigen-presenting cells	Professional antigen-presenting cells are macrophages, dendritic cells, and Langerhans cells.
B cells	Cells of adaptive immune response, derived from bone marrow, that differentiate into *plasma cells.* They are responsible for humoral immunity.
chemotaxis	Movement of a cell corresponding to a gradient in concentration of a particular substance. For example, PMNs move from areas with lesser concentration toward areas with increased concentrations of IL-8.
clinical attachment level (CAL)	The distance from the cementoenamel junction to the tip of the periodontal probe during periodontal diagnostic probing.
complement system	(Innate immunity.) Soluble protein effector molecules synthesized mainly in the liver and circulating in blood; a group of enzymes involved in a cascade reaction, ultimately producing the *membrane attack complex (MAC)*, which causes pore formation in bacterial cell walls (bactericidal action). By-products of the cascade reaction are *opsonins* and *chemotaxins.*
dysbiosis	Microbial imbalance where the usually dominant beneficial bacterial species are outcompeted to accommodate harmful species that grow in influence or number.
fibroblasts	Cells residing within connective tissue that are primarily responsible for collagen turnover (formation and destruction).
gingipains	Microbial virulence factors usually secreted by the bacterium *Porphyromonas gingivalis*. Group of enzymes that can degrade host proteins (proteases). Major forms identified are lysine-specific gingipains (Kgp) and arginine-specific gingipains (RgpA and RgpB).
gingival crevicular fluid (GCF)	Tissue fluid that seeps through the JE and sulcus; increased flow is seen during inflammation.
interleukins (IL)	Cytokines. Class of glycoproteins produced by leucocytes for regulating immune responses. They can be proinflammatory (e.g., IL-1) or antiinflammatory (e.g., IL-10).
junctional epithelium (JE)	Stratified squamous nonkeratinizing epithelium that forms the gingival seal around teeth. It forms the base of the gingival sulcus and is the primary mechanical barrier against sulcular plaque microbes.
lipopolysaccharide (LPS)	Cell wall component of gram-negative bacteria. Also called *endotoxin*. Extremely antigenic.
lipoteichoic acid (LTA)	Cell wall component of gram-positive bacteria. Also called *exotoxin*.
macrophage	When monocytes migrate from blood into tissues, they differentiate into macrophages that get involved in phagocytosis and the processing and presentation of antigens.

Continued

Relevant Terminology—cont'd

Terminology/abbreviation	Explanation
matrix metalloproteinases (MMP)	Group of enzymes that destroy host structural proteins (collagen fibers, extracellular matrix components); primarily released by PMNs and fibroblasts. Inhibited by TIMPs and the tetracycline group of antibiotics.
osteoprotegrin (OPG)	Decoy receptor that inhibits osteoclastogenesis by interfering with RANK-RANKL interactions and stopping the differentiation of progenitor cells into bone-resorbing cells (osteoclasts).
periodontal ligament (PDL)	Soft tissue connection that attaches tooth to alveolar bone proper; composed of collagen fibrils oriented in different directions to accommodate forces exerted on the tooth.
phagocytosis	Ingestion of bacteria, dead cells, etc. by a cell (phagocyte) either to clear debris or to present certain components of the ingested materials as antigens to immune cells. Examples of phagocytes include PMNs and macrophages.
plasma cells	Cells of the adaptive immune response "(humoral immunity)", derived from B cells that secrete *immunoglobulins* (antibodies).
pocket epithelium	Stratified squamous epithelium that forms the soft tissue wall of a deepened sulcus or pocket. Can be derived from both SE and JE cells.
polymorphonuclear leucocyte (PMN)	Neutrophil; granular leukocyte involved in phagocytosis. First line of defense (innate immunity); "respiratory burst" action releases oxidative radicals and lytic enzymes that cause tissue destruction.
receptor activator of nuclear factor-κB (RANK)	Molecule that binds to RANK on preosteoclasts. Can also bind to the decoy receptor OPG.
RANK ligand (RANKL)	Receptor on the surface of osteoclastic progenitor cells. When activated by ligands, stimulates further differentiation of preosteoclasts into osteoclasts (bone-resorbing cells).
sulcular epithelium (SE)	Stratified squamous nonkeratinizing epithelium that forms the soft tissue lateral wall lining of the gingival sulcus and links JE with the keratinized oral epithelium.
symbiosis (in the context of periodontal pathogenesis)	A relationship between host and microbe from which both derive benefit. Normally, microbes can be both beneficial and harmful to the host. A *symbiotic climax community* is a mature plaque biofilm in which beneficial bacteria dominate by outcompeting the harmful ones.
T cells	Cells of the adaptive immune response, derived from the thymus, that differentiate into *helper cells*, *cytotoxic cells*, and *regulator cells*. They are responsible for *cell-mediated immunity*.
tissue inhibitors of MMPs (TIMP)	Molecules that inhibit MMPs.
virulence	The degree of harmfulness (or) severity of disease-causing ability.

Fast Facts

Periodontal disease pathogenesis	• The word *pathogenesis* is defined as "the origination and development of a disease" • Periodontal disease pathogenesis or periodontal etiopathogenesis refers to the step-by-step process by which an etiologic factor (or factors) causes the disease (i.e., a series of changes in the structure and function of the periodontium)
Gingivitis versus periodontitis	• Gingivitis precedes periodontitis, but not all cases of gingivitis progress to periodontitis • In gingivitis, the inflammatory lesion is confined to the gingiva; in periodontitis, the inflammatory lesion involves the gingiva, periodontal ligament, and alveolar bone
Host-microbe interplay	Periodontal disease results from a complex interplay between the subgingival plaque biofilm and the response of host gingival and periodontal tissues (immune-inflammatory events) to plaque challenge. The tissue damage resulting from this interaction is clinically called *periodontitis*.
Dysregulated immune-inflammatory response	Subgingival plaque biofilm presents a chronic low-grade infection to which the normal host mounts a low-grade inflammatory response that is protective in nature. However, a disease-susceptible host mounts an excessive or dysregulated immune-inflammatory response for a similar bacterial challenge, leading to increased tissue breakdown compared with individuals who have a more normal inflammatory response.
Net result of periodontitis	The net result of inflammatory changes within periodontal tissues is the breakdown of the fibers of the periodontal ligament, resulting in clinical loss of attachment and alveolar bone resorption. If left untreated, periodontitis will result in tooth loss.

Core Knowledge

Introduction

Periodontitis is a complex disease process that develops in a nonlinear fashion; that is, small causes result in disproportionately large effects. The pathogenetic process discussed in this chapter includes, in a step-by-step manner, the series of structural and functional changes within the periodontium that cause disease. Some factors to be kept in mind while discussing periodontal disease pathogenesis are:

- Bacteria must be present to initiate periodontal disease, but bacteria alone are not solely responsible for this disease.
- What begins as a protective host response (inflammatory reaction) to plaque accumulation fails to resolve in susceptible individuals, resulting in a shift to chronic infection.

CLINICAL CORRELATE

Why do we need to study the pathogenesis of a disease and remain updated on current scientific thinking about it?

Pathogenesis refers to the process of disease development. At any given point in time, the treatment strategies used to manage a disease are based on the prevailing understanding of its pathogenesis. The treatment/management options that are taken for granted at one point in time may change in future. This is because the rationale behind tackling disease pathogenesis changes with more research, and our understanding of a disease improves with focused efforts undertaken by a responsible scientific community. Hence it is vital to stay abreast of the current scientific thinking on the subject of pathogenesis.

Current thinking therefore emphasizes that periodontal tissue destruction is more a result of the inability of the host to resolve inflammation rather than a direct result of the initial inflammation itself.

Histopathology of Periodontal Disease

In 1976, Page and Schroeder attempted to describe the histologic appearance of periodontal tissues in various stages of disease development (Fig. 4.1).[1] These are histologic descriptions only, and should not form part of a clinical diagnosis. The histopathological stages described approximately correspond to four clinical scenarios:

1. Initial lesion—clinically normal gingiva
2. Early lesion—gingival crevicular fluid (GCF) is detectable; can last indefinitely in children but quickly progresses to established lesion in adults
3. Established lesion—pocket epithelial ulcerations manifest as bleeding on probing; can last indefinitely without progression to the next stage in disease-resistant adults
4. Advanced lesion—clinical loss of attachment presents with or without radiographic evidence of bone loss.

CLINICAL CORRELATE

How Does a Periodontal Pocket Develop?

Step 1: Inflammation of the gingival tissues caused by microbial pathogens results in swelling and increased probing depth.
Step 2: Spreading inflammatory response causes breakdown of collagen in the connective tissues.
Step 3: Apical migration of the junctional epithelium into collagen-depleted areas occurs to maintain an intact epithelial barrier that seals around the tooth; the resulting increase in probing depth with deeper sulcus (which may be associated with alveolar bone loss) is referred to as a periodontal pocket.
Step 4: Microbial pathogens exploit the conducive (anaerobic) microenvironment within the deepened sulcus and perpetuate the disease with the apical advancement of the pocket.

Inflammatory Responses in the Periodontium

While prolonged accumulation of undisturbed subgingival plaque can cause the release of inflammatory molecules from both microbes and the host (Fig. 4.2), it is now clear that most of the tissue breakdown results from the host's dysregulated inflammatory processes. Inflammatory responses can be:

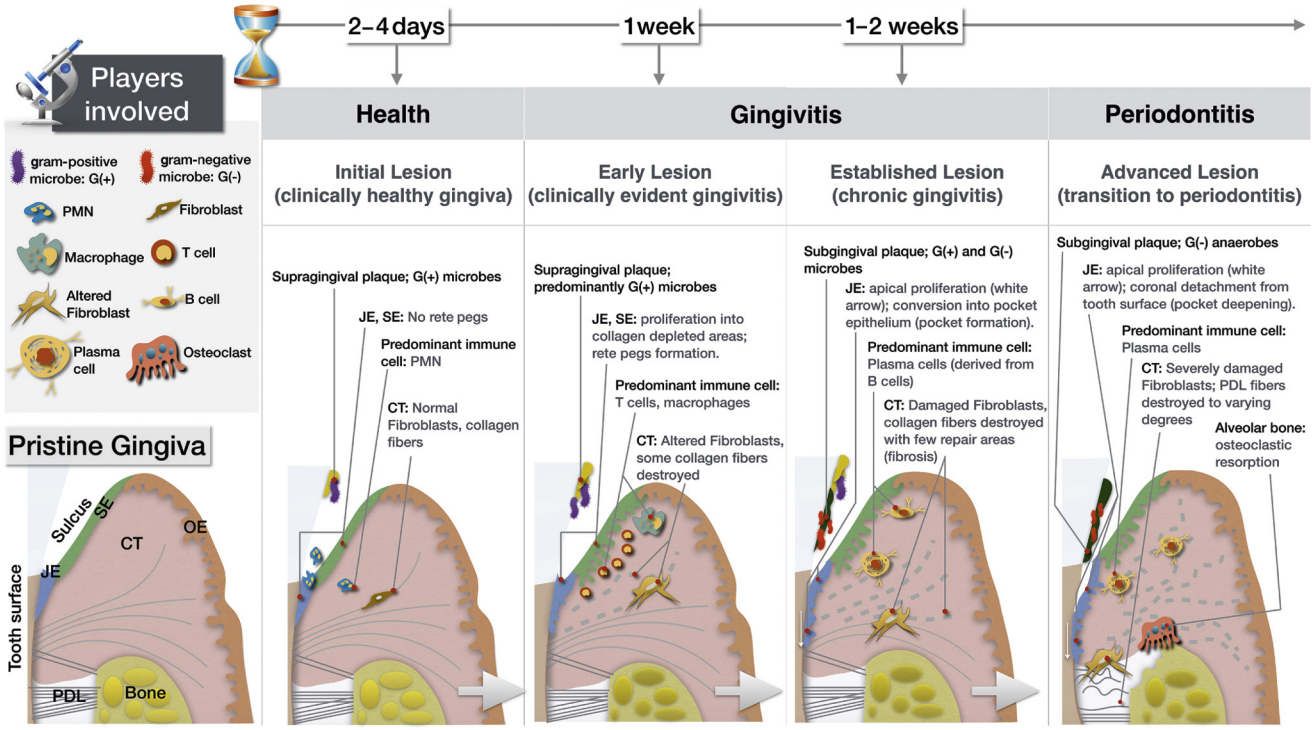
- **Acute (PMN hyperactivity)**—abscesses and acute inflammation mainly occur when PMNs are activated by very high levels of chemokines within periodontal tissues. During the process of "respiratory burst," they release massive amounts of lytic enzymes that mediate host tissue destruction.
- **Chronic (macrophage hyperactivity)**—in chronic conditions, bacterial metabolites stimulate the macrophage–T cell axis, which in turn alters resident fibroblasts and causes them to secrete tissue-destroying secondary mediators (e.g., MMPs, PGE_2).

The reader is referred to the section on inflammatory responses in the periodontium in the textbook chapter (Chapter 7) for detailed information on individual molecules and their role in periodontal disease pathogenesis.

Linking Pathogenesis to Clinical Signs of Periodontal Disease

While the process of periodontal connective tissue destruction and bone resorption is complicated and involves many cells, molecules, and regulatory mediators, the major "players" can be identified as follows (Fig. 4.3):

- **Instigating molecules**—LPS, LTA (bacterial origin); IL-1, TNFα (host derived).
- **Effector cells**—fibroblasts, osteoclasts.
- **Effector molecules**—MMPs (connective tissue breakdown); PGE_2 (osteoclastic bone resorption)



• **Fig. 4.1** Histopathology of Periodontal Disease. This figure displays the various histopathological stages during periodontal disease development.

☐ The top left corner box shows the key to the various cells involved in periodontal disease pathogenesis.

☐ The lower leftmost diagram shows the histologic appearance of the periodontium under *pristine* conditions, displaying the location of the junctional epithelium (JE), sulcular epithelium (SE), oral epithelium (OE), subepithelial connective tissue (CT), periodontal ligament (PDL), and alveolar bone structure.

☐ The figure displays and describes the few main zones where histopathologic changes occur during pathogenesis—namely, plaque zone, gingival sulcus zone, JE/SE zone, subepithelial connective tissue zone, and PDL/alveolar bone zone. When plaque is left undisturbed for a certain time period, there are four progressive stages observed histologically: initial lesion, early lesion, established lesion, and advanced lesion. These histologic descriptions emphasize how the progressive massive infiltration of tissues by immune/inflammatory cells leads to extracellular release of destructive enzymes, resulting in disruption of connective tissue anatomy (collagen depletion) and subsequent proliferation of JE cells into depleted areas, reflecting clinically as attachment loss and pocket formation.[1] Note: This is a diagrammatic representation only and not to exact scale of actual microscopic cellular events. B cells, bone marrow–derived lymphocytes; PMN, polymorphonuclear leucocytes/neutrophils; T cells, thymus-derived lymphocytes.

Bone Resorption and Remodeling

It is now clear that RANKL and osteoprotegerin are the key regulators of bone remodeling and are directly involved in the differentiation, activation, and survival of osteoclast precursors and osteoclasts.

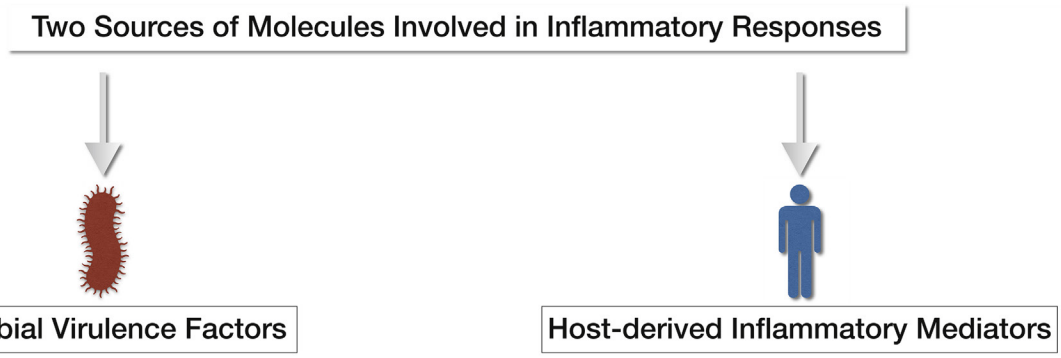
The RANK/RANKL/OPG system comprises:

- **Receptor: RANK**—receptor on preosteoclasts that needs to be activated for their further differentiation into mature osteoclasts
- **Ligand: RANKL**—molecule required to attach to RANK to activate differentiation into osteoclasts that add to the pool of bone-resorbing cells
- **Decoy receptor: OPG**—binds to RANKL. Prevents differentiation of progenitor cells into osteoclasts by antagonizing RANK-RANKL interaction
- **RANKL:OPG ratio**—the relative amounts of these two molecules regulates bone turnover; for example, when RANKL is higher, the ratio is higher and the balance tilts in favor of bone resorption

CLINICAL CORRELATE

What is meant by the term "attachment loss"? Which periodontal tissue is most referred to when attachment loss is mentioned?

The term "attachment loss" refers to destruction of periodontal ligament (PDL) fibers, which begins at the most coronal end and progresses in any direction (though predominantly apical progression may be seen), and the phenomenon of the tooth losing its anchorage within the bony socket because of losing periodontal ligament fiber attachment. The crucial feature to be considered here is collagen destruction without further replacement of destroyed fibers. While bone resorption may also occur due to spreading inflammation, this bone loss need not always be a part of attachment loss. Hence the periodontal tissue most affected by attachment loss is the PDL.



• **Fig. 4.2** Sources of Molecules Involved in Inflammatory Responses Within the Periodontium. The subgingival plaque biofilm can directly release tissue-damaging noxious products, but more important for the pathogenesis of periodontal disease is the fact that this biofilm induces host immune-inflammatory responses within the periodontium that can also cause tissue damage; this damage is perhaps more destructive than that caused directly by bacteria. The figure lists the most important molecules involved in inflammatory responses within the periodontium, which are derived from both microbes and host.[2] LPS, lipopolysaccharide/endotoxin; LTA, lipoteichoic acid; Kgp, lysine-specific gingipain; Rgp, arginine-specific gingipain; H_2S, hydrogen sulfide; PGE_2, prostaglandin E_2.

Resolution of Inflammation

"Turning off" inflammation is an active process, rather than simply a passive dwindling of proinflammatory signals; it is a vital step in restoring tissue homeostasis once the offending agents have been dealt with. When inflammation is not regulated or "switched off," dysregulated inflammatory and immune responses continue to cause collateral damage to host tissues. Resolution of inflammation is mediated by specific molecules, including a class of endogenous, proresolving lipid mediators that could potentially offer new adjunctive treatments for the management of periodontitis. The reader is referred to Chapter 7 of this book for further review of this topic.

Mode of action:

- Inhibition of PMN infiltration and transmigration
- Stimulation of monocyte infiltration and macrophage phagocytosis of dead cells without stimulating the release of inflammatory cytokines

Mediators of resolution of inflammation:

- lipoxins
- resolvins
- maresins
- protectins

Immune Responses in Periodontal Pathogenesis

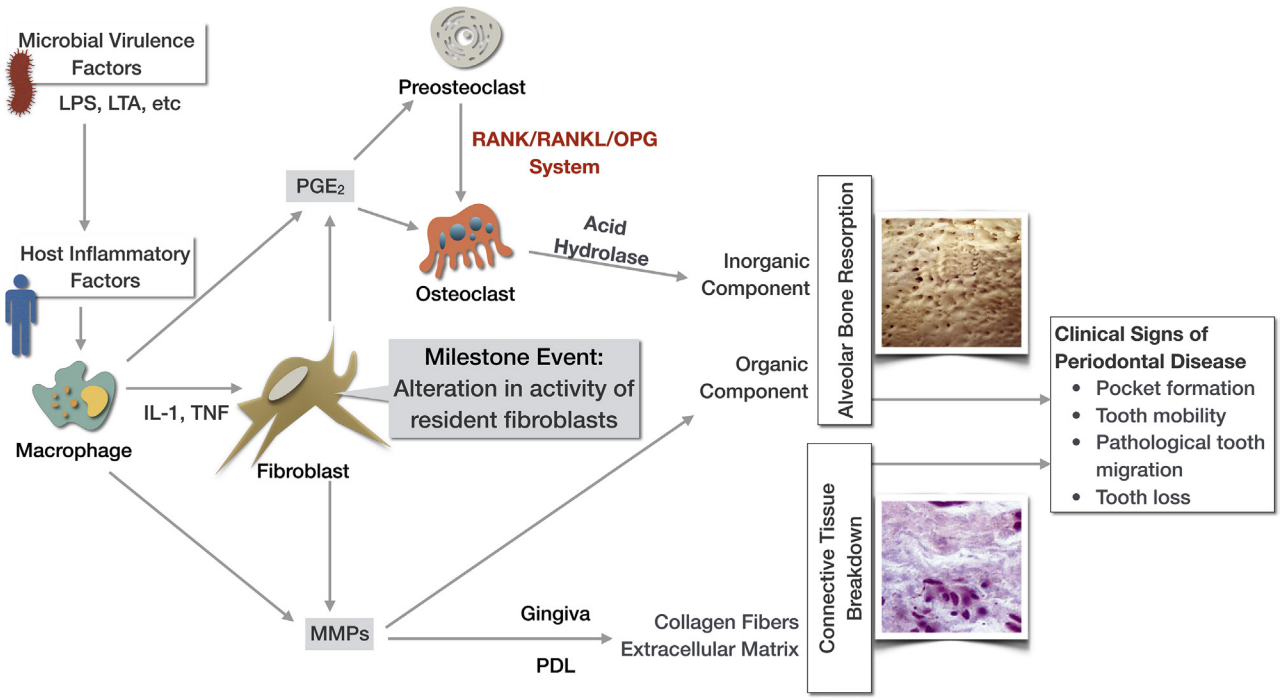
Immune-inflammatory responses are initially dominated by PMNs and macrophages (effector cells of innate immunity).

PMN actions against bacteria include:

- **Diapedesis**—moving out of blood vessels and into tissues
- **Chemotaxis**—moving toward sites of infection
- **Phagocytosis**—engulfing, lysing, digesting antigens
- **Oxidative burst**—rapid release of oxygen radicals with bactericidal activity
- **Inflammatory mediator secretion**—prostaglandins (PGE_2), leukotrienes (LTB_4), cytokines (IL-1)

Macrophage actions against bacteria include:

- **Phagocytosis**—engulfing and lysing microbes, antigen-antibody complexes
- **Antigen processing and presentation**—macrophages are professional antigen-presenting cells (APC) that process and present antigens of engulfed and lysed microbes to the T cells
- **Cytotoxicity**—killing host cells that contain intracellular antigens (to tackle viruses, tumor cells, intracellular parasites)

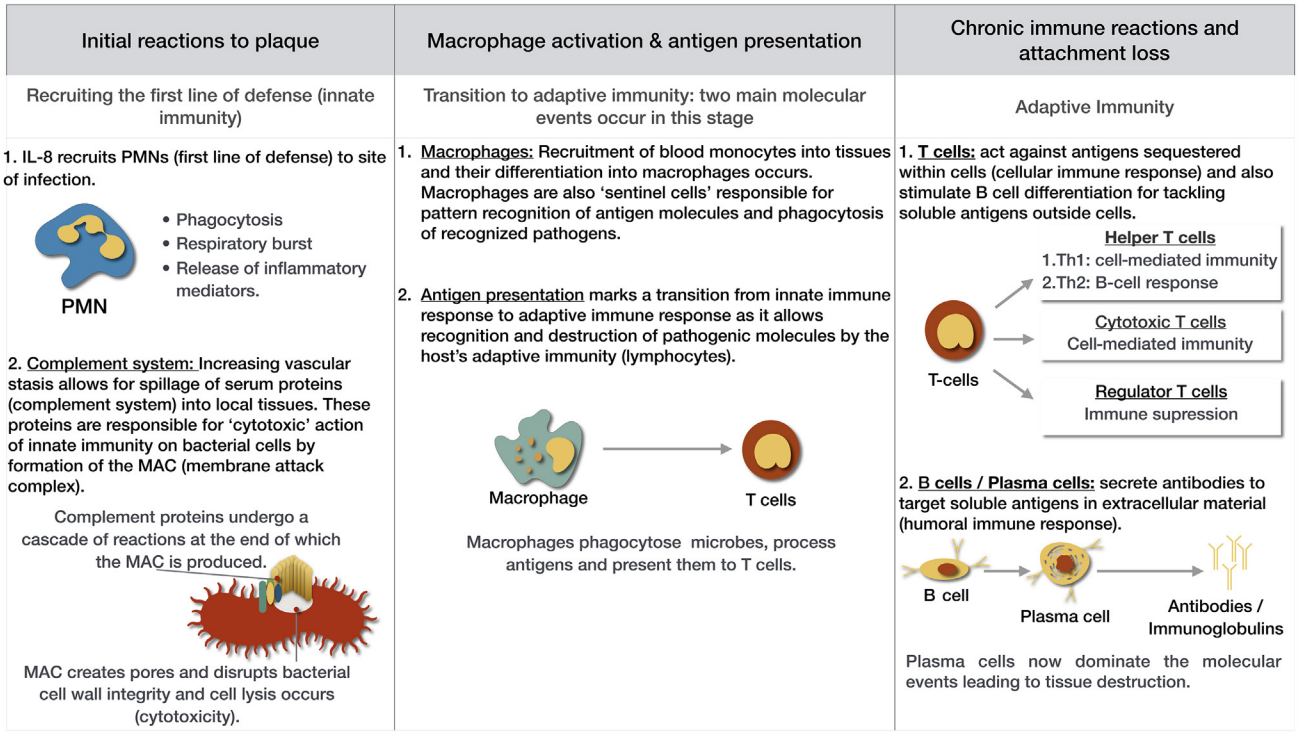


• **Fig. 4.3** Attachment Loss: Significant Molecular Events Leading to Clinical Signs of Periodontal Disease.[2] When subgingival plaque biofilm continues to be present, chronic inflammation sets in. Bacterial products (e.g., lipopolysaccharides/LPS) activate macrophages to produce cytokines like interleukins (IL-1) and tumor necrosis factor (TNFα). These stimulate resident fibroblasts and instigate changes within them, causing them to produce excessive amounts of collagen-destroying enzymes like matrix metalloproteinases (MMPs) and inflammatory mediators like prostaglandins (PGE_2). MMPs cause destruction of collagen fibers and extracellular matrix components within the gingiva and periodontal ligament (PDL) space; they also cause breakdown of the organic components of alveolar bone. PGE_2 activates osteoclastic cells to secrete enzymes like acid hydrolases that work to destroy the inorganic mineralized components of bone. The conversion of osteoclastic precursors to osteoclasts is highly dependent on the RANK/RANKL/OPG system which may be indirectly enhanced by PGE2. Altogether, dysregulated inflammatory-immune responses, induced by plaque bacteria and their products, come together to cause connective tissue breakdown and bone resorption within the periodontium. This results clinically in loss of attachment of gingival/PDL collagen fibers to the tooth surface, leading to pocket formation, tooth mobility, tooth migration, and even tooth loss. Select images are from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier. OPG, osteoprotegerin; RANK, receptor activator of nuclear factor-κB; RANKL, receptor activator of nuclear factor-κB ligand.

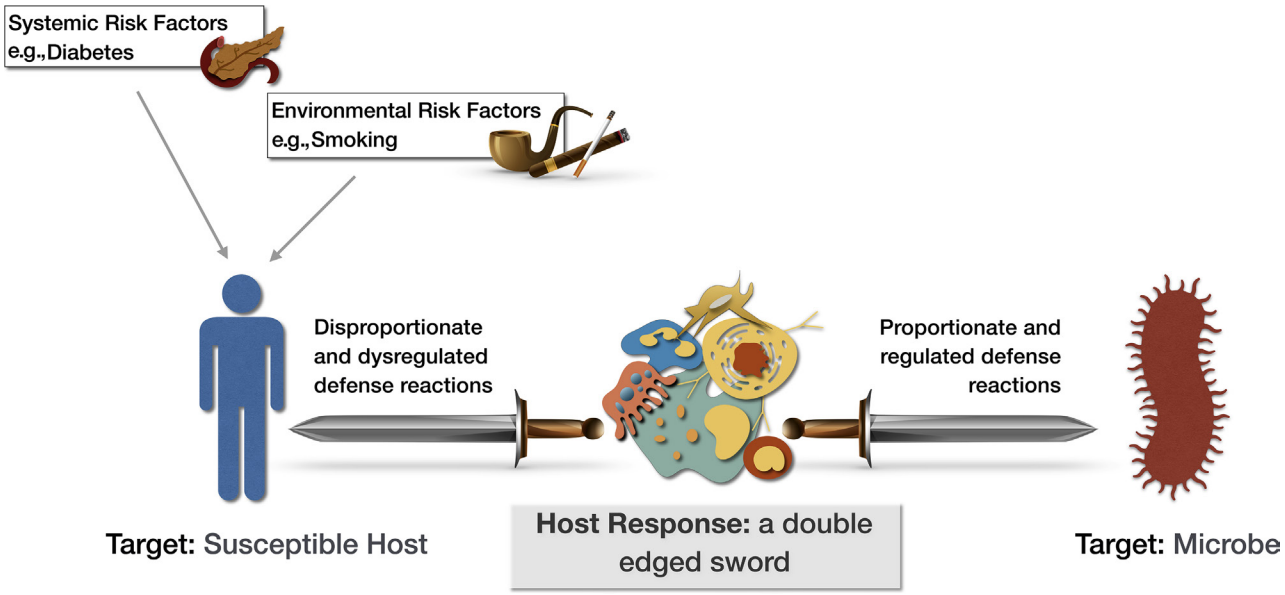
If innate immunity fails to eliminate infection, effector cells of the adaptive immunity system (lymphocytes: T cells and B cells) are recruited. Immune responses are not actually a "linear progression" of events. Innate and adaptive immunity are closely integrated and do not function in isolation to tackle the etiologic factors contributing to periodontal disease (Fig. 4.4).

Concept of Host Susceptibility

Individuals vary with respect to their susceptibility to periodontal disease, leading to an uneven disease experience among the general population. Such variations are affected by genetic, systemic, and environmental factors. Although bacteria are required to initiate disease, the host response is mostly responsible for tissue destruction in disease-susceptible individuals (Fig 4.5).



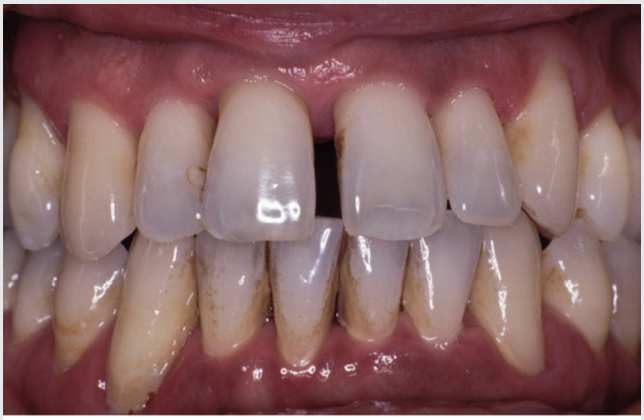
• **Fig. 4.4** Immune Responses in Periodontal Pathogenesis. Reactions to periodontal pathogens within plaque mass begin when junctional epithelial cells secrete IL-8 in response to noxious products released by microbes within the gingival sulcus. Neutrophils (PMN) are recruited as the first line of defense and move toward the sulcus from within subepithelial connective tissue, following a chemotactic gradient of IL-8. The inflammatory mediators secreted by PMNs, and subsequent vascular reactions, stimulate serum protein systems (acute phase proteins, complement system) that also act against pathogens. Subsequently, macrophages are recruited; if innate immunity is unable to contain the threat, they process and present the antigens to the more sophisticated and fine-tuned effector cells of adaptive immunity: T and B cells. T cells are involved in mainly cell-mediated immunity, targeting antigens safely sequestered within host cells and intracellular pathogens trying to avoid host immune surveillance. Extracellular, solubilized antigens are tackled by antibodies secreted by plasma cells which are differentiated from B cells. Note: This figure is simplified for fundamental understanding and does not represent feedback mechanisms and cross-relationships, which are also a vital part of immune responses.

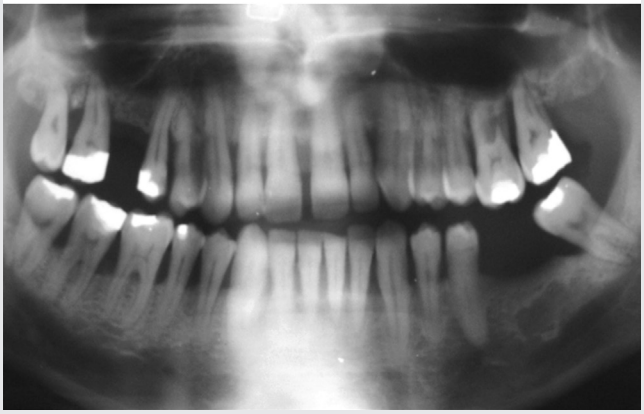


• **Fig. 4.5** Host Susceptibility in Periodontal Disease.[3] Periodontal disease manifestation and progression are determined by the nature of the immune response to the bacterial complexes in plaque. Depending on the nature of the immune response, the disease will either remain stable and not progress, or may progress and result in chronic inflammatory disease. A host who is susceptible to the disease experiences clinical attachment loss due to periodontal tissue destruction, whereas an individual who is not susceptible does not experience tissue destruction even in the presence of persistent plaque biofilm. The susceptibility of an individual to periodontal disease is modulated by various factors, including systemic risk factors (e.g., diabetes) and environmental risk factors (e.g., smoking).

CASE-BASED LEARNING EXERCISE

Scenario: A 56-year-old male presented with a chief complaint of bad breath and bleeding gums when brushing. There was no relevant medical history, but he was a former smoker (quit 6 months ago, 20 cigarettes/day for 15 years). He had a history of irregular dental care (no previous history of periodontal treatment) and reported using a manual toothbrush, twice per day for at most 1 minute each time. The patient presented with generalized probing depths of 5 to 8 mm with bleeding on probing (BOP) of 70%. Grade 1 mobility was noted in tooth #9 and diastema remained the same between central incisors over the last few months. Panoramic radiograph revealed advanced bone loss (>80%) at the maxillary molar areas.





Clinical images are from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

Questions

1. What is the periodontal diagnosis?
   - **a.** Gingivitis
   - **b.** Periodontitis
   - **c.** Healthy periodontium
   - **d.** Perio-endo lesion
2. What is the most likely etiology for periodontitis?
   - **a.** Bacterial plaque
   - **b.** Defective restorations
   - **c.** Open contacts
   - **d.** Root morphology
3. What is the most likely explanation for this high BOP?
   - **a.** Past smoking history
   - **b.** Current nonsmoking status
   - **c.** Plaque-induced inflammation
   - **d.** Occlusal factors
4. Why is more advanced bone loss noted in maxillary molar areas?
   - **a.** Host susceptibility
   - **b.** Smoking history
   - **c.** Open contacts
   - **d.** Difficult access to clean

This chapter was developed from Chapter 7 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: b

Explanation: Based on clinical and radiographic presentation and the presence of local factors such as generalized biofilm deposits, this is a case of periodontitis.

2. Answer: a

Explanation: Poor oral hygiene and the long-term challenge of the bacterial biofilm result in chronic inflammation in the periodontal tissues. The immune-inflammatory response is protective by intent, but it causes the tissue damage that we recognize clinically as periodontitis. Defective restorations, open contacts, and root morphology are considered contributing factors to periodontal disease.

3. Answer: c

Explanation: Poor oral hygiene and presence of the subgingival biofilm result in inflammation of the gingival and periodontal tissues. The periodontal probe penetrates the inflamed junctional epithelium easily **and enters** the connective tissue**,** causing bleeding on probing. Note: smoking can have an inhibitory effect on the microvasculature such that smokers, on quitting, often notice an increase in gingival bleeding as the vasculature recovers.

4. Answer: d

Explanation: Maxillary molars often have more advanced bone loss, probably because of difficulties accessing these regions for cleaning (especially interproximal cleaning). Further difficulties in cleaning arise once furcation involvement occurs, leading to further progression of disease at these sites.

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5 Periodontal Microbiology

Relevant Terminology

Terminology/Abbreviation	Explanation
acquired pellicle	Organic material consisting of peptides, proteins, and glycoproteins that forms on the tooth surface and serves as the adhesion site for bacteria.
Aggregatibacter actinomycetemcomitans	• Gram-negative, facultative anaerobic nonmotile rods • Belongs to green complex • Highly associated with the pathogenesis of molar-incisor pattern of periodontitis (previously *localized aggressive periodontitis*) • A highly leukotoxic clone is known as JP2 (serotype b)
allogeneic succession	Change in composition of bacterial community due to external, nonmicrobial factors (e.g., smoking).
autogenic succession	Change in composition of bacterial community due to microbial factors (e.g., interbacterial interactions).
calculus	Hard deposit that forms via the mineralization of dental plaque, generally covered by a layer of unmineralized dental plaque (considered a plaque-retentive factor).
Candida albicans	• Mucosal and blood stream infections • Associated with candidiasis
coaggregation	The phenomenon of genetically distinct bacteria attaching to one another by specific molecules and influencing the growth of a multispecies biofilm.
corn cob and test-tube brush formation	A structure established through coaggregation of coccal cells (corn cob) or gram-negative rods (test-tube brush) attached to gram-negative filamentous core. Examples of coaggregation in plaque biofilm: • **Corn cob formation**—seen in supragingival plaque; central gram-negative core supporting outer coccal cells • **Test-tube brush formation**—seen in subgingival plaque; gram-negative rods attach to filamentous microbes
coxsackievirus A	• Picornavirus; nonenveloped, single-stranded RNA virus • Associated with herpangina and hand, foot, and mouth disease
dental plaque	• Highly organized biofilm primarily composed of bacteria in a matrix of salivary glycoproteins and extracellular polysaccharides • Cannot be removed by rinsing or water spray (spontaneous cleaning such as chewing fibrous food or tongue movement is insufficient)
dysbiosis	• Gradual shift in the microbial community • Decreased number of beneficial species and increased number of pathogenic species
echovirus	• Picornavirus, nonenveloped single-stranded RNA virus • Associated with herpangina and Guillain–Barré syndrome
ecological plaque hypothesis	• Proposed in 1990s • Both the total amount of dental plaque and its specific microbial composition may contribute to the transition from health to disease • Dynamic relationship whereby the inflammatory response results in an environmental change that produces a shift in the balance of the resident microbiota, predisposing a site to disease
enterovirus	• Picornavirus, nonenveloped single-stranded RNA virus • Associated with herpangina and hand, foot, and mouth disease (enterovirus 71)

Relevant Terminology—cont'd

Terminology/Abbreviation	Explanation
Epstein–Barr virus (EBV)	Enveloped double-stranded DNA virus associated with Burkitt lymphoma, infectious mononucleosis, Hodgkin lymphoma, and B lymphoproliferative diseases.
fimbriae and pili	Polymeric fibrils associated with bacterial cell adhesion.
gingipain	Virulence factor produced by *Porphyromonas gingivalis*. Belongs to the cysteine protease family, plays important roles in adhesion, tissue degradation, and the evasion of host responses.
herpes simplex virus 1 (HSV-1)	• Enveloped double-stranded DNA virus associated with herpetic gingivostomatitis (primary HSV-1 infection), recurrent orolabial lesions • Latency in trigeminal ganglia
herpes simplex virus 2 (HSV-2)	• Enveloped double-stranded DNA virus associated with genital infection • Latency in sensory ganglia
human herpesvirus 8	• Enveloped double-stranded DNA virus • Associated with Kaposi sarcoma
human immunodeficiency virus 1 (HIV-1)	• Retrovirus, enveloped single-stranded RNA virus • Global infection • Infects cells that contain CD4 receptors (T helper cells and macrophage lineage)
human immunodeficiency virus 2 (HIV-2)	• Retrovirus, enveloped single-stranded RNA virus • Infection mainly in West Central Africa • Less virulent than HIV-1
keystone pathogen hypothesis	• Certain pathogens may trigger the disruption of microbial homeostasis, thereby leading to the development of periodontal disease, even when they are present only in low numbers • *P. gingivalis* is a good example of a keystone pathogen
leukotoxin	Virulence factor produced by *A. actinomycetemcomitans* that has leukotoxic (pore-forming) effects on immune cells.
lipopolysaccharide	• Endotoxin that consists of a lipid and a polysaccharide composed of O antigen on the outer membrane of gram-negative bacteria • Recognized by toll-like receptor 4 • Induces strong immune response
materia alba	• Soft accumulation of salivary proteins, bacteria, desquamated epithelial cells, and food debris without organized structure • Can be easily displaced with a water spray
niche	A function space/structure within an ecological system to which an organism is especially suited. Examples are: • Intraoral and supragingival hard surfaces • Subgingival regions adjacent to hard surface • Buccal and palatal epithelium, floor of the mouth • Dorsum of tongue • Tonsils • Saliva
nonspecific plaque hypothesis	• Proposed in mid-1900s • Periodontal diseases result from the "elaboration of noxious products by the entire plaque flora"
papillomavirus	• Nonenveloped double-stranded DNA virus • Associated with genital and cutaneous warts, cervical and anogenital cancers, condyloma acuminata, and recurrent respiratory papillomatosis
primary colonizers	Bacterial species (e.g., *Streptococcus*, *Actinomyces*, *Capnocytophaga*, *Eikenella*, *Veillonella*) that adhera to the acquired pellicle and provide new binding sites for secondary colonizers.

quorum sensing	• Method of communication between biofilm bacteria
	• Bacteria secrete a signaling molecule that accumulates in the local environment. Once they reach a critical threshold concentration, several responses are triggered:
	  ◦ modulating the expression of genes for antibiotic resistance
	  ◦ encouraging the growth of beneficial species in the biofilm
	  ◦ discouraging the growth of competitors

secondary colonizers	Lack the ability to initially colonize clean tooth surfaces but rather adhere to bacteria that are already in the plaque mass.

Continued

Relevant Terminology—cont'd

Terminology/Abbreviation	Explanation
Socransky's red complex	*P. gingivalis*, *Tannerella forsythia* and *Treponema denticola.*
specific plaque hypothesis	• Proposed in early 1960s • Plaque that harbors specific bacterial pathogens may provoke periodontal disease
subgingival plaque and calculus	• Found below the gingival margin • Gram-negative rods, filaments, and spirochetes predominate • Inorganic component of calculus is mainly from crevicular fluid; green or dark brown color of calculus indicates the presence of blood products from subgingival hemorrhage
supragingival plaque and calculus	• Found above the gingival margin • Gram-positive cocci and short rods predominate • Inorganic component of calculus is mainly from saliva
translocation	Intraoral transmission of bacteria from one niche to another via saliva, probing, etc.
varicella zoster virus	• Enveloped double-stranded DNA virus associated with herpes zoster (shingles), varicella (chickenpox), and Hunt syndrome • Latency in sensory ganglia
virulence factor	Specific molecule produced by a microbe that allows it to effectively bypass host immunity and colonize a niche within the host.

Fast Facts

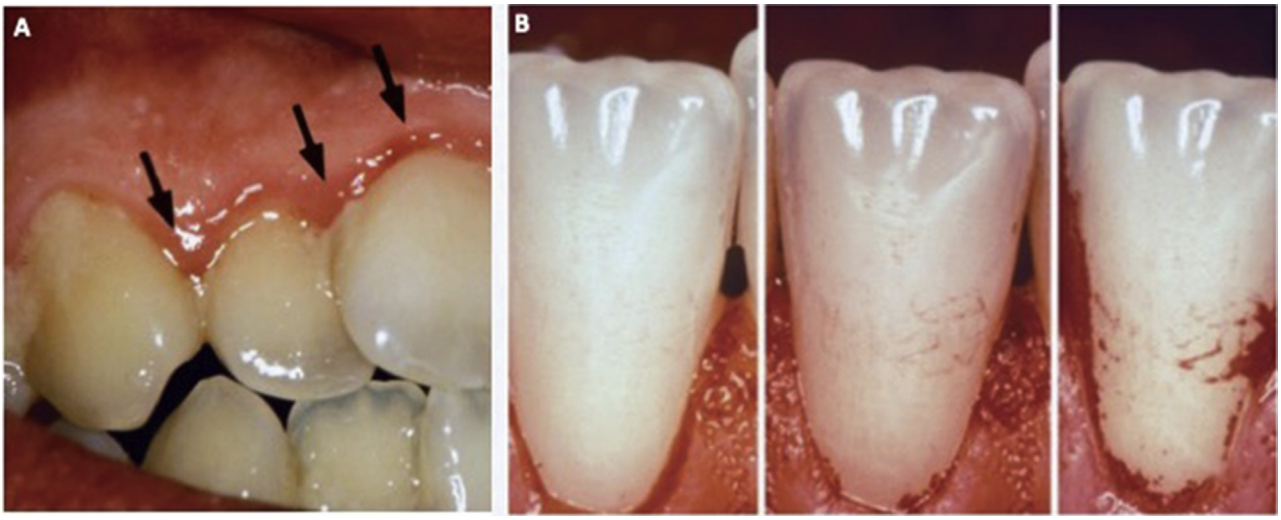
Bacterial species associated with periodontal health	*Planobacterium, Cardiobacterium, Corynebacterium, Kingella, Capnocytophaga, Eubacterium, Peptostreptococcus, Alloprevotella, Hallella, Johnsonella,* and *Mycoplasma.*
Bacterial species associated with periodontitis	*Porphyromonas, Filifactor, Treponema, Fusobacterium, Tannerella, Streptococcus, Actinomyces,* and *Veillonella.*
Removal of microorganisms from the oral cavity	Nonadherent bacteria are removed through: • Swallowing, mastication, or blowing the nose • Tongue and oral hygiene practices • The washout effect of the salivary, nasal, and crevicular fluid outflow • The active motion of cilia on the nasal and sinus walls
Bacterial adhesion on soft tissue surfaces	• Shedding (desquamation) is a natural cleansing mechanism of soft tissues • A positive correlation exists between the adhesion rate of pathogenic bacteria to different epithelia and the susceptibility of the affected patient to certain infections
Oral cavity as a unique environment for microbial populations	The oral cavity provides both soft (shedding) and hard (nonshedding) surfaces such as teeth that are accessible for microbial colonization.
Composition of the subgingival biofilm	• Depth of periodontal pocket affects composition • Spirochetes, cocci, and rods dominate the apical part of the pocket • Filaments dominate the coronal part of the pocket
Phases of accumulation of dental plaque biofilm	1. Formation of the pellicle on the tooth surface 2. Initial adhesion/attachment of bacteria 3. Colonization/plaque maturation
Three phases of initial adhesion	1. Transport to the surface 2. Initial reversible adhesion via van der Waal forces and electrostatic repulsive forces 3. Strong attachment
Microbial composition of the dental plaque	As plaque matures, the microbial composition shifts from cocci to filamentous forms and rods, and later to vibrios and spirochetes (a more anaerobic and a more gram-negative flora).
Individual variables in plaque formation	Factors that influence plaque formations are: • Local factors: smoking, restorations and prosthesis, and oral hygiene implements • Host factors: antimicrobial factors in saliva, the chemical composition of the pellicle, the clinical wettability of the tooth surfaces, the relative salivary flow • Bacterial factors: the colloid stability of bacteria in the saliva, aggregation of bacteria • Other factors: diet, chewing fibrous food

Fast Facts—cont'd

Plaque formation: variation within the dentition	Plaque formation occurs faster: • In the lower jaw compared with the upper jaw • On buccal surfaces compared with palatal surfaces • In interdental regions compared with buccal or lingual surfaces
Impact of gingival inflammation and saliva on plaque formation	• Plaque formation is more rapid around tooth surfaces facing inflamed gingival margins compared to sites facing healthy gingivae • Increase in gingival crevicular fluid production (as in inflammation) enhances plaque formation
Impact of subject's age on plaque formation	• Subject's age does not influence de novo plaque formation • Reduced dexterity in the elderly may affect the amount of plaque formation
Beneficial species of bacteria	Act by: • Passively occupying a niche that may otherwise be colonized by pathogens • Actively limiting a pathogen's ability to adhere to appropriate tissue surfaces • Adversely affecting the vitality or growth of a pathogen • Affecting the ability of a pathogen to produce virulence factors • Helping with the degradation of virulence factors

Criteria for identifying pathogens in classical infection	• **Koch's postulates**—Suspected pathogen must
	  • Be routinely isolated from diseased individuals
	  • Once isolated, be grown in pure culture in the laboratory
	  • Produce a similar disease when inoculated into susceptible laboratory animals
	  • Be recovered from lesions in the diseased laboratory animal

Criteria for identifying periodontal pathogens	Difficulties exist with regard to the application of Koch's postulates to polymicrobial diseases such as periodontitis. **Socransky's criteria** are followed for identifying periodontal pathogens: • **Association**—causative agent must be found in higher numbers in "active" sites when compared to "inactive" sites • **Elimination**—removal of causative agent must stop disease "progression" • **Host response**—host immune response to causative agent must be activated • **Virulence factors**—causative agent must possess virulent factors responsible for disease initiation and progression • **Animal models**—similar periodontal disease symptoms must be elicited in animal experiments
Microbial shift from health to disease	Characterized by shift from: • Gram-positive to gram-negative • Cocci to rods (and, at a later stage, to spirochetes) • Nonmotile to motile organisms • Facultative anaerobes to obligate anaerobes • Carbohydrate fermenting (saccharolytic) to proteolytic species
Gingipains	• Virulence factors elaborated by *P. gingivalis* • Multifunctional proteins that play important roles in adhesion, tissue degradation, and the evasion of host responses • Responsible for at least 85% of the total host protein degradation activity • Three enzymes, classified as "Arg-gingipains" (RgpA and RgpB) or "Lys-gingipains" (Kgp) on the basis of their ability to cleave arginine– or lysine–peptide bonds
Leucotoxins	Virulence factors associated with *A. actinomycetemcomitans* having the capability to directly inhibit important components of the human immune system (e.g., neutrophils, immunoglobulins, complement).
Virulence factors helping in adhesion to host surfaces or to other microbes (coaggregation)	• Fimbriae (major and minor fimbriae of *P. gingivalis*) • Pili (*A. actinomycetemcomitans*) • Adhesins (major sheath protein of *T. denticola*)
Virulence factors of periodontal pathogens that help destroy/evade complement system	• Gingipains (*P. gingivalis*) • Api A (*A. actinomycetemcomitans*) • Interpain A (*Prevotella intermedia*)



• **Fig. 5.1** Supragingival Plaque Accumulation. (A) Black arrows point to inflammation of the marginal gingiva adjacent to the accumulated plaque. (B) Clinical photos of a mandibular incisor showing the typical growth pattern of supragingival plaque using disclosing solution. Note the coronal spread of plaque from the gingival margin toward the incisal edge. From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

Core Knowledge

Introduction

Many bacterial species colonize the oral cavity from birth. The interactions of bacteria range from sharing weapons of defense (e.g., antibiotic resistance) to competing for available resources (e.g., commensals that are beneficial to the host by not allowing the growth of pathogenic species). The oral cavity can also be colonized by yeasts, protozoa, and viruses. This chapter describes the various microorganisms and plaque biofilm that are responsible for health and disease in the periodontium.

Bacteria and Their Biofilm Mode of Living

One type of defense against host immunity employed by microbes within the oral cavity is the formation of dental plaque as a biofilm. This biofilm structure is more complex than the individual bacteria that comprise it, and adheres strongly to tooth surfaces in the presence of water. Some of the characteristics of plaque are:

- Plaque biofilm comprises microbial cells encased within a matrix of extracellular polymeric substances (polysaccharides, proteins, and nucleic acids).
- Biofilm bacteria are 1000 times less sensitive to antimicrobial agents than free-floating (planktonic) bacteria.
- Microbes form communities within the biofilm that exhibit highly complex ecology. A primitive circulatory system consisting of water channels running between colonies facilitates waste product removal from, and nutrient supply to, the deeper layers of the biofilm by diffusion. Also, steep chemical gradients (e.g., oxygen or pH) exist; these produce distinct microenvironments within the biofilm.
- The inorganic component of plaque mainly comprises calcium and phosphorus derived predominantly from saliva (for supragingival plaque) or gingival crevicular fluid (GCF) (for subgingival plaque). When plaque biofilm mineralizes, it forms dental calculus.

A clinical photo depicting 10-day-old supragingival dental plaque with associated inflammation of the marginal gingiva is presented in Fig. 5.1A. Dental plaque accumulation starts near the gingival margin and interdental spaces; over time, it extends generally in the coronal direction (Fig. 5.1B), with the exception of its individualized growth pattern following surface irregularities.

Composition of Dental Plaque Matrix

The dental plaque matrix includes both organic and inorganic components.

Organic components:

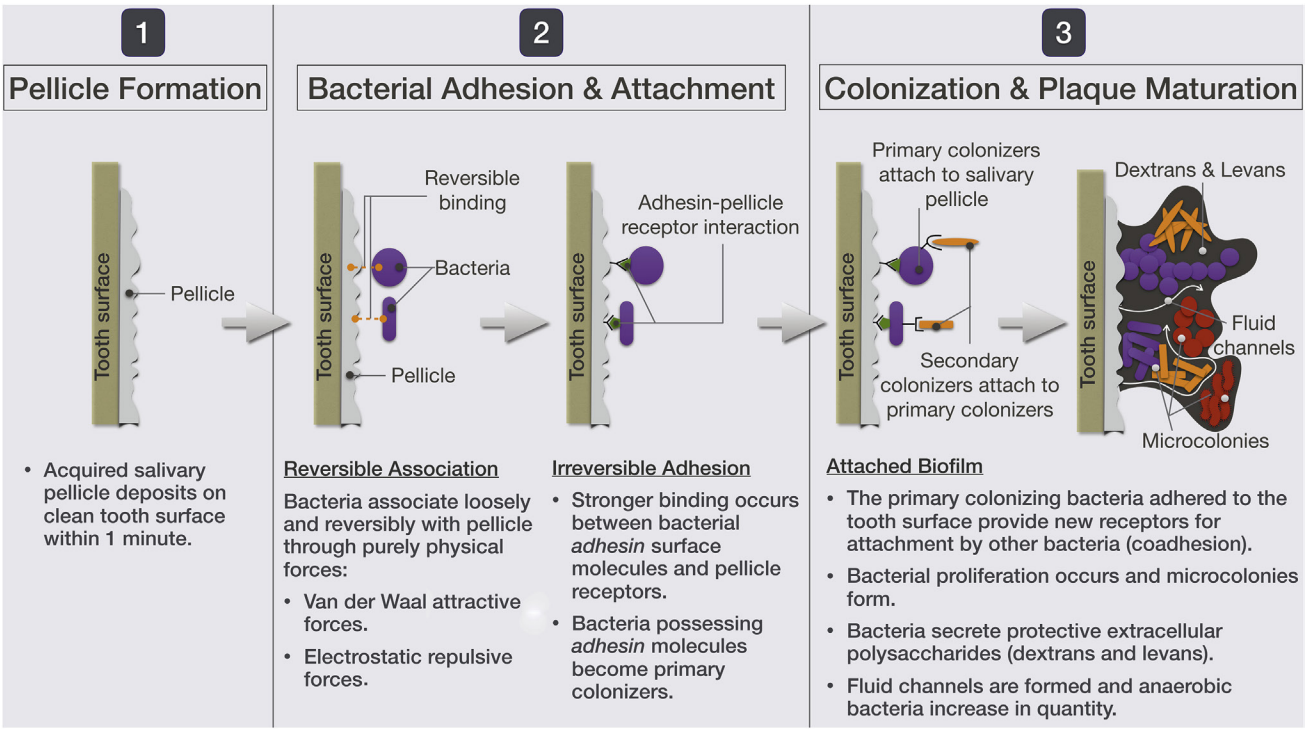
- Proteins (e.g., albumin from GCF)
- Glycoproteins—important part of pellicle that initially coats a clean tooth surface (from saliva)
- Polysaccharides—important part of plaque matrix (from bacteria)
- Lipids—from dead bacterial and host cells, food debris
- Nucleic acids

Inorganic components:

- Minerals—calcium and phosphorus
- Trace—sodium, potassium, fluoride

Plaque bacteria relevant to dental diseases can be classified as:

- "Planktonic" bacteria—free-floating
- "Biofilm" bacteria—embedded in an extracellular matrix



• **Fig. 5.2** Dental Plaque: Biofilm Formation. The figure represents the various steps in the formation of plaque biofilm. **Pellicle formation—**all hard and soft tissues within the oral cavity are coated with an organic layer from the saliva, the pellicle, which is made up of a thinner basal layer (hard to remove) and a thicker globular layer (easily removed). This pellicle, formed by adsorption of salivary glycoproteins onto the tooth surface, contains more than 180 proteins, including the pellicle receptors that serve as sites for bacterial attachment. **Bacterial adhesion and attachment—**the initial transport of a bacterium to the tooth surface occurs by sedimentation of microbes, liquid flow, brownian motion, etc. Bacteria initially associate loosely with the pellicle purely by physical forces of attraction and repulsion. The sum of the attractive and repulsive forces causing bacteria-pellicle interaction is represented as the *total Gibbs energy*. Later, the bonds become stronger and irreversible due to the interaction of *adhesins* on bacterial surfaces with the salivary pellicle receptors (e.g., salivary glycoprotein gp340). **Bacterial colonization and plaque maturation—**bacteria that attach directly to the pellicle via adhesins (primary colonizers) now expose receptors for binding other bacteria (secondary colonizers), resulting in *coadhesion*. Bacterial proliferation results in the formation of microcolonies that are embedded in a matrix of extracellular polysaccharides secreted by bacteria (e.g., streptococci). A primitive circulatory system comprising fluid channels between colonies is formed to allow for nutrient exchange and waste removal. The plaque biofilm now matures, presenting with more anaerobic bacteria in the deeper layers. The transition from early supragingival dental plaque to mature plaque growing below the gingival margin involves a shift in the microbial population from primarily gram-positive organisms to high numbers of gram-negative bacteria.

Plaque formation and maturation involve several steps, the most important of which are pellicle formation, bacterial attachment and adhesion to tooth surface, bacterial colonization, and plaque growth and maturation (Fig. 5.2).

Microbiologic Specificity of Periodontal Diseases

Current thinking supports the fact that both host and microbes have important roles to play in the final microbial composition of dental plaque. Several theories have been proposed over the years to determine whether plaque quantity, quality, microenvironment, or specific pathogens within plaque matters the most in periodontal disease pathogenesis. Understanding the various plaque hypotheses, their influence on treatment rationale, and drawbacks is important for proper management of patients (Table 5.1).

Virulence Factors of Periodontopathogens

Rationale behind studying virulence factors:

- It is often difficult to determine precisely how individual organisms contribute to disease.
- Targeting one or more "pathogens" will not necessarily cure disease, because other organisms with similar activities may take their place.
- Therefore it might make sense to focus on the specific molecules that contribute to disease (i.e., virulence factors) rather than on the microorganisms that produce them.

CLINICAL CORRELATE

Which Of The Proposed Plaque Hypotheses Models Best Fits Various Oral Diseases? Which Model Is The Basis For Treatment Decisions Regarding Periodontal Disease?

All available hypotheses fall short of combining actual microbial and host behaviors that lead to maintenance of health or the shift to disease. But if we must choose a best fit, certain points need to be considered:

- For the caries process, the best-fitting model is the ecologic plaque hypothesis. It considers the role of fermentable carbohydrates and other microenvironmental changes in the plaque ecology as being responsible for the demineralization process.
- The recently described polymicrobial synergy and dysbiosis (PSD) model for periodontitis highlights the importance of the idea that bacteria other than the classical "red complex" species could have similar keystone roles in periodontitis. The PSD model is currently the most extensive; however, it is modeled only for periodontitis.

Treatment rationale: While further research takes place to find the best model that fits all oral plaque–induced diseases, one must keep in mind that periodontal disease is not an infection in the classical sense, where one can pinpoint a specific organism as being responsible for the disease. Consequently, all plaque must be controlled. Thus the starting point of periodontal therapy is based on the nonspecific plaque hypothesis.

Following are some of the mechanisms by which virulence factors in periodontal pathogens play a role in the periodontal disease process:[2]

- Establishment of periodontal pathogens close to host tissues (adhesins, fimbriae, or pili)
- Host defense evasion (capsule, leukotoxins)
- Host defense nullification (leukotoxins, collagenases, immunoglobulin proteases)
- Pathogen colonization and proliferation (proteases for food sources)
- Destruction of periodontal tissues—microbe-derived factors (collagenases, lipopolysaccharide, lipoteichoic acid, invasins, butyric acid) or host-derived factors (matrix metalloproteases, cytokines, prostaglandins)

Conclusion

It is now widely accepted that periodontal diseases are not infections caused by specific bacteria, in the classical sense, because the host response to the bacteria plays a vital role in disease development. This paradigm shift does not mean that studying the putative periodontal pathogens, their virulence factors, or roles in triggering a dysregulated immune reaction within the host becomes any less important. In fact, the importance of periodontal microbiology is underscored even more because:

- Polymicrobial synergy can facilitate destructive host responses, and it is important to understand this phenomenon better.
- The recognition of the beneficial activity of several groups of commensal species may open new strategies for the treatment of periodontal disease, such as the use of probiotics or microbial replacement therapy.

Questions

1. The yellow-grayish matter in the clinical image above is: _____
- **a.** Dental plaque
- **b.** Materia alba
- **c.** Necrotic tissue
- **d.** Food remnants

2. What would a plaque sample look like under a phase contrast microscope for a case of necrotizing periodontal disease (NPD)?
- **a.** All coccoid bacteria
- **b.** All filamentous bacteria
- **c.** Combination of motile rods and coccoid bacteria
- **d.** Combination of filamentous and motile spiral bacteria

3. Which specific bacteria could you primarily expect in a plaque sample of affected lesions of NPD?
- **a.** *P. gingivalis* and *P. intermedia*
- **b.** *P. gingivalis*, *T. denticola*, and *T. forsythia*
- **c.** *F. nucleatum* and *T. denticola*
- **d.** *F. nucleatum* and *P. gingivalis*

4. Which specific microorganism would one expect in a plaque sample of affected lesions of denture stomatitis?
- **a.** *F. nucleatum* and *T. denticola*
- **b.** *C. albicans*
- **c.** *T. tenax*
- **d.** *F. nucleatum* and *P. gingivalis*

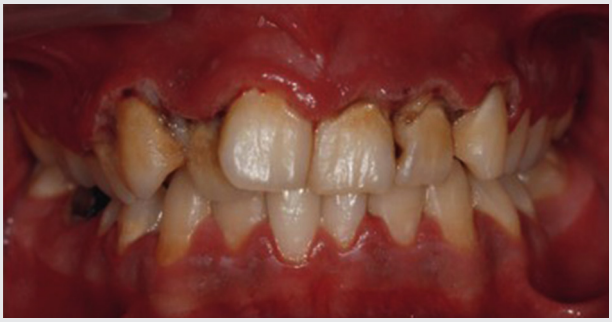
This chapter was developed from Chapter 8 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

TABLE 5.1 Plaque Hypotheses[1]

	Nonspecific Plaque Hypothesis	Specific Plaque Hypothesis	Ecologic Plaque Hypothesis	Keystone Pathogen Hypothesis / Polymicrobial Synergy and Dysbiosis Model
Hypothesis	• All plaque is equally pathogenic • Quantitative increase in plaque amounts causes disease	• Not all plaque is equally pathogenic • Some plaque biofilms cause more destruction due to the presence or increase of specific microorganisms (plaque quality matters)	• Attempts to unify specific & nonspecific plaque hypotheses • Overgrowth of plaque (quantitative increase) changes the local microenvironment (e.g., acidic pH), promoting the growth of specific bacterial pathogens (qualitative shift)	• In contrast to the idea that a dominant species can influence inflammation by their abundant presence, 'keystone' pathogens (e.g., *P. gingivalis*) can trigger inflammation even when they are present in low numbers • These pathogens can cause a normally benign microbiota to become a dysbiotic and disease-provoking one
Treatment Rationale	• Control of periodontal disease depends on reduction of the total amount of plaque	• Control of periodontal disease depends on getting rid of pathogenic plaque (e.g., targeting specific microbes using antimicrobials)	• Modify microenvironment (diet, oral hygiene, topical fluoride, etc.) to prevent nourishment of pathogens • Applies more to the prevention of dental caries than periodontal disease	• Host modulation works as adjunct to direct antimicrobial measures to tackle keystone pathogens that can hide inside host cells and also utilize products of host inflammation for their own nourishment
Drawbacks	Hypothesis could not explain the following facts: • Advanced tissue destruction can be found adjacent to healthy periodontal sites (site specificity) in same individual • Periodontal destruction is not always proportional to plaque quantity	Hypothesis could not explain the following facts: • Periodontal disease can occur even in the absence of "putative periodontal pathogens," such as red complex bacteria • The same "pathogens" may be present in the absence of disease	• Does not address the role of host genetics that contribute significantly to both dental biofilm composition and host susceptibility	• Heavy dependence on a single periodontal bacterium (*P. gingivalis*) to explain the entire theory • Relatively new disease model that requires further validation

CASE-BASED LEARNING EXERCISE

Scenario: A 32-year-old female patient presented with the chief complaint of "significant pain in my gums for the last 3 days." The pain affected the patient's sleep and her ability to brush. Background information: The patient was systemically healthy and her last professional dental cleaning was 4 years ago. She was smoking 20 cigarettes a day at the time of examination. She was recently fired and currently experiences financial problems. Current findings: yellow-grayish matter on top of the gingival margin in the maxillary anterior sextant (Figure) with punched-out appearance, spontaneous bleeding, and tenderness to probing.



Clinical images are from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

Case-Based Learning Exercise

Solutions

1. Answer: c

Explanation: Clinical cases of necrotizing periodontal diseases (gingivitis, periodontitis, and stomatitis) are defined by necrosis and ulceration of the coronal portion of the interdental papillae and gingival margin, with a painful, bright red marginal gingiva that bleeds easily, similar to the clinical presentation above.

2. Answer: d

Explanation: Tissues affected by NPD typically present with filamentous and motile spiral bacteria. This relates to the specific microbiology of these diseases.

3. Answer: c

Explanation: Although all the bacteria listed will be present, the sample will be dominated by *F. nucleatum* (filamentous) and treponemes (motile spiral). This combination is a microbiologic hallmark of NPD.

4. Answer: b

Explanation: Although bacteria and epithelial cells will be present in the sample, the sample will show elongated ellipsoidal cells and hyphae forms of candidal species.

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6 Host-Microbiome Interactions

Relevant Terminology

Terminology / Abbreviation	Explanation
adaptive immune cells	• T lymphocytes and B lymphocytes
antimicrobial peptides	• Provide defense against bacteria, viruses and fungi • Involved in innate immune response • Example: defensins and cathelicidins
complement system	• A system of plasma proteins that can be activated directly by pathogens or indirectly by pathogen-bound antibodies, leading to a cascade of reactions that occurs on the surface of pathogens • Three main pathways exist: the classical pathway, the mannose-binding lectin pathway, and the alternative pathway • All pathways converge to C3 activation, leading to generation of anaphylatoxins (C3a and C5a), opsonin (C3b), and membrane attack complex (C5b-9)
extracellular matrix cells	Fibroblasts, cementoblasts, osteoblasts.
immunomodulatory therapies	• Modify the host response by reducing host-damaging aspects of inflammatory response that lead to tissue destruction • Low-dose doxycycline (20 mg twice daily) as a matrix metalloproteinase (MMP) inhibitor (currently used as an adjunct to periodontal therapy) is an example of host immunomodulatory therapy
key innate immune cells	• Neutrophils, monocytes, macrophages, dendritic cells, natural killer cells
microbe-associated molecular patterns (MAMPs)	• Molecular patterns found only in microbes, whose recognition by the host immune system plays a key role in innate immunity • Examples: bacterial cell wall macromolecules, nucleic acids, flagellin
NOD-like receptors	• Cytoplasmic sensors (such as NOD1 and NOD2) which play a critical role in sensing invading microorganisms and prompting the immune response • Expressed in human oral epithelium, gingival fibroblast cells, and periodontal ligament fibroblast cells
pattern-recognition receptor (PRR)	• MAMP recognition by the corresponding PRR induces host immune responses • Toll-like receptors, nucleotide-binding oligomerization domain–like receptors (NLR), C-type lectin receptors and RIG-1-like receptors (RLR) are the main PRRs • Expressed by innate immune cells, epithelial cells, extracellular matrix cells, and adaptive immune cells • Downstream signaling leads to an increase in the expression of proinflammatory cytokines, driving periodontal tissue destruction
tolerance	• The prevention of an immune response against a particular antigen • The immune system is generally tolerant of self-antigens (the system does not attack the body's own cells) and oral commensal bacteria (microbes beneficial to the host)

Continued

Relevant Terminology—cont'd

Terminology / Abbreviation	Explanation
toll-like receptor (TLR)	• A family (currently 10 in number) of predominantly trans-membrane signaling receptors, involved in triggering pro-inflammatory cascade of reactions linking innate immunity to inflammation
vigilance	• The activation of an immune response against a particular antigen • Example: PRR signaling against periopathogenic bacteria

Fast Facts

Periodontal innate immune response	The first line of defense against the bacterial challenge through MAMP recognition by PRRs of innate immune cells (neutrophils, monocytes, macrophages, dendritic cells, natural killer cells), together with epithelial cells, ECM cells.
Pattern-recognition receptor (PRR) signaling	After ligand binding to PRRs, all TLRs result in the activation of target genes in the nucleus which ultimately lead to increased cytokine production
Significance of complement system in periodontitis	• The dysregulation of complement activities may lead to a failure to protect the host against pathogens and amplify inflammatory tissue damage • In patients with periodontitis, increased levels of complement components are seen in gingival crevicular fluid and in gingival biopsy samples when compared with non-periodontitis patients
Role of antimicrobial peptides in periodontitis	• Antimicrobial peptides such as α-defensins and cathelicidin LL-37 are expressed by neutrophils and elevated in gingival crevicular fluid of periodontitis patients • β-defensins are expressed by epithelial cells in both clinically healthy and diseased tissues, with increased levels in periodontitis patients
Immunomodulatory therapies	• Immunomodulation in periodontal therapies targets the host response to lipopolysaccharide-mediated inflammation and tissue destruction • Matrix metalloproteinase (MMP) inhibitors (low-dose doxycycline) have been used as an adjunct to nonsurgical or surgical periodontal therapy • Low-dose doxycycline (20 mg twice a day for 3–9 months) is the only drug approved by the US FDA for use as host modulatory therapy for periodontitis
Cellular components of host immunity	• Inflammatory cells: neutrophils (PMN), eosinophils, basophils, mast cells, thrombocytes • Host cells: resident fibroblasts, endothelial cells, epithelial cells • Antigen-presenting cells: monocytes/macrophages, Langerhans cells/dendritic cells • Immune cells: T and B lymphocytes, natural killer cells
Humoral (soluble) components of host immunity	• Complement proteins: C1-9 cascade and membrane attack complex • Enzymes: MMPs • Cytokines: interleukins (IL), interferon (IFN), growth factors, tumor necrosis factor (TNF), etc • Eicosanoids: prostaglandins (e.g., PGE_2), leukotrienes (e.g., LTB_4) • Receptors and antigens: PRRs and MAMPs/DAMPs

Core Knowledge

Introduction

Periodontitis is induced in susceptible hosts by a polymicrobial community, in which different microbes act synergistically to cause destructive inflammation. The primary mediator of periodontal tissue destruction is the host immune-inflammatory response triggered by periodontal pathogens. Hence understanding the immunopathophysiology of periodontal disease involves the study of the destructive interactions between:

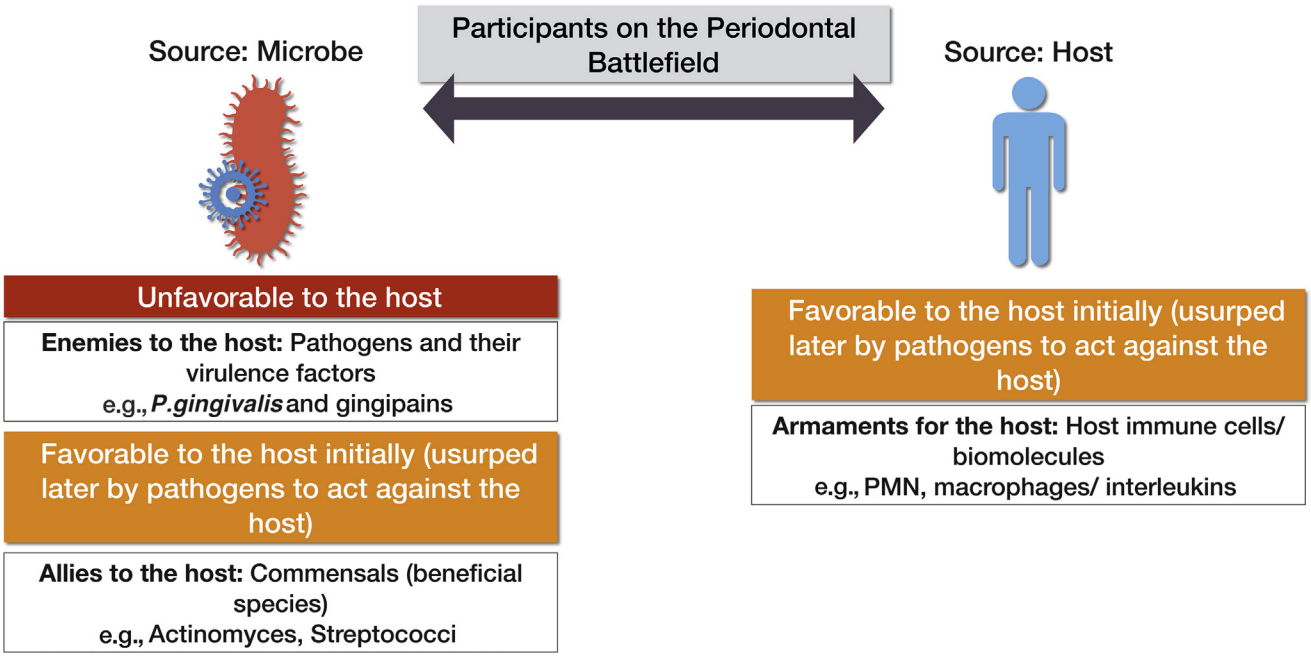
- Host: immune system and tissues
- Microbes: putative periodontal pathogens and their products in plaque biofilm

Sources of Main Participants in Host-Microbe Interactions

Host-microbe interactions involve the release of major "players" into the periodontal battlefield by both parties. Fig. 6.1 presents the sources of the main participants in a simplified manner.

Tracing the Pathway of Host-Microbe Interactions: From Plaque Biofilm to Periodontal Tissue Destruction

Bacteria are required to initiate periodontal disease, but bacteria alone are not sufficient to cause tissue destruction.



• **Fig 6.1** Host-Microbe Interactions: Sources of Main Participants.[1]

In an individual "susceptible" to periodontal disease, the host response initiated against bacteria sometimes becomes disproportionate to the challenge and attacks the host tissues. This dysregulated host response fails to resolve after dealing with the microbial challenge, and the result is collateral damage to host periodontal tissues. Thus the response initiated against microbes within periodontal tissues can turn on the host itself, resulting in attachment loss (Fig. 6.2).

Host-Microbe Interactions of Relevance to Periodontal Disease

Cells of the host immune response should ideally act only against pathogenic bacteria and not against beneficial bacteria or host tissues. This important discriminative aspect of innate immunity is tightly regulated by host-microbe interactions that involve recognition of specific molecules common to pathogens (microbe-associated molecular patterns or MAMP) that are not present in host cells, using special receptors present in host immune cells (pattern-recognition receptors or PRR). Examples include:

- MAMP (ligands)—microbial cell wall macromolecules (endotoxin/lipopolysaccharide, lipoteichoic acid), nucleic acids, flagellin
- PRR (receptors)—Toll-like receptors (TLR), nucleotide-binding oligomerization domain–like Receptors (NLR)

See Fig. 6.3 to understand the role of MAMP-PRR interactions in conditions of periodontal health.

PRR location: In addition to innate immune cells (neutrophils, monocytes, macrophages, dendritic cells, natural killer cells), PRRs are also expressed by epithelial cells, extracellular matrix cells (fibroblasts, cementoblasts, osteoblasts), and adaptive immune cells (T and B lymphocytes). PRRs that predominantly recognize bacteria include TLRs and nucleotide-binding oligomerization domain–like receptors (NLRs):

- TLRs—transmembrane receptors (situated on plasma membrane or endolysosomal membrane of host cells)
- NLRs—cytosolic receptors (e.g., NOD1/NOD 2)

DAMPs: PRRs also recognize immunostimulatory by-products derived from damaged host tissues, known as damage-associated molecular patterns (DAMPs).

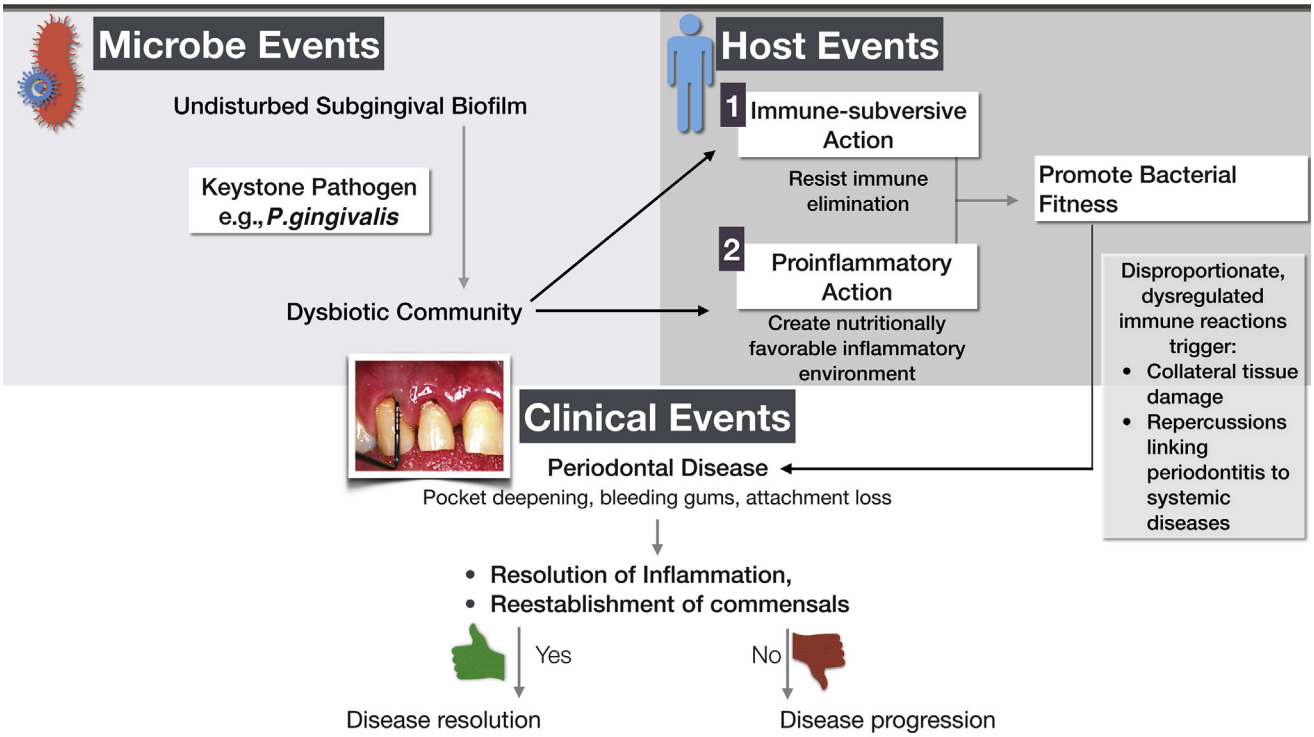
Consequences of MAMP-PRR interactions: While MAMP signaling immunomodulation plays a critical role in the homeostatic regulation of colonizing commensal microbes in health, it also contributes to pathophysiologic tissue destruction in chronic inflammatory disease states such as periodontitis.

Table 6.1 provides details of the host PRRs that interact with MAMPs from periodontal pathogens in periodontal disease.

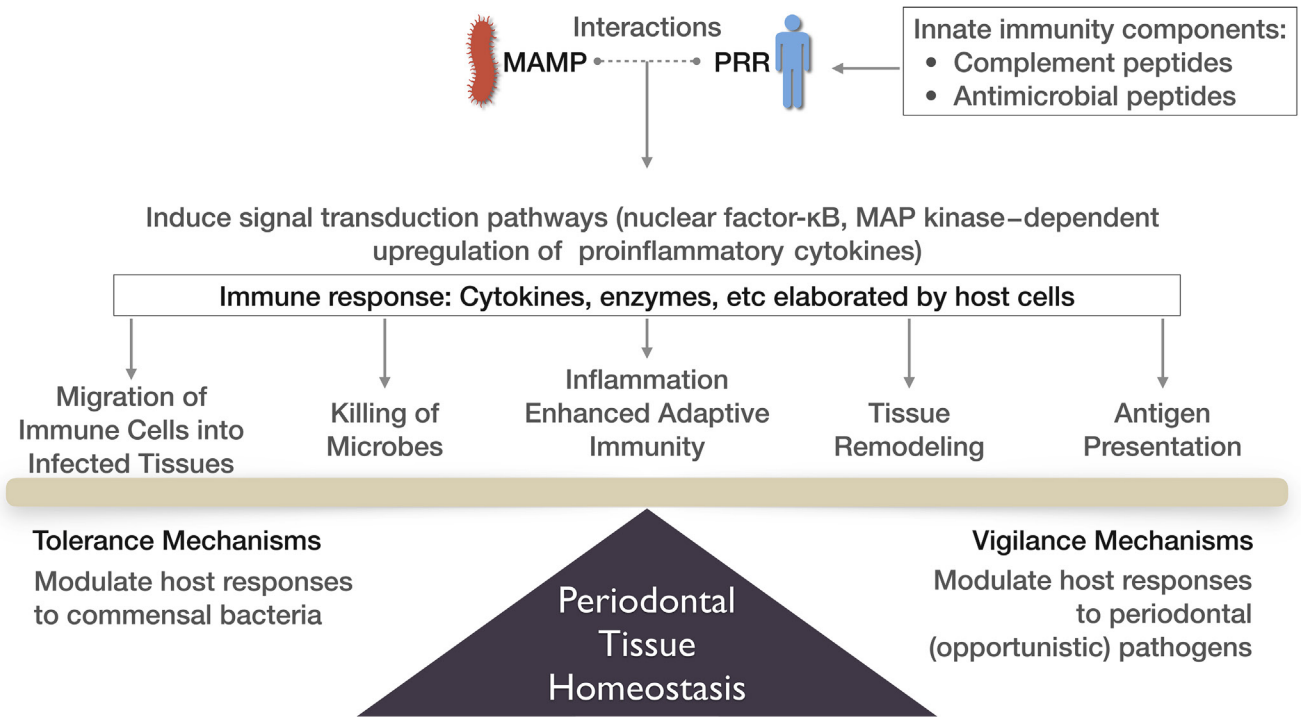
CLINICAL CORRELATE

What happens to MAMP-PRR signaling pathways during periodontal disease?

In periodontal health, pattern-recognition receptor (PRR) signaling is effectively modulated to regulate the oral commensal microbiota (tolerance) and protect against periopathogenic bacteria (vigilance), thus supporting periodontal tissue homeostasis. Failed tolerance and vigilance mechanisms in periodontal disease states lead to shifts in the oral microbiota from beneficial toward a pathogenic community. This in turn drives PRR signaling to induce proinflammatory periodontal tissue destruction.



• **Fig 6.2** Host-Microbe Interactions From Plaque Biofilm to Periodontal Disease. The periodontitis phenotype is characterized by an exaggerated, ineffective, and nonresolving inflammation of the connective tissues supporting the teeth that leads to tissue destruction. This figure reviews the major events in the interactions between host and microbe under three headings: **Microbe events—**If the biofilm is not disrupted frequently and is allowed to accumulate, the conditions within it start to favor bacterial species (e.g., *Fusobacterium nucleatum*) that are capable of sensing and influencing their environment by employing chemical cues (quorum-sensing). This in turn causes microenvironmental changes that encourage the succession and proliferation of pathogens such as *Porphyromonas gingivalis* (keystone pathogen). This pathogen has a disproportionately large effect on its environment relative to its abundance; i.e., even in low numbers *P. gingivalis* can influence a commensal (symbiotic) microbial community into a disease-provoking, dysbiotic microbiota. **Host events—**The dysbiotic biofilm bacteria evade immune-mediated killing by the host (immune subversion) by specific molecular mechanisms (for example, subverting complement function such that they interfere with microbe killing by neutrophils helps periodontal bacteria survive attacks by the host's immune response. In addition, the breakdown products of inflammatory tissue are used as nutrients by dysbiotic microbial communities, thus promoting their fitness which in turn propagates and positively reinforces both phenomena (i.e., dysbiosis and inflammation). Damage-associated molecular patterns (DAMPs) are released and a subsequent failure of inflammation-resolving mechanisms results in a chronic inflammatory lesion. Viruses appear to play a role in priming inflammatory immune cells, as well as subverting various signaling pathways within those cells to create dysregulation in the ordered nature of specific immunity. **Clinical events—**In susceptible patients, this dysbiosis can trigger an excessive (dysregulated) host response, resulting in collateral periodontal tissue damage. Clinically, this manifests as pocket deepening, bleeding gums, and attachment loss within the periodontium. In this stage of progressing periodontitis, intervention is necessary to remove sufficient biofilm to allow commensal microbial species to reestablish themselves. This also allows for reduction in inflammation, a process that failed to be activated naturally by proresolution pathways (see Chapter 7 in this book for details on resolution of inflammation).[2] (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)



• **Fig. 6.3** Role of MAMP-PRR Interactions in Maintaining Periodontal Health. The host is capable of discerning between commensal and pathogenic bacteria. Microbe-associated molecular patterns (MAMPs) of microbes are directly recognized by pattern recognition receptors (PRRs) of host immune cells leading to the appropriate immune response (tolerance or vigilance) that helps the host differentiate between commensals and opportunistic pathogens. The MAMP-PRR interactions induce signal transduction pathways that cause the immune cells to secrete cytokines, enzymes, and so on that help with many immune functions, including migration of appropriate cells into infected tissues, killing of microbes, tissue remodeling, and antigen presentation. In many ways, this interaction is considered the bridge between innate and adaptive immunity; MAMP signaling to innate immune cells also upregulates the production of costimulatory molecules that are critical for the activation of adaptive immunity. In periodontal health, PRR signaling is effectively modulated to regulate the oral commensal microbiota (tolerance) and protect against periopathogenic bacteria (vigilance), thus supporting periodontal tissue homeostasis.[3] MAP kinase, mitogen-activated protein kinase.

Immunomodulatory Therapies

Various therapeutic strategies are being explored to target host-microbe interactions in order to manage periodontal disease. These include:

- **Inhibiting the signal transduction pathways involved in inflammation.** For example, pharmacologic inhibitors of NF-κB and mitogen-activated protein kinase (MAPK) pathways have been shown to inhibit inflammatory bone loss in animal studies.[6]
- **Inhibiting complement receptors CR3 and CR5.** Because C3 is a central component of the complement system, blockade at this level may help treat complement-associated diseases, including periodontitis. For example, CR3 antagonists and C5aR inhibitors have shown some promise in preclinical studies.[8]
- **Enhancing antimicrobial peptide activity.** Novel analogues of defensins have shown even higher antibacterial activity than the endogenous β-defensins 1 and 3, without any cytotoxic effects on host cells.[10]
- **Inhibiting proteases that degrade connective tissue fibers.** For example, sub-antimicrobial dose doxycycline has been used to inhibit select matrix metalloproteinases.

CASE-BASED LEARNING EXERCISE

Scenario: A healthy 36-year-old male presented saying, "I am just here for an exam." However, his wife stated, "he wakes up with blood on his pillow and has been putting off dental visit for two years." The patient had no known medical conditions. Periodontal findings: gingiva appeared erythematous (see clinical figure) and clinical exam revealed the presence of plaque, generalized calculus, bleeding on probing (80%), generalized mobility.

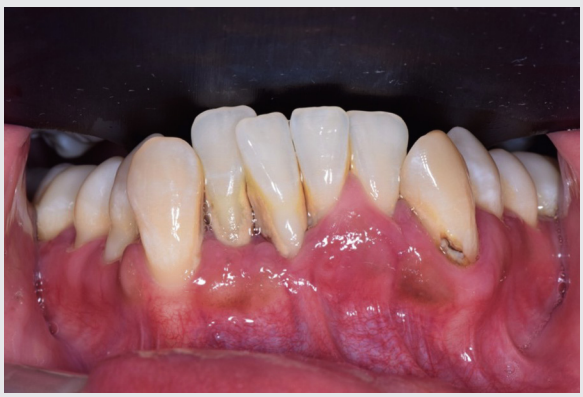


TABLE 6.1 PRR-MAMP Interactions in Periodontitis

PRR	Localization	MAMP Ligand	Ligand Origin
TLR-2/TLR-1	Plasma membrane	Triacylated lipoproteins	G- bacteria
TLR-2/TLR-6	Plasma membrane	Diacylated lipoproteins	G+ bacteria
		Lipoteichoic acid (LTA)	G+ bacteria
		Peptidoglycan	G+ bacteria
TLR-4	Plasma membrane and endolysosome	Lipopolysaccharide (LPS)	G- bacteria
TLR-9	Endolysosome	CpG-DNA	Bacterial and viral
NOD1	Cytoplasm	Gamma-d-glutamyl-mesodiaminopimelic acid (iE-DAP)	G+ bacteria G- bacteria
NOD2	Cytoplasm	Muramyl dipeptide (MDP)	G+ bacteria G- bacteria

- Knowledge of the molecular architecture of gram-positive versus gram-negative bacterial cell walls is central to a conceptual understanding of MAMP recognition by the host.
- MAMPs present in the biofilm can simultaneously activate TLRs and NOD1/2 signaling, which converge at the MAPK and NF-κB signaling pathways.
- TLRs—The human TLR family currently consists of ten known functional receptors. TLR family members are generally subdivided into two groups according to their localization at (1) the plasma membrane (TLR-1, TLR-2, TLR-4, TLR-5, TLR-6, TLR-10) or (2) the endolysosomal membrane (TLR-3, TLR-7, TLR-8, TLR-9). TLR-4 is unique in that it has the ability to localize to both types of membrane.
- Triacylated lipoproteins commonly expressed by gram-negative bacteria are recognized by TLR-2/TLR-1 heterodimer complexes, whereas diacylated lipoproteins primarily expressed by gram-positive bacteria or mycoplasmas are recognized by TLR-2/TR-6 heterodimer complexes. TLRs localized to the plasma membrane recognize extracellular microbial cell wall components (TLR-1, TLR-2, TLR-4, TLR-6) or flagellin (TLR-5), whereas TLRs localized to the endolysosomal membrane recognize microbial nucleic acids (TLR-3, TLR-7, TLR-8, TLR-9).
- Nucleotide-binding oligomerization domain–like receptors (NLRs)—NLRs are localized to the cytosol, and play a critical role in sensing invading microorganisms and prompting the immune response. NOD1 recognizes gamma-d-glutamyl-meso-diaminopimelic acid (iE-DAP), a component of peptidoglycan present in most gram-negative and some gram-positive bacteria, whereas NOD2 recognizes muramyl dipeptide (MDP), which is found in peptidoglycan from all gram-negative and gram-positive bacteria.[4,5]

CpG, cytosine-phosphodiester bond- guanine; DNA, deoxyribonucleic acid; G+, gram-positive; G-, gram-negative; MAMP, microbe-associated molecular pattern; NOD, nucleotide-binding oligomerization domain; PRR, pattern-recognition receptor; TLR, Toll-like receptor.
From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

Questions

1. If microbiological testing is performed in this patient, the possible beneficial species identified will be:
a. *Porphyromonas gingivalis.*
b. *Treponema denticola.*
c. *Actinomyces.*
d. *Fusobacterium.*

2. Which of the following is a viable therapeutic strategy to target host-microbiome interactions to treat periodontal disease?
a. Enhancing the signal transduction pathways
b. Inhibiting complement receptors CR3 and CR5
c. Inhibiting antimicrobial peptide activity
d. Enhancing proteases that degrade connective tissue fibers

3. Which of the following pattern-recognition receptors interacts with lipopolysaccharide of *P. gingivalis* to initiate an immune response?
a. TLR-2
b. TLR-4
c. TLR-9
d. NOD-2

4. Which of the following is not a proinflammatory cytokine?
a. IL-4
b. IL-1
c. TNF-α
d. IL-6

5. Which of the following is true regarding antimicrobial peptides?
a. They are components of the adaptive immune system.
b. They do not exist in the oral cavity.
c. Defensins are examples of antimicrobial peptides.
d. They are anionic peptides.

This chapter was developed from Chapter 9 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: c

Explanation: During the inflammation process for periodontitis, there are beneficial species (allies to the host) and periodontal pathogens (enemies of the host). *Actinomyces* is recognized as a beneficial species (see Fig. 6.1).

2. Answer: b

Explanation: Multiple therapeutic strategies targeting host-microbiome interactions for periodontal diseases are being explored: inhibiting the signal transduction pathways, inhibiting complement receptors CR3 and CR5, enhancing antimicrobial peptide activity, and inhibiting proteases that degrade connective tissue fibers.

3. Answer: b

Explanation: Of the PRRs listed, TLR-4 interacts with LPS, while lipoproteins and CpG-DNA of *P. gingivalis* interact with TLR-2 and TLR-9, respectively.

4. Answer: a

Explanation: Of the cytokines listed, IL-4 is considered to be an antiinflammatory cytokine; the rest are proinflammatory.

5. Answer: c

Defensins and cathelicidins are examples of antimicrobial peptides that are cationic in nature and exist in the oral cavity. They are a component of the innate immune system and act by depolarizing the bacterial cell membrane, resulting in its death.

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7

Resolution of Inflammation

Relevant Terminology

Terminology/ Abbreviation	Explanation
antiinflammation	Therapeutic strategy aimed at blocking inflammation. Example: systemic intake of nonsteroidal antiinflammatory medication (e.g., ibuprofen) to control symptoms of inflammation such as fever.
arachidonic acid	Polyunsaturated fatty acid located in the phospholipid layer of the cell membrane, released by a group of enzymes called phospholipases. The released arachidonic acid is acted upon by cyclooxygenase (COX) and lipoxygenase (LOX) enzymes to produce prostaglandins (PGs) and leukotrienes (LTs), respectively.
class switching	The switch from the synthesis of proinflammatory lipid mediators to the synthesis of proresolution lipid mediators is essential for the active resolution of inflammation. A defect in this "class switching" will lead to chronic continuation of inflammation without resolution.
cyclooxygenases (COX)	Enzymes involved in the conversion of arachidonic acid into lipid mediators, primarily PGs. There are two types of COX, COX-1 and COX-2. COX-1 is constitutively expressed and helps maintain basal levels of PGs, while COX-2 is expressed by cells challenged with an inflammatory stimulus.
experimental periodontitis	Experimental procedures performed in animals, employed in research as a model to test novel interventions for their safety and efficacy in preventing or treating periodontal disease.
leukotrienes (LTs)	Lipid mediators produced by LOX enzymes. Leukotrienes are produced by inflammatory cells such as neutrophils, mast cells, and macrophages. LTB4, one of the most potent LTs, plays an important role in neutrophil chemotaxis.
lipid mediators	Molecules derived from polyunsaturated fatty acids (PUFA) by specific enzymatic action that play important roles in inflammation and resolution of inflammation.
lipoxygenases (LOX)	Enzymes that metabolize arachidonic acid and eventually produce LTs. Three types—5-, 12-, and 15-LOX—which exist in leukocytes, platelets, and endothelial cells respectively.
omega-3 polyunsaturated fatty acid	Derived primarily from the diet (rich in marine mammals and fish) and from which SPMs such as resolvins, protectins, and maresins are biosynthesized.
proresolution	Therapeutic strategy aimed at enhancing resolution of inflammation. Example: delivery of SPM such as resolvin to treat periodontal disease.
prostaglandins (PG)	PGs are lipid mediators that play an important role in inflammation and are produced by the enzymatic action of COX. They have 10 subclasses, of which PGs D through I are important mediators of inflammation. Antiinflammatory medications act primarily by inhibiting PGs and their actions.
specialized proresolving mediators (SPM)	Lipid mediators that play a major role in the resolution of inflammation.

Fast Facts

Cardinal signs of inflammation	Redness (rubor), pain (dolor), high temperature (calor), swelling (tumor), and loss of function (functio laesa).
Acute inflammation	Immediate host response (within minutes or hours) to tissue injury; beneficial when the response dies down soon after the pathologic challenge is effectively met and dealt with.
Chronic inflammation	Inflammation that lasts for prolonged/indefinite periods (months or years); occurs predominantly due to ineffective shutting down of acute inflammation.
Resolution of inflammation	An active process (not passive, as previously thought).
Lipid mediators	Play a major role in the resolution of inflammation and are called specialized proresolving mediators (SPMs).
Examples of SPMs	Lipoxins, resolvins, protectins, and maresins.
Mode of activity	During inflammation, lipid mediator class switching occurs; as a result, a switch occurs from production of the initiators of acute inflammation (like prostaglandins and leukotrienes) to production of SPMs, resulting in the resolution of inflammation.
Mechanism	Rhetoric SPMs block infiltration of neutrophils and eosinophils, recruit monocytes, and enhance phagocytosis and clearance of neutrophils by macrophages, all leading to inflammation resolution.
Lipoxins	Lipoxins are natural proresolving molecules derived from arachidonic acid. Aspirin induces the synthesis of a special class of lipoxins called aspirin-triggered lipoxins (ATLs).
Other SPMs	Resolvins, protectins, and maresins are biosynthesized from dietary omega-3 polyunsaturated fatty acids.
Resolvin series	E-Series and D-series resolvins are biosynthesized from eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), respectively. EPA and DHA are omega-3 polyunsaturated fatty acids.
Plausible Pathology	In periodontitis, there is a failure of the signals that activate resolution of inflammation and return to homeostasis, and so chronic inflammation persists.
Therapeutic potential	Recent preclinical (animal) studies indicate that SPMs are excellent candidates to prevent and/or treat chronic inflammatory conditions such as periodontitis.
Regeneration potential	Animal studies using an experimental periodontitis model indicate that in addition to effectively resolving inflammation, SPMs also promote the regeneration of lost periodontal tissues.
Approaches	Proresolution and antiinflammation are completely different therapeutic strategies.
Benefits of SPMs	Stimulation of resolution versus the conventional blocking of active inflammation (antiinflammation) is a novel and exciting area that has potent clinical implications with fewer adverse effects.

Core Knowledge

Introduction

Resolution of inflammation is an active process that results in a return to tissue homeostasis. "Turning off" inflammation is an important health-restoring phenomenon mediated by a class of endogenous lipid mediators (lipoxins, resolvins, protectins, maresins). Failure to "turn off" the inflammatory host response may result in continued damage to host tissues, long after the elimination of the microbial challenge that initiated inflammation in the first place.

It is important to review certain aspects of the phenomenon of inflammation before its resolution can be understood.

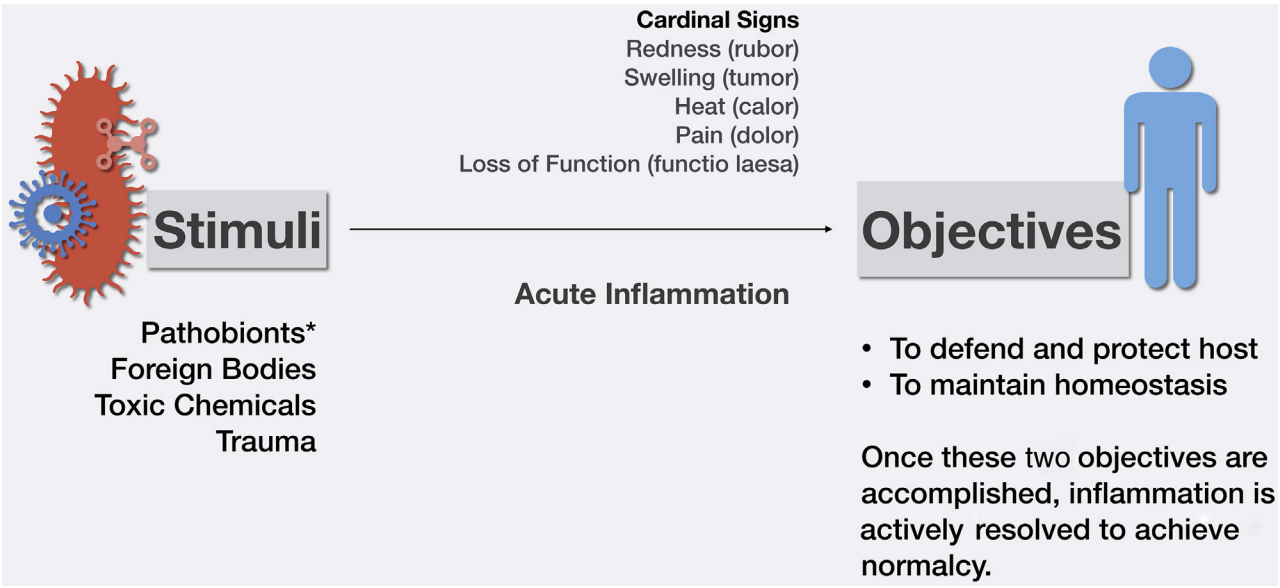
Inflammation

Inflammation is a protective physiologic response that defends the host from infection or injury and returns the body to normal by ensuring tissue homeostasis. It is protective only as long as it subsides after the threat of insult/injury/infection has been dealt with and resolved (Fig. 7.1).

CLINICAL CORRELATE

Why is it vital for a clinician to be aware of the importance of inflammation resolution and its mediators?

New evidence indicates that chronic inflammatory conditions like periodontitis may be associated with "frustrated" resolution of inflammation and not just the presence of initial inflammation. It is now important to distinguish between "protective" and "destructive" inflammation. This new knowledge forces the scientific community to rethink existing approaches to therapeutic control of chronic inflammation. Hence it is important for a clinician to be aware of advances in research regarding inflammation resolution, because it is a host modulatory approach that holds great promise.



• **Fig. 7.1** Protective Inflammation: Cardinal Signs and Objectives. Acute inflammation is considered a protective response when the cardinal signs are actively resolved after the main objectives of defense against pathogenic stimuli are fulfilled. **A pathobiont is any potentially disease-causing organism which, under normal circumstances, is beneficial (symbiotic) to the host.*

Acute Inflammation Is Self-Limited

A localized inflammatory response to an injury or infection—one that is **spatially** (restricted to a limited area/space) and **temporally** (stops in a timely manner) regulated by specialized proresolution lipid mediators (SPMs)—is considered to be beneficial for restoration of tissue health.

The sequence of vascular and cellular responses in acute inflammation is:

1. **Vascular response**, the clinical result of which is redness, heat and edema/swelling due to increased blood flow, vasodilation, and increased vessel permeability.
2. **Polymorphonuclear neutrophil (PMN) recruitment** from within blood vessels and infiltration of neutrophils into affected tissue. Clinical result is pain, tissue damage, and pus formation.
3. **Monocyte recruitment** from blood vessels and differentiation into macrophages. Clinical result is tissue repair/healing after inflammation resolution. If inflammation does not resolve, macrophages usher in dysfunctional adaptive immunity and aggravate persistent inflammation, and chronic disease sets in. Fig. 7.2 shows a representation of the three major events in self-limiting acute inflammation.

A better understanding of the complex mechanisms of lipid mediators (proinflammatory versus proresolution) allows us to use this information and develop novel therapeutic strategies to manage chronic inflammatory diseases, such as periodontitis Table 7.1 lists the differences between proinflammatory and proresolution lipid mediators.

Resolution of inflammation, initiated by an active class switch from proinflammatory lipid mediators to the production of proresolution mediators, results in:

- Cessation of leukocyte infiltration—due to the selective interaction of SPMs with receptors on innate immune cells
- Vascular permeability/edema returning to normal with neutrophil death (apoptosis)
- Efferocytosis—the nonphlogistic infiltration of monocytes and macrophages that effect removal of necrotic debris, dead microbes and apoptotic neutrophils from inflammatory site.

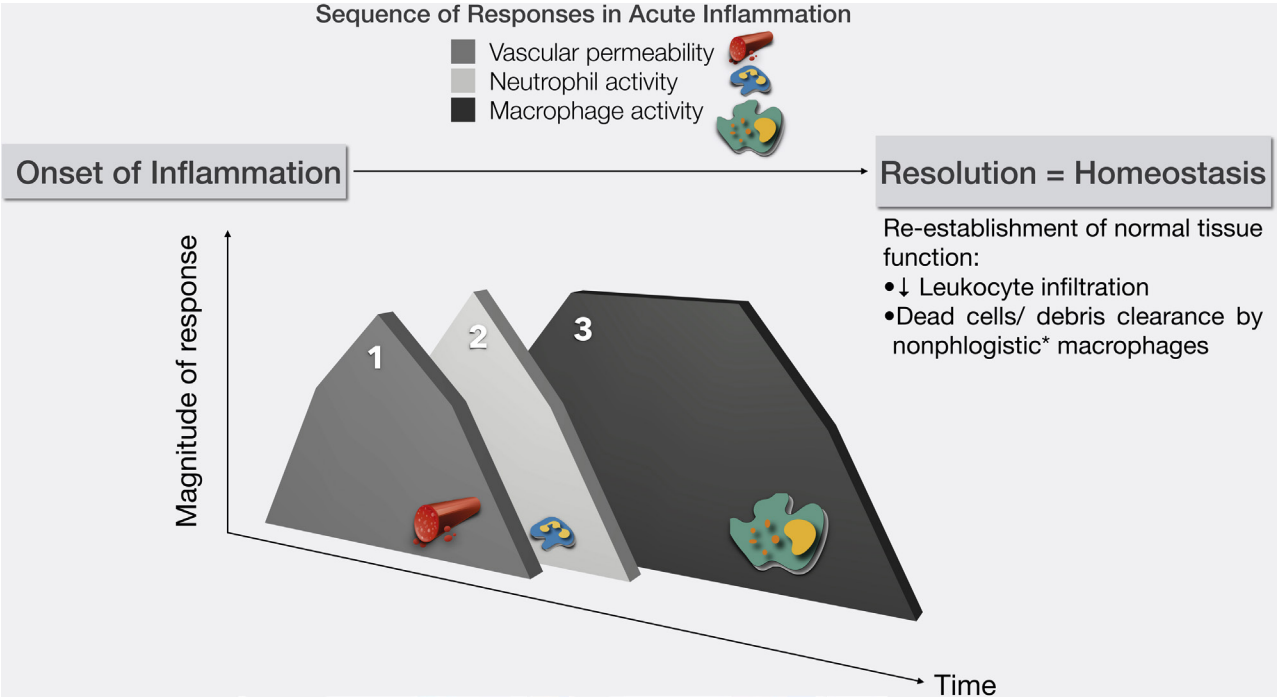
CLINICAL CORRELATE

What is the limitation of proresolution therapeutic strategies?

Multiple Heterogenous resolution pathways exist that are both tissue- and stimulus-specific. This makes it difficult to identify a single proresolution mediator as a therapeutic panacea or universal solution for multiple disease states.

See Fig. 7.3 for a diagrammatic representation of events occurring during class switching.

Specialized proresolution mediators (SPM)—the SPMs are derived from fatty acids (see Table 7.1) and include lipoxins, resolvins, protectins, and maresins. They exhibit overlapping activities that work to actively resolve inflammation. Table 7.2 provides an overview of the functions of individual SPMs known so far. While all the SPMs can stimulate efferocytosis and control pain, resolvins alone have been shown to affect all the hallmark functions contributing to the resolution of inflammation.

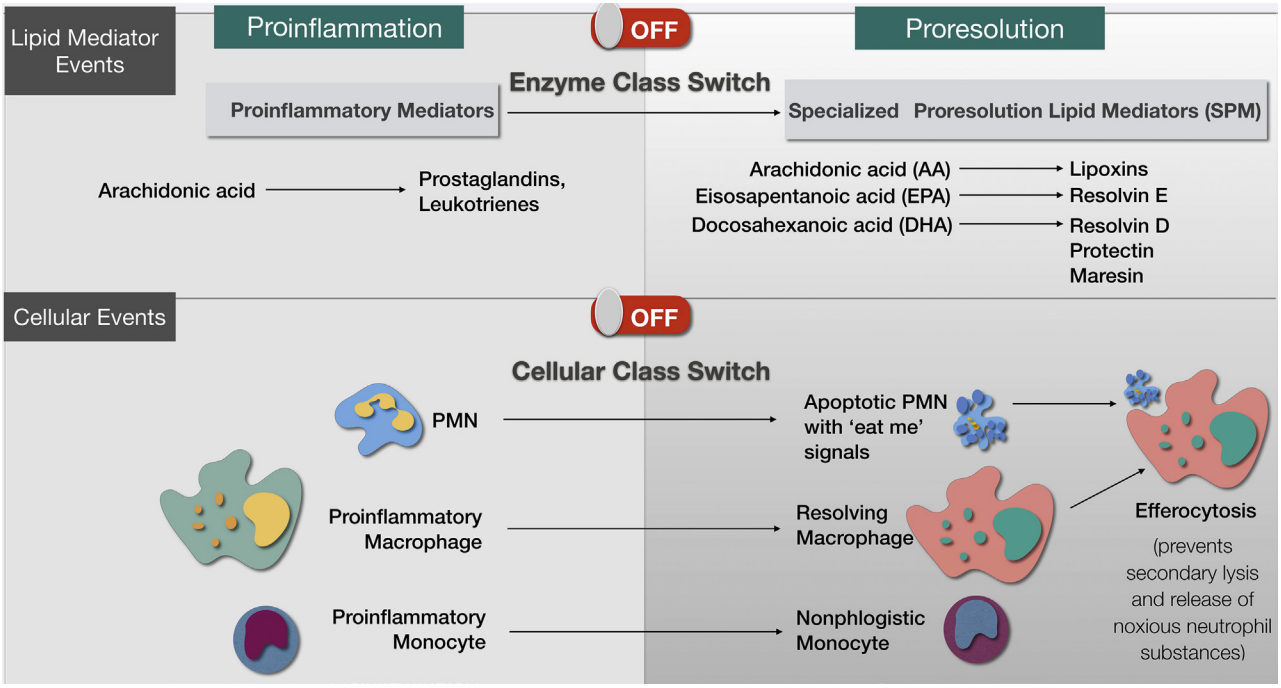


• **Fig. 7.2** Self-Limiting Inflammation. Self-limiting inflammation includes three major events that exhibit peak activity in sequence (with some degree of overlap before active resolution begins): vascular events, neutrophil activity, and macrophage activity. The graph displays the normal rise and fall in these phenomena in response to pathogenic stimuli, from inflammation onset to inflammation resolution. Resolution of inflammation is considered as "failed" when the respective cellular/molecular activities do not decline effectively after reaching their peaks. When acute inflammation fails to resolve, the tissue does not attain homeostasis, and chronic disease sets in.[1] **Nonphlogistic: non-fever-producing*.

TABLE 7.1 Differences Between Proinflammatory and Proresolution Lipid Mediators

	Proinflammatory mediators	Proresolution mediators
Objective	Induce inflammation as immediate host defense	Limit inflammation to restore tissue homeostasis
Derivatives of polyunsaturated fatty acids (PUFA)	From arachidonic acid: prostaglandins, leukotrienes, prostacyclins, thromboxane, endoperoxidase	From arachidonic acid: lipoxins
		From eicosapentanoic acid (EPA): resolvin E series
		From docosahexanoic acid (DHA): resolvin D series, protectins, maresins
Cellular functions elicited	↑ chemotaxis	↓ chemotaxis
	Diapedesis	Nonphlogistic phagocytosis
	NF-κB activation	NF-κB inhibition
	Inflammatory cytokine production	Class switch of enzymes and cellular activity
	Apoptosis	Efferocytosis
Tissue response	Vasodilation	Vasoconstriction
	Pain	Analgesic
	Swelling, heat	Return to homeostasis

Apoptosis: cell suicide. Chemotaxis: movement of an immune cell in response to gradient concentration of a substance (can be a cytokine, chemical, etc). Diapedesis: the exit of blood cells from within capillaries into extravascular spaces. Efferocytosis: phagocytosis of apoptotic neutrophils by "resolving" macrophages that do not induce a concomitant inflammatory response.[2] NF-κB: nuclear factor kappa light chain enhancer of activated B cells is a protein complex that controls proinflammatory signaling pathways. Nonphlogistic: non-fever-producing.



• **Fig. 7.3** "Class Switch" During Resolution of Inflammation. Two kinds of events occur: Lipid mediator events: Enzyme class switches result in production of SPMs instead of proinflammatory mediators. SPMs then actively start resolving inflammation by decreasing leucocyte infiltration and clearing out dead cells and debris, thus restoring tissue homeostasis. Cellular events:

1. PMN changes—After the fight against pathogenic challenges via acute inflammation, neutrophils undergo apoptosis (cell suicide) and efferocytosis. In efferocytosis, dead cells are removed before their membrane integrity is lost, thus preventing toxic content leakage into tissues.
2. Monocyte/macrophage changes—Monocytes recruited from blood vessels into target tissue during inflammation resolution are non-fever-producing (nonphlogistic). Resident macrophages within tissues convert to "resolving macrophages" from the "proinflammatory" phenotype.[1]

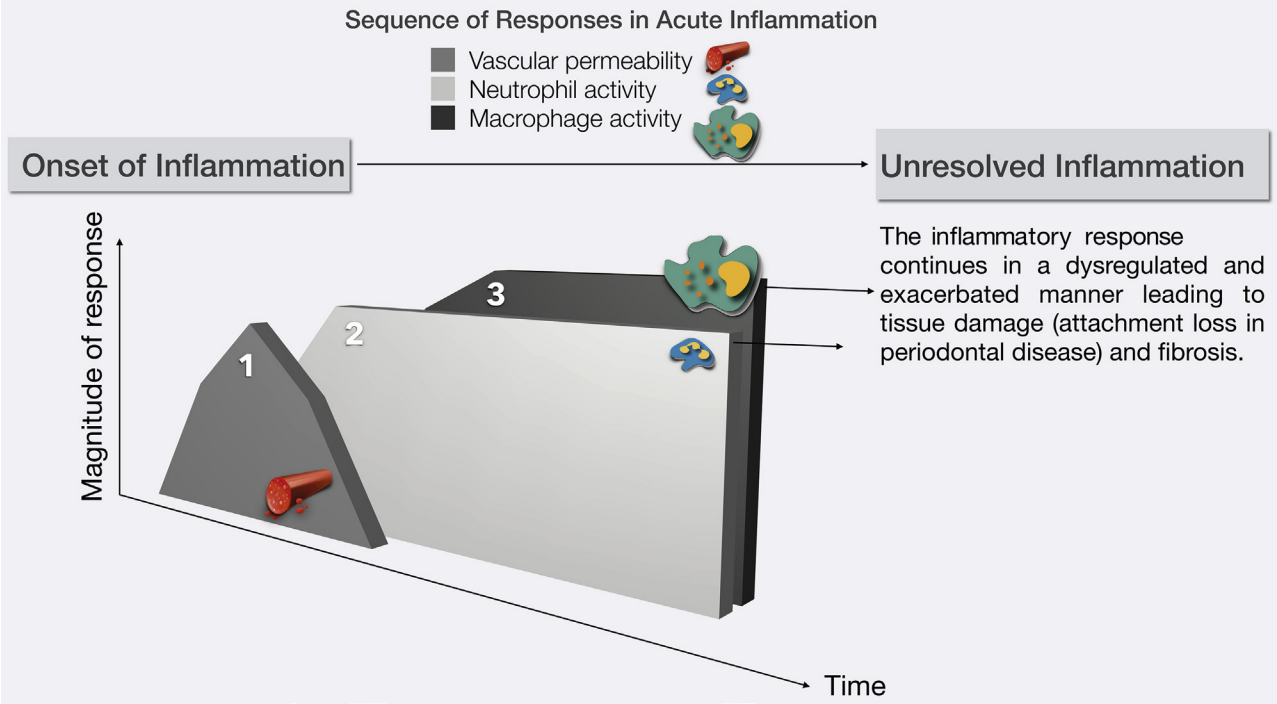
TABLE 7.2 Actions of Specialized Proresolving Mediators

	Lipoxins	Resolvins	Protectins	Maresins
Decrease PMN infiltration	✓	✓	✓	
Stimulate efferocytosis	✓	✓	✓	✓
Promote tissue regeneration/ remodeling		✓		✓
Pain control	✓	✓	✓	✓
Protective action in neural tissues			✓	

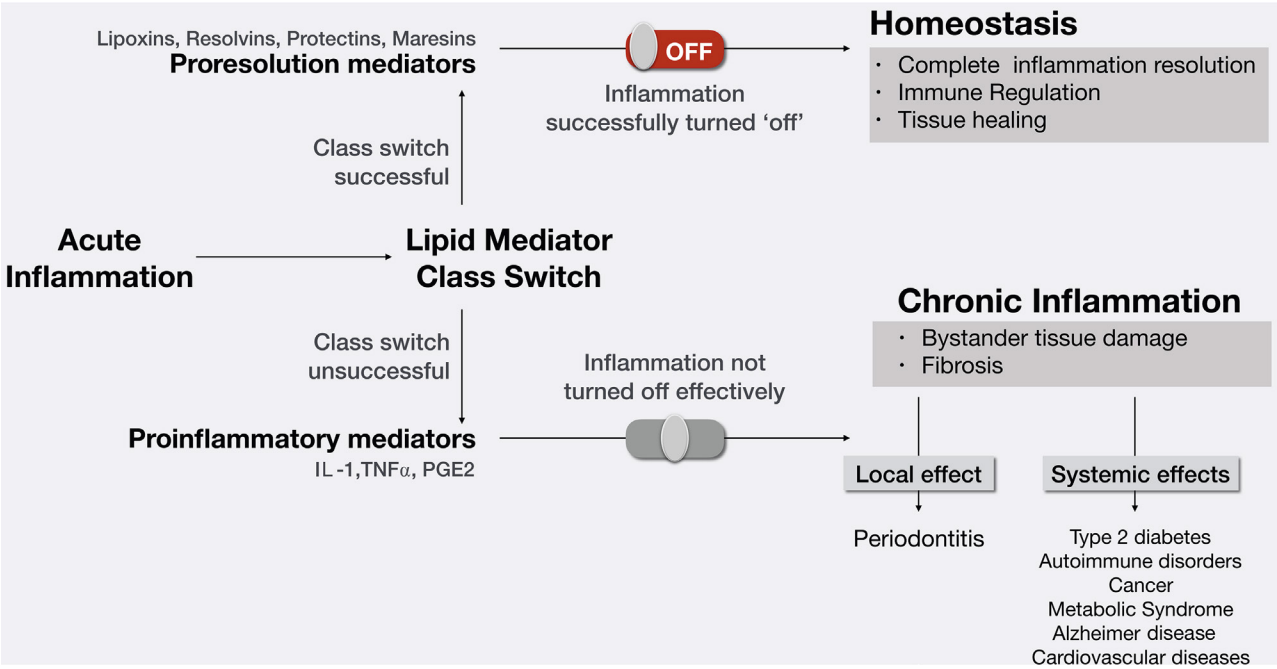
Unresolved Chronic Inflammation in Periodontal Disease

Bacteria initiate periodontal disease, but are not solely responsible for disease progression. In susceptible individuals the progression to chronicity is characterized by failure of the immune system to remove pathogenic microbes, and inefficient clearing of dead neutrophils. When the acute host response that defends the periodontium against pathologic challenges continues as a chronic exacerbated inflammation, tissue integrity is threatened (Fig. 7.4).

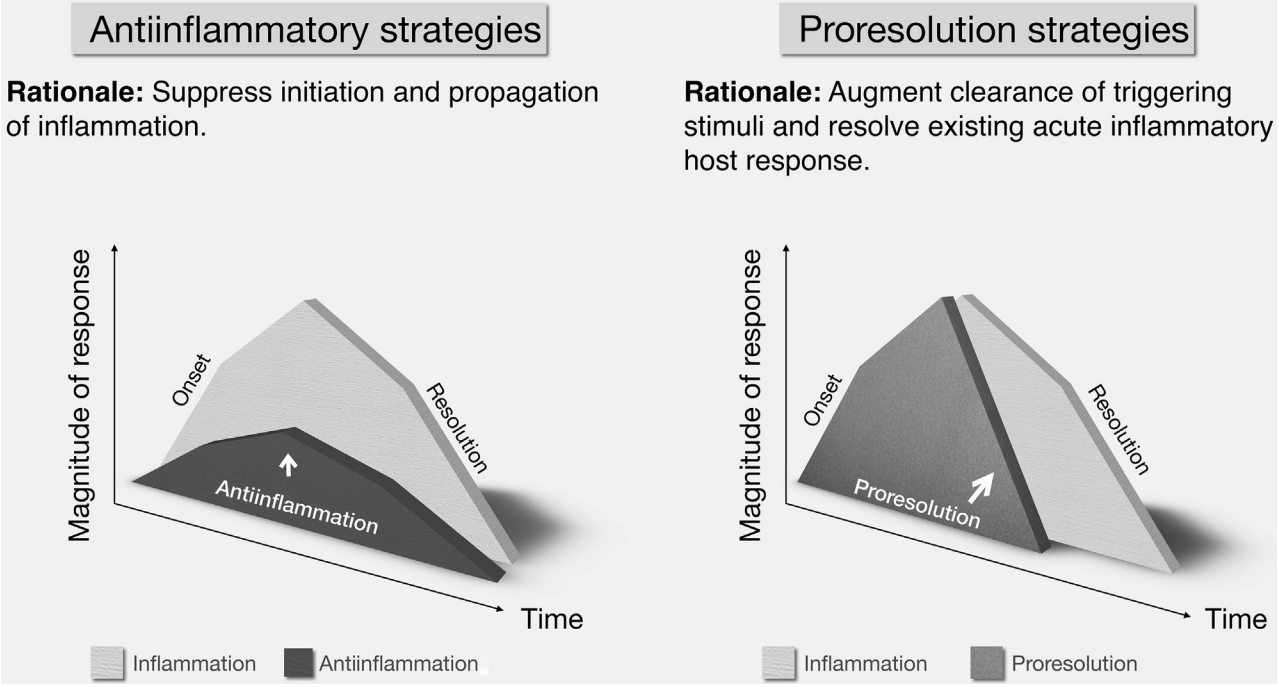
Systemic Link—Excessive inflammatory response to pathologic insults results in sustained local production of proinflammatory mediators (IL-1, TNFα, PGE_2, etc.) within periodontal tissues. Their spillage into the systemic circulation can lead to harmful impacts on general health, contributing to worsening of existing medical conditions. The hyperinflammatory response contributing to many diseases like Alzheimer's, diabetes, metabolic syndrome, etc., may reflect a failure in class switching of lipid mediators (see Fig. 7.5).



• **Fig. 7.4** Unresolved Inflammation: Sequence and Magnitude of Cellular Response. Compare with Fig. 7. 2, where the activity of inflammatory cells (PMNs and macrophages) is self-limiting. Inflammation resolution failure is a plausible explanation for the pathologic transition from reversible gingivitis to irreversible periodontitis.[3]



• **Fig. 7.5** Consequences of Ineffective/Failed Class Switching of Lipid Mediators. The outcome of acute inflammation—whether chronicity or resolution—is influenced by class switching of lipid mediators. When this lipid mediator class switch fails to occur, dysregulated immune-inflammatory events lead to the release of proinflammatory mediators. Unresolved inflammation is also a hallmark of other conditions (e.g., diabetes, autoimmune disorders, metabolic syndrome); periodontal disease may aggravate such conditions due to spilling of excessive proinflammatory mediators into the systemic circulation.[1] PGE2: prostaglandin E2; TNFα: Tumor Necrosis Factor-α.



• **Fig. 7.6** Differences Between Antiinflammation and Proresolution Therapeutic Strategies.

Therapeutic Actions of Resolution Mediators

The biosynthesis of SPMs by key enzymes inducing a "class switch" from proinflammatory to proresolving actions is a potential therapeutic intervention to treat chronic inflammatory conditions, including periodontal disease.

Antiinflammatory and proresolution therapeutic strategies—Proresolution and antiinflammation are completely different therapeutic strategies. While antiinflammatory mediators block and suppress pathways that initiate acute inflammation, proresolution mediators are specialized lipids that exert their effects after acute inflammation has played its part in host defense (Fig. 7.6).

CLINICAL CORRELATE

What are the differences between antiinflammatory and proresolution therapeutic strategies?

Proresolution strategies may be beneficial and perhaps even superior to antiinflammatory strategies because of their potential to enhance tissue healing and function with minimal adverse effects.

Timing of action: proresolving mediators (SPM) act *after* allowing for normal inflammatory response, whereas antiinflammatory mediators act to block or suppress inflammation itself.

Animal studies of experimental periodontitis have been able to identify few possible treatment interventions using application of exogenous lipid mediators (See Table 7.3).

TABLE 7.3 Therapeutic Actions of SPMs in Periodontitis (Animal Models)[4,5]

SPMs	Therapeutic Actions
Lipoxin A4	Reduction (↓) in attachment loss (bone and connective tissue)
Aspirin-triggered lipoxin (ATL)	Increase (↑) in tissue healing ↓ PMN infiltration
Resolvin E1	↓ Bone loss
Maresin 1	↓ Osteoclast number ↑ Tissue regeneration

CLINICAL CORRELATE

What other potential proresolution strategies (other than SPMs) have been identified?

Apart from SPMs, which are lipid mediators, other classes of mediators are proteins (annexin, melanocortin), gaseous mediators, and purines (adenosine).

Conclusions

Termination of acute inflammation, previously recognized as a *passive* and natural decay of proinflammatory signals, is now understood to be a tightly regulated, *active*, biochemical program that returns tissues to homeostasis.

- Disproportionate host responses that continue in an exacerbated manner result in a chronic inflammatory environment within the periodontium that ultimately results in tissue damage.
- The chronicity of the lesion modifies overall tissue responses in remote regions of the body, with a transient or permanent impact on general health.
- SPM-based therapeutics are effective in utilizing exogenous lipid mediators to restore tissue homeostasis by limiting and actively resolving inflammation. The potential for clinical use of such host-modulatory therapy in many disease conditions, including periodontitis, is promising and needs to be investigated further.

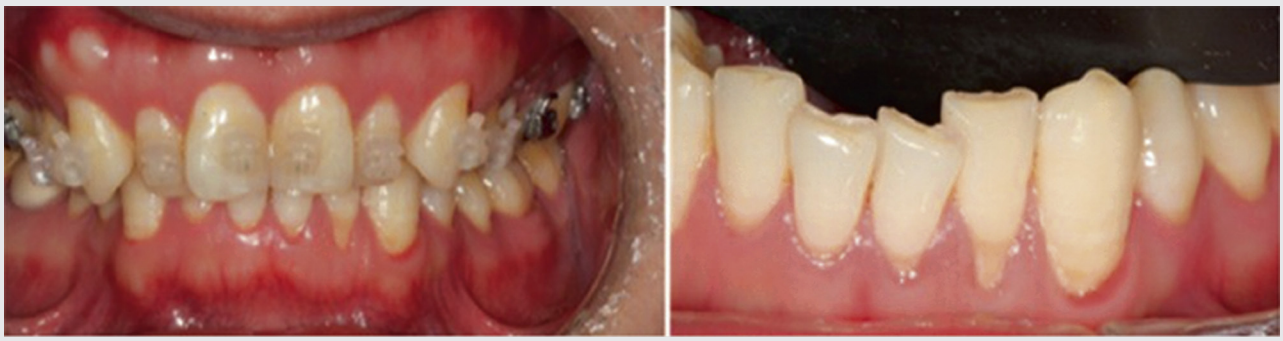
CLINICAL CORRELATE

How will tackling "defective inflammation resolution" potentially help with periodontal therapy?

When defective endogenous resolution of inflammation underlies the inflammatory phenotype in periodontitis, application of exogenous SPM therapeutic molecules has the potential to prevent further attachment loss and to enhance regeneration of lost tissue, leading to attachment gain.

CASE-BASED LEARNING EXERCISE

Scenario: "My gums are inflamed." A healthy 30-year-old male patient with crowding presented with localized plaque and generalized inflammation. The patient, a graduate student, reported stress that was affecting his sleep. His Oral Hygiene Index was average. The patient avoided brushing areas where inflammation and bleeding were persisting. He wanted his gums to be "healthy" prior to orthodontic treatment. Clinical exam revealed probing depths to be in the range of 2–3 mm, BOP was 42%. Recession noted in the mandibular left canine, lateral incisor, and central incisors.



Questions

1. All of the following factors are associated with gingivitis, except:
- **a.** Nutrition deficiency.
- **b.** Hormonal changes (in women).
- **c.** Biofilm.
- **d.** Medications.
- **e.** Bone loss.

2. All of the following are resolution of inflammation mediators, except:
- **a.** Resolvins.
- **b.** Protectins.
- **c.** Lipoxins.
- **d.** Maresins.
- **e.** Prostaglandins.

3. Why has the inflammation not resolved on its own in this case?
- **a.** Teeth malalignment
- **b.** Sleep disorder
- **c.** Microbial plaque
- **d.** Orthodontic brackets

4. Proresolution strategies may be _____ to anti inflammatory strategies:
- **a.** Inferior
- **b.** Similar
- **c.** Superior

This chapter was developed from Chapter 10 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: e

Explanation: Bone loss is not characteristic of gingivitis. Gingivitis is a reversible inflammatory mechanism modulated by hormones, genetic factors, medications, and environmental factors. Upon removal of the etiologic challenge, resolution of inflammation ensues, which returns the tissue to a healthy state.

2. Answer: e

Explanation: Prostaglandins are proinflammatory mediators derived from COX-2 and responsible for increasing inflammation. Proresolution lipid mediators are active lipids responsible for activating cells to return to homeostasis. Upon binding to cells, resolution lipids are capable of increasing phagocytosis and activating the clearance of dead cells and microbes.

3. Answer: c

Explanation: The etiologic factor of gingivitis in this case is the microbes in dental plaque. Due to the presence of the etiologic factor, inflammation is sustained, not allowing the resolution phase to take over. Once the cause of the inflammation is addressed, it will allow the resolution to take its course.

4. Answer: c

Explanation: Proresolution strategies may be superior to antiinflammatory strategies because of their potential to enhance tissue healing and function with minimal adverse effects.

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8

Local Predisposing Factors for Periodontal Disease

Relevant Terminology

Terminology	Explanation
calculus	• Mineralized dental plaque • Does not itself contribute directly to gingival inflammation
food impaction	• Forceful wedging of food into the periodontium by occlusal forces
materia alba	• An accumulation of microorganisms, desquamated epithelial cells, leukocytes, salivary proteins and lipids • Lacks internal pattern, less adherent than dental plaque
plunger cusps	• Cusps that tend to forcibly wedge food into interproximal embrasures • Can be seen when missing teeth are not replaced and the relationship between the proximal contacts of adjacent teeth is altered
reversal phenomenon	• Calculus formation continues until it reaches a maximum, after which it may be reduced in amount. The time required to reach the maximal level has been reported to be between 10 weeks and 6 months • Bulky calculus is vulnerable to mechanical wear from food and from the cheeks, lips, and tongue movement. The decline from maximal calculus accumulation is referred to as the *reversal phenomenon*
subgingival calculus	• Located apical to the gingival margin, detected by tactile perception or radiographs • Typically hard and dense; dark brown or greenish black in color • Firmly attached to the tooth surface
supragingival calculus	• Located coronal to the gingival margin • White or whitish-yellow in color; hard with a claylike consistency • Easily detached from tooth surface
tongue thrusting	• Excessive lateral pressure on the anterior teeth, which may result in the spreading and tilting of the anterior teeth • Contributing factor to tooth migration and development of an anterior open bite • Mouth breathing may be observed with this habit

Fast Facts

Composition of dental calculus	• 70%–90% inorganic content • The four main crystal forms are hydroxyapatite (58%), magnesium whitlockite (21%), octacalcium phosphate (12%), and brushite (9%) • Brushite is common in mandibular anterior regions, magnesium phosphate is common in posterior regions
Calculus formation	• The first areas to exhibit calculus formation are the facial aspect of maxillary molars and the lingual surfaces of mandibular incisors • Precipitation of minerals starts at 1–14 days of plaque formation; calcification can start after as little as 4–8 hours • Calcification starts at the inner surface adjacent to the tooth structure

Continued

Fast Facts—cont'd

Calculus attachment to cementum	Calculus attachment is through: • Organic pellicle • Mechanical locking • Close adaptation to gentle depression or sloping mounts of unaltered cementum • Bacterial penetration into cementum surface
Local predisposing factors that affect periodontium	• Margins of restorations (open margins, overhang margins, subgingival margins) • Retained cement • Root perforations, vertical root fractures, endodontic failures • Overcontoured crowns, open contacts • Self-curing acrylic • Design of removable partial denture • Restorative procedures (rubber dam clamps, burs, retraction cords, etc.) • Malocclusion
Posterior teeth with open contact	• Posterior teeth with open contact and food impaction exhibit greater probing depth and clinical attachment loss than sites without food impaction
Excessive anterior overbite	• Common cause of food impaction on the lingual surfaces of the maxillary anterior teeth and the facial surfaces of the opposing mandibular teeth • Can result in attachment loss with gingival recession
Traumatic occlusion	• Occlusal disharmonies may cause injury to the supporting periodontium, widened periodontal ligament (PDL) space, a reduction in the collagen content of PDL fibers, increased vascularity, and increased leukocyte infiltration and osteoclasts on alveolar bone • Teeth with initial occlusal discrepancies show worse prognosis and more mobility than teeth without initial occlusal discrepancies
Orthodontic treatment and alveolar bone loss	• Orthodontic treatment has little effect on bone level in adolescents • Orthodontic treatment in adults with active periodontitis (deep probing depth with bleeding on probing) could accelerate the periodontal disease process
Risk factors for root resorption	• Duration of treatment • Magnitude of the force applied • Direction of tooth movement • Continuous versus intermittent application of forces
Extraction of impacted third molars	• The extraction of impacted third molars often results in a vertical bone defect distal to the second molars irrespective of flap design. More frequently seen in patients >25 years of age • Presence of plaque, bleeding on probing, root resorption, widened follicle, and the inclination of the third molar are associated with vertical defects
Self-inflicted injuries that affect periodontium	• Improper use of toothbrushes • Wedging toothpicks between teeth • Application of fingernail pressure against gingiva • Pizza burn • Chemical injury caused by aspirin, cocaine, allergic reaction to toothpaste or chewing gum, chewing tobacco, concentrated mouth rinses
Aspirin-induced chemical burn	• Exhibits Vacuoles with serous exudates and an inflammatory infiltrate in the connective tissue
Smokeless tobacco	• Leukoplakic lesion (smokeless tobacco hyperkeratosis) • A chevron-like pattern of hyperkeratosis with focal areas of inflammation • Hyperplasia in the basal cell layer • Increased incidences of gingival recession, cervical root abrasion, and root caries
Trauma associated with oral jewelry	• Lip or tongue piercing is associated with gingival injury or recession; damage to teeth, restorations, and fixed porcelain prostheses; scar tissue formation; and potentially metal hypersensitivity
Aggressive tooth brushing	• Acute trauma from brushing: painful gingival ulcer and gingival abrasions • Chronic trauma from brushing: gingival recession on the buccal and lingual surfaces • Improper use of floss may result in the laceration of interdental papilla
Radiation therapy	• Typical dose for head and neck: 5000–8000 cGy, usually given with incremental dose (fractionation); 100–1000 cGy per week • Effects: Soft tissue ischemia and fibrosis, bone becomes hypovascular and hypoxic • Dermatitis and mucositis develops 5–7 days after the initiation of radiation therapy • Chlorhexidine rinse containing alcohol can irritate and intensifies pain • Customized tray for fluoride application is used for caries prevention

Fast Facts—cont'd

Osteoradionecrosis	• Allow complete soft tissue healing before the commencement of radiotherapy • Risk factors for developing osteoradionecrosis include sites with probing depth >5 mm, plaque score >40%, alveolar bone loss >60% • Nonrestorable teeth and teeth with significant periodontal problems should be extracted before radiation therapy • Elective periodontal surgery should be performed in consultation with oncologist • The administration of pentoxifylline with vitamin E as antioxidant therapy may promote revascularization and treatment of osteoradionecrosis sites • Hyperbaric oxygen therapy has limited efficacy for osteoradionecrosis

Core Knowledge

Introduction

The main cause of gingival inflammation is dental plaque biofilm, and there are several factors (local and systemic) that predispose an individual toward plaque accumulation. This chapter focuses on the local factors within the oral cavity that predispose to plaque accumulation and consequent gingival inflammation and disease of periodontal structures. These may be categorized as:

- **Pathophysiologic factors**—dental calculus
- **Iatrogenic factors**—faulty restorations, periodontal complications associated with orthodontic therapy, extraction of third molars
- **Anatomic factors**—tooth-related, mucosa-related, and jaw-related factors

Dental Calculus

Dental calculus is formed as a result of mineralization (calcification) of the plaque biofilm. Mineralized biofilms, penetrated by crystalline calcium phosphates of various types, can develop above (supragingival calculus) or below (subgingival calculus) the free gingival margin as moderately hard deposits on the surfaces of teeth and dental prostheses. Table 8.1 lists the different characteristics of both types of calculus.

Attachment of Dental Calculus to Tooth Surface

The manner of attachment of dental calculus to the tooth surface affects the ease of its removal during instrumentation. Four modes of attachment have been identified and these are described in Fig. 8.1.

Formation of Dental Calculus

- The presence of plaque biofilm is a prerequisite for calculus formation; it is within this soft deposit that mineralization occurs. However, all plaque does not necessarily undergo calcification. Plaque that does not develop into calculus reaches a plateau of maximal mineral content within 2 days.
- Saliva and gingival crevicular fluid (GCF) furnish the minerals required for supragingival and subgingival calculus formation, respectively.
- Phosphorus may be more critical than calcium for plaque mineralization.
- Calcification requires the binding of calcium ions to the carbohydrate-protein complexes of the plaque organic matrix and the precipitation of crystalline calcium phosphate salts. This begins along the inner surface of the biofilm adjacent to the tooth structure.
- Crystals form initially in the intercellular matrix, then on the bacterial surfaces, and finally within the bacteria.
- Separate foci of calcification increase in size and coalesce to form solid masses of calculus.
- Calculus is formed in layers, which are often separated by a thin cuticle that becomes embedded in the calculus as calcification progresses.

Clinical photos of supragingival and subgingival calculus are shown in Fig. 8.2.

Theories Regarding the Mineralization of Calculus

The exact mechanism of mineralization of plaque that leads to calculus formation is still unknown. Various theories have been put forward, which mainly relate to propositions of mineral precipitation and mineral seeding.

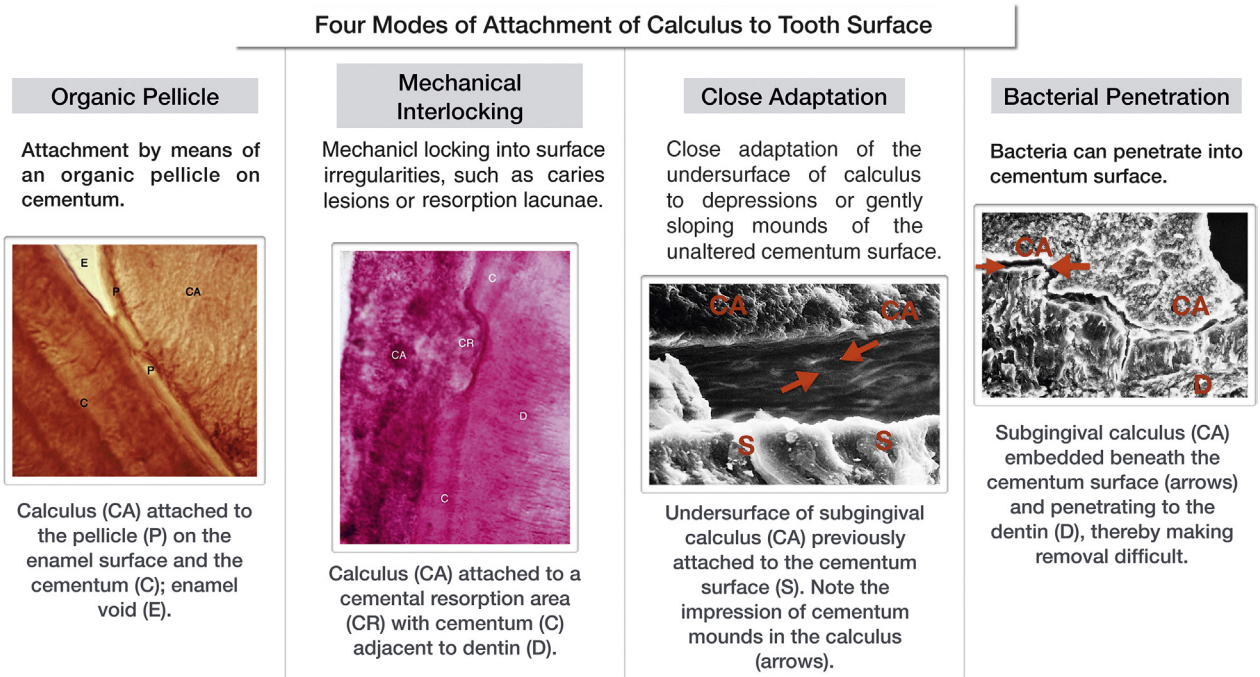
Mineral Precipitation

This is based on the theory that salts of calcium phosphate precipitate out, leading to calculus formation. The precipitation of salts may be attributed to:

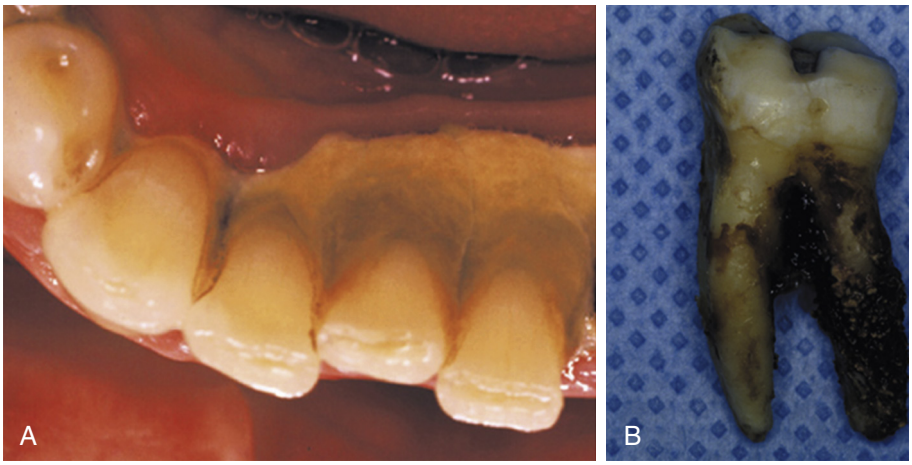
- **Lowering of the precipitation constant by a local rise in pH (alkaline environment)**—this can be brought about by:
  - Losing carbon dioxide when saliva flows out of duct orifices (hence the quick formation of calculus opposite to salivary gland duct openings)
  - Ammonia formation by plaque bacteria

TABLE 8.1 Supragingival and Subgingival Calculus

	Supragingival Calculus	Subgingival Calculus
Primary source of minerals	Saliva	Gingival crevicular fluid (GCF)
Location	Coronal to the crest of marginal gingiva and visible in oral cavity	Apical to the crest of marginal gingiva and not visible on routine clinical examination; deflecting gingival margin away from the tooth surface (e.g., by blowing air) can expose subgingival deposits
Color	White/whitish yellow; influenced by tobacco smoking and food pigments	Dark brown/greenish black
Consistency	Hard, clay-like	Dense, flint-like
Strength of adherence to tooth/prosthesis surface	Easily detachable	Firmly attached
Rate of recurrence	Rapid	Not as rapid as supragingival calculus
Predominant crystalline forms of minerals found within structure	Hydroxyapatite, octacalcium phosphate	Hydroxyapatite, magnesium whitlockite
Calcium-to-phosphate ratio	Lower than subgingival calculus	Higher than supragingival calculus
Microbial profile	Filamentous microbes dominate and are oriented at right angles to the surface of teeth	Cocci, rods, and filaments found with no distinct pattern of orientation
Other notable features	Commonly noticed in relation to lingual aspects of mandibular incisors and buccal surface of maxillary molars due to proximity to the orifices of salivary gland ducts Also noticed covering the occlusal surfaces of teeth lacking functional antagonists	When gingival tissues recede, subgingival calculus becomes exposed and is reclassified as supragingival calculus



• **Fig. 8.1** Modes of Attachment of Calculus to Tooth Surface. Calculus may attach to cementum via: (1) organic pellicle, (2) mechanical locking into surface irregularities, (3) close adaptation to gentle depression or sloping mounts of unaltered cementum, or (4) bacterial penetration into cementum surface.[1,2] (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)



• **Fig. 8.2** Clinical Images of Supragingival and Subgingival Calculus. Extensive supragingival calculus deposits in the mandibular anterior sextant (A) and subgingival calculus deposits on an extracted molar tooth root (different patient) (B). (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

- **Stagnation of saliva**—this in turn causes colloidal proteins (previously bound to calcium and phosphate ions within saliva) to settle out, resulting in calcium phosphate salt precipitation.
- **Enzyme actions**—phosphatases secreted by plaque bacteria increase the availability of free phosphate ions. Esterases secreted by plaque bacteria increase free fatty acids that combine with calcium and magnesium ions to form soaps first, and later cause precipitation of calcium phosphate salts.

Mineral Seeding

- Also called *epitactic concept* or *heterogenous nucleation*
- Seeding agents (plaque intercellular matrix being a possible candidate) induce small foci of calcification within the plaque matrix that enlarge and coalesce to form a calcified mass

Other Predisposing Factors

The local factors (other than calculus) that predispose an individual to plaque formation and consequent gingival inflammation include:

Plaque-retentive iatrogenic factors:

- Faulty restorations with:
  - Overhanging margins
  - Subgingival margin placement
  - Gaps between restoration margins and finish lines on teeth
  - Overcontoured restoration
  - Marginal ridge discrepancies and improper proximal contacts
  - Grooves and scratches in the surface of acrylic resin, porcelain, or gold restorations
  - Retained cement within gingival sulcus
  - Excessive pontic-to-tissue contact
  - The use of rubber dam clamps, matrix bands, and burs in such a manner as to lacerate the gingiva
- Periodontal complications associated with orthodontic therapy. Orthodontic therapy may affect the periodontium by favoring plaque retention, by directly injuring the gingiva as a result of overextended bands, or by creating excessive or unfavorable forces (or both) on the tooth and its supporting structures.
- Extraction of impacted third molars. This often results in the creation of vertical defects distal to the second molars.

Plaque-retentive anatomic factors:

- Tooth-related factors (e.g., developmental grooves, cervical enamel projections)
- Mucosa-related factors (e.g., gingival recession, gingival enlargement, abnormal frenal pull)
- Jaw-related factors (crowding, malocclusion)

Conclusion

While many factors cause plaque retention, this does not mean they are the main etiologic factors of periodontal disease. Their role is primarily in triggering plaque biofilm accumulation and changing the local microenvironment such that the individual becomes predisposed to developing periodontal disease. Even so, removal of calculus and other local predisposing factors is still considered one of the main starting points of comprehensive periodontal therapy.

CLINICAL CORRELATE

What is the role of dental calculus in the development of periodontal disease?

Calculus by itself does not contribute directly to gingival inflammation. Following the mineralization process, the bacteria within calculus lose their virulence, because mineralized microbes are hardly capable of any metabolic activity. Like other retentive factors such as open crown margin or an overhanging restoration, calculus retains dental plaque, which contributes to gingival inflammation.

CLINICAL CORRELATE

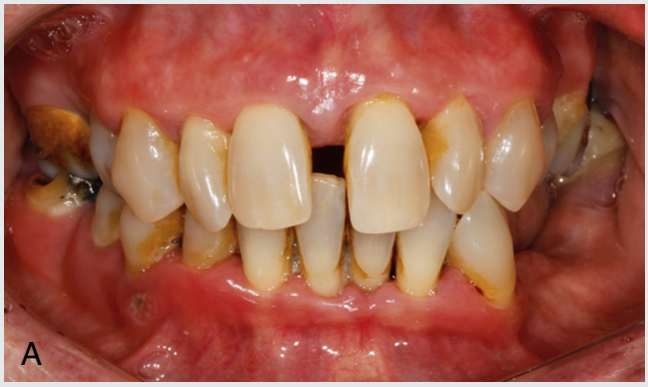
What factors may contribute to the pathogenicity of dental calculus?

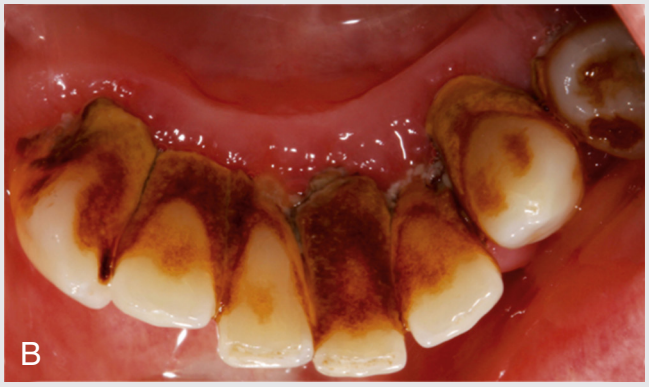
Dental calculus provides an ideal environment for bacterial biofilm growth and it is an important local predisposing factor in the development and progression of periodontitis. The pathogenicity of calculus may be attributed to the following factors:

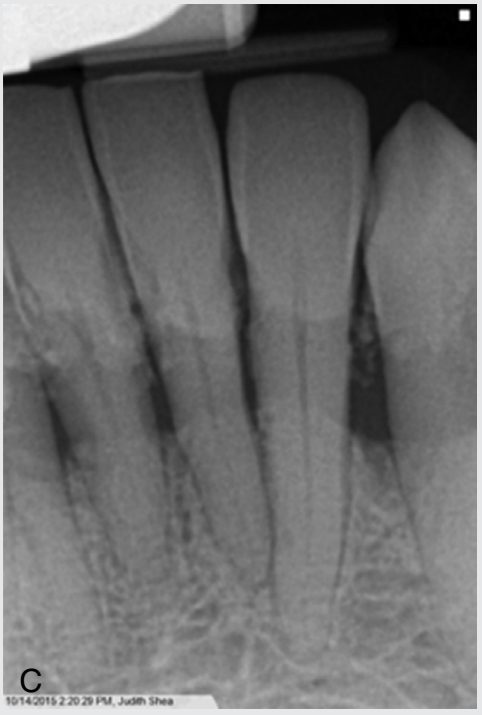
1. Plaque retentive action—*in vivo*, calculus always has a surface layer of plaque attached to it and hence contributes to periodontal disease development.
2. Mechanical obstruction to personal oral hygiene measures—calculus prevents adequate plaque removal.
3. Porous nature—calculus can absorb and act as a reservoir for antigenic material, toxins, and bone-resorbing factors of bacterial origin.[3]

CASE-BASED LEARNING EXERCISE

Scenario: A healthy 50-year-old female presented with the chief complaint "I need a lot of work—I haven't been at a dentist in a while." She had no known medical conditions, no allergies, and was taking no medications. Periodontal findings: gingiva appeared enlarged and erythematous (A), generalized calculus (B), bleeding on probing, localized mobility, and furcation involvement were noted. Fremitus was noticed on tooth #9. Radiographic findings: generalized horizontal bone loss with funneling and widening of the PDL (C).







Questions

1. All of the following characteristics are associated with supragingival calculus, except:
   - **a.** Consistency is hard, clay-like
   - **b.** Rate of recurrence is rapid
   - **c.** Mainly composed of hydroxyapatite and magnesium whitlockite inorganic phases
   - **d.** Microbial profile is dominated by filamentous bacteria
2. What type of microbial pathogens are found in subgingival calculus?
   - **a.** Cocci, rods, and filaments
   - **b.** Cocci and rods
   - **c.** Rods and filaments
3. Which of the following is not a plaque-retentive iatrogenic factor?
   - **a.** Overhanging margins
   - **b.** Supragingival margin placement
   - **c.** Marginal ridge discrepancy in restorations
   - **d.** Orthodontic brackets
4. Which of the following is a mode of attachment of calculus to the lingual surface of the tooth structure (Figure B)?
   - **a.** Inorganic pellicle
   - **b.** Mechanical interlocking
   - **c.** Open adaptation

This chapter was developed from Chapter 13 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: c

Explanation: All are characteristics of supragingival calculus, except option c. Supragingival calculus is predominantly composed of hydroxyapatite and octacalcium phosphate phases; magnesium whitlockite is present in the subgingival calculus. A direct comparison between supragingival and subgingival calculus is presented in Table 8.1.

2. Answer: a

Explanation: Various types of microbial pathogens are present in subgingival calculus, including cocci, rods, and filaments. The supragingival calculus is dominated by filaments.

3. Answer: b

Explanation: All the factors mentioned are considered plaque-retentive iatrogenic factors, except option b. Subgingival, not supragingival, margin placement is considered a plaque-retentive iatrogenic factor.

4. Answer: b

Explanation: Mechanical interlocking into surface irregularities (resorption lacunae) is one of the four modes of attachment of calculus to the tooth surface. The other modalities include organic pellicle, close adaptation, and bacterial penetration.

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1. Zander, H. A. (1953). The attachment of calculus to root surfaces. *Journal of Periodontology*, *24*(1), 16–19.
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3. Patters, M. R., Landesberg, R. L., Johansson, L. A., Trummel, C. L., & Robertson, P. B. (1982). Bacteroides gingivalis antigens and bone resorbing activity in root surface fractions of periodontally involved teeth. *Journal of Periodontal Research*, *17*(2), 122–130.

9

Influence of Systemic Conditions and Smoking on Periodontal Disease

Relevant Terminology

Terminology / Abbreviation	Explanation
advanced glycation end product (AGE)	Nonenzymatic glycosylation of proteins and matrix molecules results in the accumulation of AGEs.
agranulocytosis	• More severe form of neutropenia involving neutrophils, basophils, and eosinophils • Absolute neutrophil count < 100 cells/μl • Severe infections, ulcerative necrotizing lesions of oral mucosa, the skin, and gastrointestinal and genitourinary tracts
anemia	• Reduction in the number of erythrocytes and the amount of hemoglobin • Pernicious anemia results in red, smooth, shiny tongue as a result of atrophy of the papillae, and marked pallor of the gingiva • Iron deficiency anemia—similar to pernicious anemia • Sickle cell anemia—hereditary form of chronic hemolytic anemia with higher prevalence in African Americans. Characterized by pallor, jaundice, weakness, rheumatoid manifestations, leg ulcers, stepladder alignment of trabeculae • Aplastic anemia—caused by toxic drugs; increased susceptibility to infection due to the concomitant neutropenia
angular cheilitis	• Cause can be riboflavin deficiency • Associated with drooling of saliva or candidiasis
bisphosphonate-related osteonecrosis of jaw (BRONJ)	Exposure and necrosis of portions of the jawbone in patients exposed to bisphosphonates that has persisted for longer than 8 weeks with no history of radiation therapy to the jaws.
Bisphosphonates	Antiresorptive agents used to treat osteoporosis, Paget disease, bone metastasis, multiple myeloma, and other conditions involving fragile, breakable bone.
Chédiak–Higashi syndrome	• Autosomal recessive genetic disorder caused by a mutation of lysosomal trafficking regulator protein, which leads to a decrease in phagocytosis and delayed killing of microorganisms • Severe periodontal destruction is common
Down syndrome	Trisomy 21; characterized by intellectual disability and growth retardation. Poor neutrophil chemotaxis, phagocytosis, and intracellular killing are the likely underlying causes of periodontal destruction.
former smoker	Individual who has smoked ≥ 100 cigarettes in their lifetime and does not currently smoke.
hypothalamic-pituitary-adrenal axis (HPA axis)	• Central role in regulating homeostatic systems • Corticotropin-releasing factor plays a central role in the stress response by regulating HPA axis
leukemia	• Malignant neoplasia of white blood cell precursors • Can be classified into lymphocytic or myelogenous, acute, subacute, or chronic • Poor cellular defense and increased susceptibility to infections • Leukemic infiltration, bleeding, oral ulcerations, and infections are common oral manifestations

Relevant Terminology—cont'd

Terminology / Abbreviation	Explanation
leukocyte adhesion deficiency (LAD)	• Autosomal recessive genetic disorder characterized by immunodeficiency resulting in recurrent infections • Inability to produce or failure to normally express the leukocyte surface integrin (CD18) that is necessary for adhesion to the vessel wall, resulting in impaired defense against bacterial challenge
neutropenia	• Low levels of circulating neutrophils • Can be caused by infection or chemicals, or be idiopathic or hereditary • May be chronic or cyclic • Severe neutropenia—absolute neutrophil count (ANC) <500 cells/μl; severe periodontal destruction is common in this condition
nonsmoker	Individual who has *not* smoked ≥100 cigarettes in their lifetime and does not currently smoke.
osteoporosis and osteopenia	• Characterized by low bone mass and structural deterioration with increased risk of bone fracture • T-score is a comparison of the patient's bone mineral density with that of a healthy 30-year-old adult with peak bone mass, using standard deviation to define osteopenia and osteoporosis • Osteopenia: low bone mineral density (T-score of –1 to –2.5) • Osteoporosis: T-score ≤ –2.5
pack-years	• Pack-years = number of packs per day × number of years of smoking • Assesses the cumulative effect of smoking
Papillon–Lefèvre syndrome	• Autosomal recessive genetic disorder with no gender predilection, characterized by palmoplantar hyperkeratosis and severe destruction of periodontium • Decreased neutrophil function
RAGE	Receptor for AGE.
smoker	Individual who has smoked ≥100 cigarettes in their lifetime and currently smokes.
thrombocytopenic purpura	• Purplish appearance of the skin or mucous membranes where bleeding has occurred • Low platelet count, prolonged clot retraction and bleeding time, and normal or slightly prolonged clotting time • Spontaneous bleeding of skin or from the mucous membranes • Petechiae and hemorrhagic vesicles occur in the oral cavity • Removal of local irritating factors (plaque or calculus) alleviates gingival inflammation and bleeding
Type I diabetes	• Formerly known as insulin-dependent diabetes • 5%–10% of all diabetes cases • Caused by cell-mediated autoimmune destruction of β-cells in pancreas
Type II diabetes	• Formerly known as noninsulin-independent diabetes • 90%–95% of all diabetes cases • Caused by resistance to insulin action, impaired insulin secretion, and increased glucose production in the liver with no destruction of β-cells in the pancreas • Frequently associated with obesity
vitamin C deficiency	• Results in *scurvy*, characterized by hemorrhagic (bleeding) tendency and delayed wound healing • Common oral signs of scurvy: bleeding gums, swollen gingiva, and loosened teeth

Fast Facts

Smoking and periodontitis	• Smoking is a major risk factor for periodontitis • Smoking has effects on the prevalence, severity, etiology, and pathogenesis of periodontal disease, and a negative impact on treatment
Toxic chemicals of tobacco smoke	• Gaseous phase—carbon monoxide, ammonia, formaldehyde, hydrogen cyanide, and carcinogenic toxins • Particulate phase—tar • Nicotine—highly addictive, causes rise in blood pressure, increased heart and respiratory rates, and local vasoconstriction
Toxicity of tobacco smoke	Attributed to tar yield.
Dependence on smoking habit	• Attributed to nicotine (the most pharmacologically active compound in tobacco smoke) • Nicotine mimics the action of acetylcholine due to its close structural resemblance, causing peripheral vasoconstriction
Assessing smoking status	Exhaled carbon monoxide or measuring cotinine (the major metabolite of nicotine) in serum, saliva, or urine.
Effects of smoking	Reduced gingival inflammation and bleeding on probing (BOP), higher prevalence and severity of periodontal destruction, increased probing depth, attachment and tooth loss.
Results from NHANES III (Association of Periodontitis and Smoking)[1]	• Smokers were four times as likely to have periodontitis compared with nonsmokers • Former smokers have less risk for periodontitis than current smokers, but more risk than nonsmokers • The odds of having periodontitis in former smokers who quit smoking ≥11 years previously were statistically similar to those for nonsmokers
Smoking cessation program	• Brief intervention program comprising of five A's: Ask, Advise, Assess, Assist, and Arrange • Prevalence and severity of periodontitis decreases with smoking cessation
Methods of smoking cessation	• Willpower alone • Self-help materials • Brief intervention program in primary care • Nicotine replacement therapy • Varenicline—partial agonist of nicotine acetylcholine receptors • Bupropion—used at lower dose for smoking cessation; at higher dose, used as antidepressant • Other methods—meditation, counseling, cognitive behavioral therapy, hypnosis, acupuncture, etc
Effects of smoking on implant therapy	• The risk of implant failure is double the risk in nonsmokers • The risk is higher in maxillary implants placed in poor-quality bone
Effects of smoking on periodontal therapy (summary)	Smokers may: • Present with periodontal disease at an early age • Be difficult to treat effectively with conventional therapies • Continue to have progressive or recurrent periodontitis • Be at increased risk of tooth loss or periimplant bone loss, even when adequate maintenance control is established
Endocrine disorders and hormonal changes associated with periodontitis	Diabetes, metabolic syndrome, female sex hormone fluctuations, and hyperparathyroidism.
Influence of uncontrolled diabetes on periodontium	• Enlarged gingiva, sessile or pedunculated gingival polyps, polypoid gingival proliferations, abscess formation, periodontitis, and loosened teeth • Uncontrolled or poorly controlled diabetes is associated with increased susceptibility to and severity of infections, including periodontitis
Oral manifestation of uncontrolled diabetes	• Cheilosis • Mucosal drying and cracking • Burning mouth and tongue • Diminished salivary flow • Alteration in the flora (increase in *Candida albicans*, hemolytic streptococci, and staphylococci) • Increased caries rate

Fast Facts—cont'd

Epidemiologic study on diabetes and periodontitis in Pima Indians in AZ	• 40% of adult Pima Indians in AZ have Type 2 diabetes • The prevalence of destructive periodontitis and edentulism significantly increased among individuals with diabetes compared with nondiabetic individuals • The risk of developing diabetes was three times more in diabetic individuals than in nondiabetic individuals

Metabolic syndrome	• Describes a condition of abdominal obesity combined with two or more of the following:
	  • hypertension
	  • dyslipidemia
	  • hyperglycemia
	• It increases the risk for developing Type 2 diabetes and cardiovascular disease

Metabolic syndrome and periodontitis	• Obesity, obesity-related characteristics, and metabolic syndrome may be risk indicators for the severity and progression of periodontitis • Obesity is associated with increased proinflammatory cytokines (e.g., IL-6 and TNF-α) produced by macrophages in adipose tissue, and T-cell and monocyte/macrophage dysfunction
Effects of puberty and menstruation	• Hormonal changes during puberty and the menstrual cycle cause exacerbation of preexisting gingivitis with pronounced inflammation, edema, and gingival enlargement • Can be prevented or minimized with good oral hygiene
Pregnancy and gingivitis	• Pregnancy itself does not cause gingivitis • Pregnancy tumor—a nonspecific, vascularizing, and proliferative inflammation of the gingiva that occurs in some pregnant women • *Prevotella intermedia* significantly increases during pregnancy, associated with the peak levels of estradiol and progesterone • Greatest severity seen in the second and third trimesters • Can be prevented or minimized with good oral hygiene
Bacterial-hormonal interactions	*P. intermedia* significantly increases during pregnancy and is associated with elevated estrogen and progesterone levels and gingival bleeding.
Effect of menopause	• Hormonal fluctuation could cause menopausal gingivostomatitis • Dry, burning sensation with extreme sensitivity to thermal changes
Hyperparathyroidism	• Generalized demineralization of skeleton, increased osteoclasts with proliferation of connective tissue in the marrow space, forming bone cysts and giant cell tumor (osteitis fibrosa cystica or von Recklinghausen bone disease) • Brown tumor—bone cysts filled with fibrous tissue with abundant hemosiderin-laden macrophages and giant cells • Malocclusion, tooth mobility, radiographic evidence of osteoporosis, widened periodontal ligament, absence of lamina dura, radiolucent cyst-like spaces
Blood disorders associated with periodontal disease	Neutropenia, agranulocytosis, leukemia, anemia, thrombocytopenia, antibody deficiency disorder.
Genetic disorders associated with periodontal destruction	Chédiak–Higashi Syndrome, lazy leukocyte syndrome, leukocyte adhesion deficiency, Papillon–Lefèvre Syndrome, Down syndrome.
Stress and periodontal diseases	• Stress alone does not cause periodontal disease • The manner in which an individual copes with the stress affects periodontal destruction in the presence of periodontal pathogens • Patients with periodontal disease are less likely to use active coping skills (i.e., situation control) and more likely to cope with stress by averting blame (emotional) than periodontally healthy patients
Stress-induced immunosuppression	• Increased cortisol production in response to stress reduces the immune response through the suppression of neutrophil activity, IgG production, and salivary IgA secretion • Stress-induced neurotransmitters (epinephrine, norepinephrine, neurokinin, and substance P) can cause tissue destruction by activating lymphocytes, neutrophils, monocytes, and macrophages

Continued

Fast Facts—cont'd

Influence of stress on periodontal therapy outcomes	Stress impairs the inflammatory response and matrix degradation, resulting in more painful, poorer, and slower recovery.
Nutritional influences	• Riboflavin deficiency—glossitis, angular cheilitis, seborrheic dermatitis, and superficial vascularizing keratitis • Thiamine deficiency—edema, loss of appetite, and hypersensitivity and erosion of the oral mucosa • Niacin deficiency—pellagra (dermatitis, gastrointestinal disturbances, and neurologic/mental disturbances) • Vitamin C (ascorbic acid) deficiency—scurvy
Vitamin C (ascorbic acid) deficiency and periodontal disease	• Influences the metabolism of collagen within the periodontium • Interferes with bone formation • Increases the permeability of the oral mucosa • Ascorbic acid deficiency alone does not cause periodontitis (local bacterial factors required)
Characteristics of bisphosphonates	• High affinity for hydroxyapatite • Inhibit bone metabolism through inhibition of osteoclasts • Can also impair soft tissue wound healing by inhibiting epithelial migration and wound closure • The half-life of bisphosphonates entrapped in the bone is estimated to be 10 years or longer
Staging of BRONJ	• Stage 0— at-risk patients who have been treated with IV or oral bisphosphonates but who have no apparent exposed or necrotic bone • Stage 1—exposed or necrotic bone in patients who are asymptomatic with no infection • Stage 2—exposed or necrotic bone with pain and clinical evidence of infection • Stage 3—in addition to Stage 2 signs, pathologic fracture, extraoral fistula, or osteolysis that extends to the inferior border Medication-related osteonecrosis of the jaw (MRONJ) is a newer term for BRONJ.
Risks of BRONJ	• BRONJ occurs spontaneously or after a traumatic event such as a dental procedure (e.g., extractions, root canal treatment, periodontal infections, periodontal surgery, and dental implant surgery) • Patients who are treated for cancer with IV bisphosphonates are at greater risk than patients treated for osteoporosis with oral bisphosphonates
Effects of corticosteroids on periodontium	• Exogenous cortisone may have an adverse effect on bone quality and physiology • Stress-induced endogenous cortisol level may have an adverse effect on periodontium by diminishing the immune response to periodontal bacteria
Correlation between osteoporosis and periodontitis	Studies are cross-sectional in nature; association between osteoporosis and periodontitis is only suggestive.
congenital heart disease	• Delayed eruption • Positional abnormalities • Enamel hypoplasia • Blue-white appearance of teeth with large pulp chamber • Congenital heart disease alone does not increase the risk of periodontal diseases • At risk for infective endocarditis; prophylactic antibiotics may be needed prior to dental procedure
Other systemic conditions associated with periodontal destruction	• Osteopenia and osteoporosis—suggestive association • Hypophosphatasia—teeth loss without clinical evidence of gingival inflammation, disease resembles a condition formerly called localized aggressive periodontitis

Core Knowledge

Introduction

The periodontium that is prone to inflammatory responses is greatly influenced by systemic factors that alter the host response. Some examples include:

Systemic factors affecting periodontal tissues

- Effect on collagen:
  - Scurvy—associated with defective collagen formation and maturation
  - Down syndrome—associated with abnormal patterns of collagen synthesis
  - Medications—associated with collagen overproduction and (possible) slow breakdown
- Effect on epithelial and endothelial permeability:
  - Pregnancy—associated with magnified inflammatory response due to increased vascular and tissue permeability

Systemic factors affecting host response

- Phagocyte dysfunction is associated with Down syndrome, diabetes mellitus, and smoking

Systemic factors affecting routine oral hygiene practice

- Down syndrome, depression, and anxiety

This chapter reviews the influence of smoking and other key systemic conditions on periodontal disease and treatment outcomes.

Smoking and Periodontal Disease

While the initial belief was that smokers' poor oral hygiene contributed to periodontal disease, it is now known that smoking causes immunosuppression. The periodontal consequences of smoking include:

- Decreased bleeding on probing (BOP) and underdiagnosed inflammation
- Increased attachment loss, probing depth (PD), and bone loss
- Poor response to periodontal treatment.

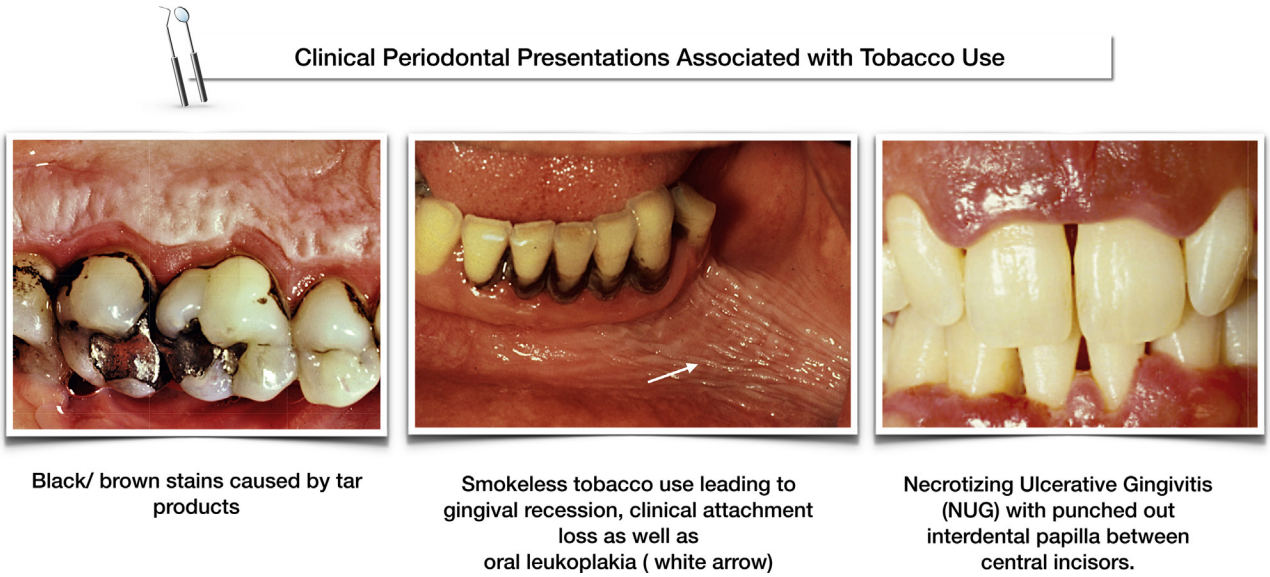
See Fig. 9.1 for clinical scenarios associated with tobacco use.

CLINICAL CORRELATE

Is nicotine carcinogenic? What is the current status of clinical research on the effects of nicotine on cells and tissues?

Nicotine is not carcinogenic, and researchers now largely accept that it has likely been blamed unfairly. The potential role of nicotine in periodontitis development is unclear. In vitro studies using nicotine often present conflicting results with regard to cellular viability and functions. Clinical studies are required to determine the clinical relevance of these in vitro results.

The effects of tobacco smoking on periodontal diseases are more significant than those of any other systemic factor, including diabetes (Table 9.1).



• **Fig. 9.1** Clinical Periodontal Conditions Associated With Tobacco Use. (Images are from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

TABLE 9.1 Effects of Smoking on Etiopathogenesis of Periodontal Disease and on Response to Periodontal Therapy

Effects of Smoking on Etiopathogenesis of Periodontal Disease	
Microbial components affected	Highly diverse, pathogen-rich, commensal-poor, anaerobic microbiome (orange and red complex periodontal pathogens) seen; more similar to the microbiome observed in patients with advanced periodontitis than that of periodontally healthy nonsmokers.
Host components affected	• Altered neutrophil chemotaxis, phagocytosis, and oxidative burst • ↓ levels of antibody against periodontal pathogens (specifically IgG2), essential for phagocytosis and killing of bacteria • ↑ release of tissue-destructive enzymes and chemical mediators (e.g., MMP-8, PGE_2)
Tissue components affected	Microcirculation altered (e.g., ↓ blood vessels).
Physiological aspects affected	• ↓ subgingival temperature • ↓ GCF and BOP in the presence of plaque-induced inflammation • ↑ time needed to recover from local anesthesia
Effects of Smoking on Response to Periodontal Therapy	
Nonsurgical therapy	• ↓ Reduction in PD • ↓ Gain in clinical attachment levels
Surgical therapy and implants	• ↓ PD reduction and ↓ gain in clinical attachment levels after access flap surgery • ↑ deterioration of furcations after surgery • ↓ gain in clinical attachment levels, ↓ bone fill, ↑ recession, and ↑ membrane exposure after guided tissue regeneration • ↓ root coverage after grafting procedures for localized gingival recession • ↓ PD reduction after bone graft procedures • ↑ risk for implant failure and periimplantitis
Maintenance therapy	• ↑ PD and attachment loss during maintenance therapy • ↑ disease recurrence in smokers • ↑ need for retreatment in smokers • ↑ tooth loss in smokers after surgical therapy

↑, increased; ↓, decreased; BOP, bleeding on probing; GCF, gingival crevicular fluid; MMP, matrix metalloproteinase; PD, probing depth; PGE_2, prostaglandin E2. Adapted from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). Newman and Carranza's Clinical Periodontology (13th ed.). Philadelphia: Elsevier. (Tables 12.3 and 12.4).

CLINICAL CORRELATE

What are the likely benefits of smoking cessation on the periodontium?

The benefits of smoking cessation on the periodontium are likely to be a shift toward a less pathogenic microbiome, the recovery of the gingival microcirculation, and improvements in certain aspects of the immune-inflammatory response.

Influence of Systemic Conditions on Periodontal Disease

It is important to recognize that the systemic diseases, disorders, or conditions themselves do not cause periodontitis; instead, they predispose, accelerate, or otherwise increase disease progression. These conditions include:

- **Endocrine disorders and hormonal changes**—diabetes mellitus, metabolic syndrome, female sex hormones, hyperparathyroidism
- **Hematologic disorders and immune deficiencies**—leukocyte (neutrophil) disorders, leukemia, anemia, thrombocytopenia, antibody deficiency disorders
- **Genetic disorders**—Chédiak–Higashi syndrome, lazy leukocyte syndrome, leukocyte adhesion deficiency (LAD), Papillon–Lefèvre syndrome, Down syndrome
- **Stress and psychosomatic disorders**—psychosocial stress, depression, and poor coping; stress-induced immunosuppression
- **Nutritional influences**—vitamin deficiencies, protein deficiency
- **Medications**—bisphosphonates, corticosteroids

Diabetes Mellitus and Periodontal Disease

Periodontal disease in individuals with diabetes follows no distinct pattern of clinical presentation. Oral effects seen (though not pathognomonic) include:

- Xerostomia (dry mouth)
- Burning mouth or tongue
- Opportunistic infections (e.g., *Candida albicans*)
- Cheilosis (cracking at the corner of the mouth);
- Increased rate of dental caries formation

A patient with well-controlled diabetes is at no greater risk for periodontal destruction than a normal individual without diabetes. However, a poorly controlled diabetic—especially when presenting with other systemic complications like retinopathy or nephropathy—appears to be more at risk for periodontal disease than those without diabetes or with well-controlled diabetes. See Fig. 9.2 for clinical conditions of the gingiva and periodontal tissues commonly associated with diabetes mellitus.

Proposed Mechanisms for Diabetes Affecting Periodontal Health

Diabetes is thought to affect the periodontal status through (Fig. 9.3):

- Direct effects of hyperglycemia—for example, hyperglycemic status leads to changes in plaque microflora
- Indirect modulation by advanced glycation end products (AGEs)—leading to an overall impairment of immune responses and wound healing (e.g., altered collagen metabolism), and changes in periodontal tissues (microangiopathy)

CLINICAL CORRELATE

What are the periodontal consequences for diabetic patients when AGEs interact with macrophages and other host cells?

AGEs initiate and amplify inflammation when they bind to their cellular receptors, RAGEs (cell surface receptors found on monocytes/macrophages, endothelial and epithelial cells). The binding of AGEs to RAGEs activates the nuclear factor NF-κB-regulated pathway, resulting in upregulation of proinflammatory cytokines. Hence AGE-RAGE interactions convert *transient* inflammatory responses to *sustained and dysfunctional* immune responses.

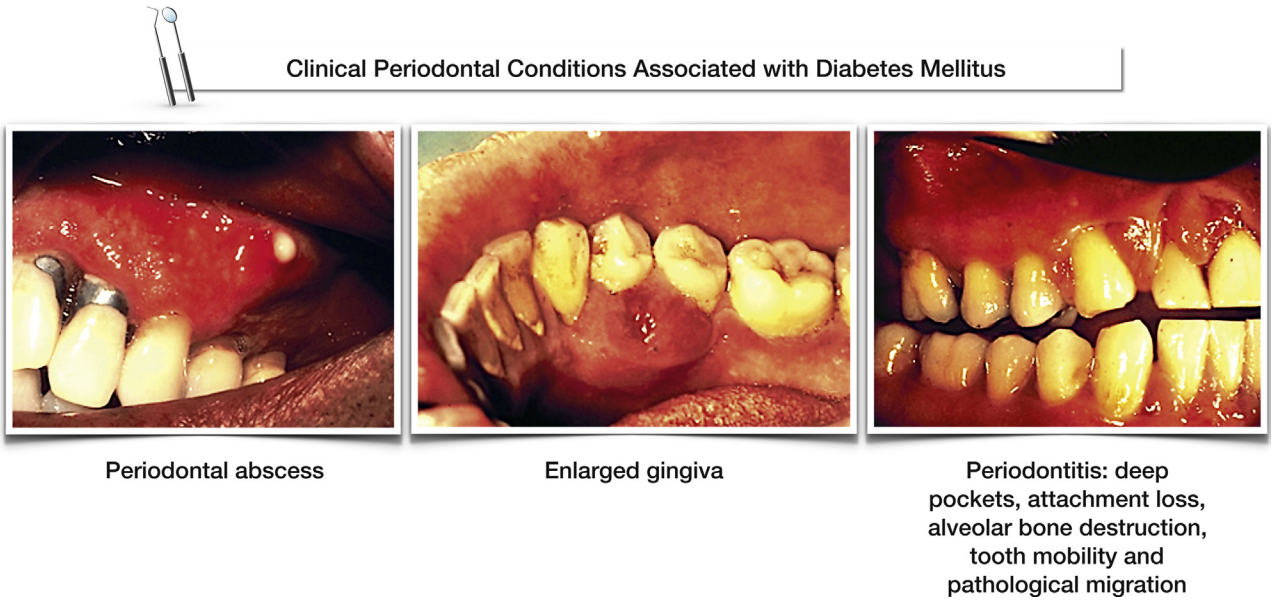
Stress and Periodontal Disease

Currently classified as a "risk indicator" for periodontal disease, "stress" has both a psychological and a physiological component. The word "stress" is used to describe adverse emotions or unpleasant experiences that may provoke such reactions. The word "stressor" denotes any stimulus, situation, or circumstance with the potential to induce stress reactions.[6]

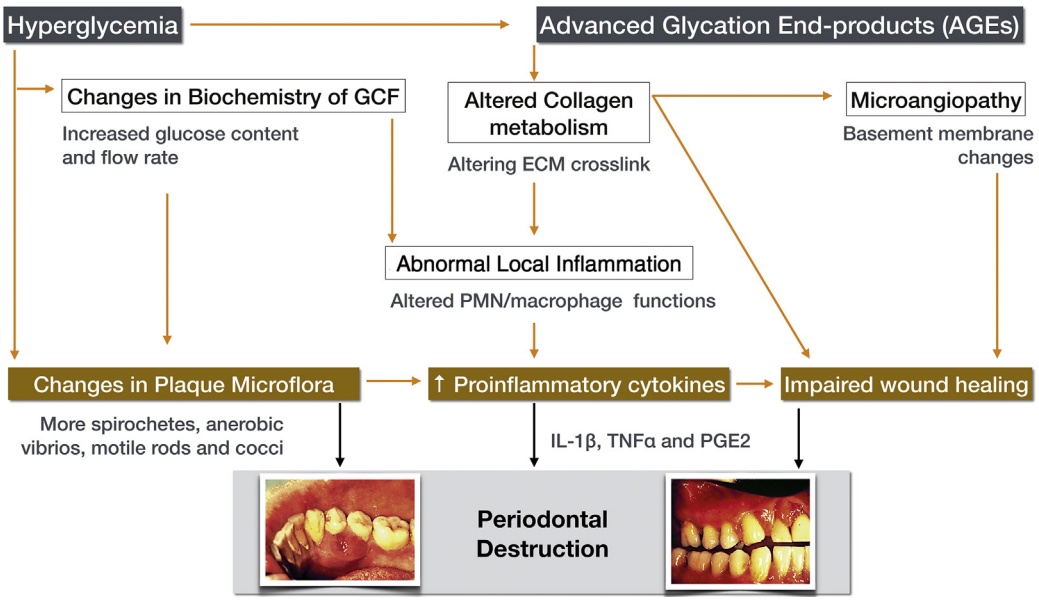
Stress can cause deregulation of the immune system mediated mainly via:

- hypothalamic–pituitary–adrenal cortex (HPA) axis
- sympathetic–adrenal medullary axis

See Fig. 9.4 for the proposed effects of stress on periodontal disease.



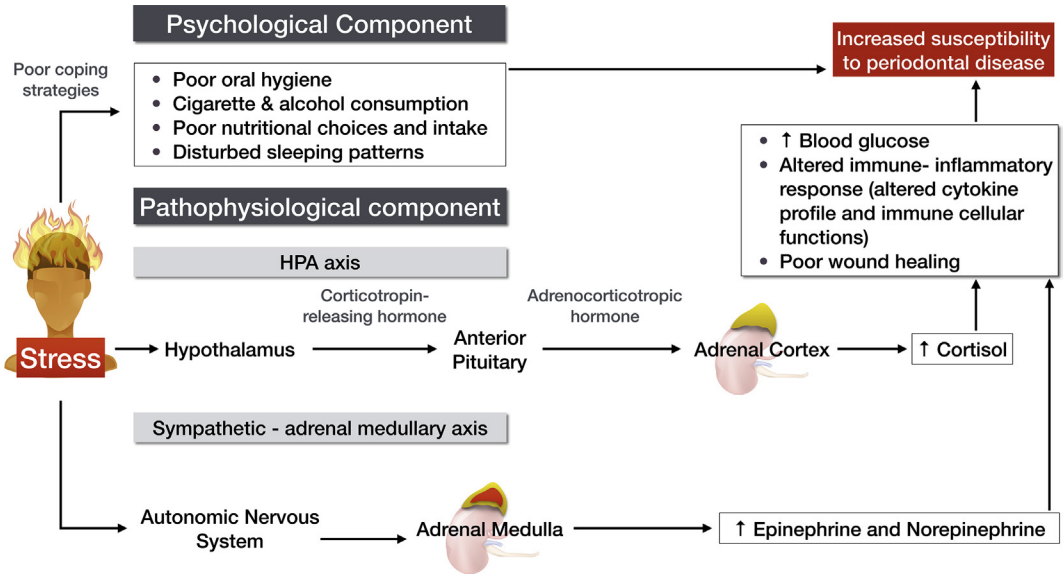
• **Fig. 9.2** Clinical Periodontal Conditions Associated With Diabetes Mellitus.[2,3] Periodontal disease has been proposed as the sixth complication of diabetes, based on the highly frequent presence of both diseases in the same patient. Severe gingival inflammation, deep periodontal pockets, rapid bone loss, and frequent periodontal abscesses often occur in patients with poorly controlled diabetes and poor oral hygiene. (Clinical photo is from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)



• **Fig. 9.3** Proposed Mechanisms of Diabetes-Mediated Periodontal Destruction.[4,5] Interplay of several factors may cause periodontal destruction in diabetics. Hyperglycemia and accumulated glycation end products (AGEs) have major roles to play in causing three major repercussions (seen in brown boxes): (1) changes in plaque microflora; (2) increase in proinflammatory cytokines that cause tissue destruction; and (3) impaired wound healing. These factors have direct consequences in periodontal disease development; the proposed pathway leading to direct periodontal destruction is shown by black arrows. Other factors (e.g., changes in GCF biochemistry, altered collagen metabolism, microangiopathy) that are a result of hyperglycemia and AGE accumulation can indirectly cause periodontal destruction by the aforementioned direct pathways. The indirect effects contributing to periodontal destruction are shown by the orange arrows. The proposed destructive mechanisms may be understood in the following simplified sequence (though considerable overlap and interplay occurs): **Hyperglycemia:** In the hyperglycemic state, numerous proteins and matrix molecules undergo nonenzymatic glycosylation, resulting in excessive accumulation of AGEs. A rise in the glucose concentration also contributes to alterations in the microbial profile in periodontal pockets.

1. **Changes in biochemistry of GCF**—increased glucose concentration and GCF flow rate correlate with increase in gingival inflammation.
2. **Changes in plaque microflora**—having more glucose in the pocket microenvironment favors the growth of periodontal pathogens like *Capnocytophaga* spp., *Aggregatibacter actinomycetemcomitans, Prevotella intermedia* and *Porphyromonas gingivalis*.
3. **AGE accumulation**—alters inflammatory response and impairs wound healing. Leads to:
   a. **Altered collagen metabolism**—collagen is cross-linked by AGE formation, making it less soluble and less likely to be normally repaired or replaced. Cellular migration through cross-linked collagen is also impeded.
   b. **Microangiopathy (vascular changes)** —impaired metabolism of type IV collagen (the main component of basement membranes) causes disruption of the basement membrane which can interfere with oxygen diffusion and PMN chemotaxis.
4. **Abnormal local inflammation**—in patients with poorly controlled diabetes, the functions of PMNs, monocytes, and macrophages are impaired. Leads to:
   a. **Altered PMN functions**—impaired chemotaxis, defective phagocytosis, decreased intracellular killing, or impaired adherence.
   b. **Hyperresponsive macrophages**—AGE-RAGE interactions cause macrophages to switch to a destructive phenotype instead of an inflammation-resolving phenotype.
   c. **Proinflammatory cytokine release**—AGE-stimulated macrophages and PMN cells show a hyperresponse to the progression of bacterial biofilm, releasing more cytokines and soluble mediators that produce destruction of connective tissue. Cytokine dysfunction in diabetes plays a bigger role than microbial changes in periodontal destruction.

AGE, advanced glycation end product; *ECM*, extracellular matrix; *GCF*, gingival crevicular fluid; *IL-1β*, interleukin-1 beta; PGE_2, prostaglandin E2; *PMN*, polymorphonuclear leukocytes or neutrophils; RAGE, receptor for AGE; *TNFα*, tumor necrosis factor alpha. (Clinical photo is from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)



• **Fig. 9.4** Stress Response and Periodontal Implications.[6] Stressful stimuli (negative life events, systemic illness, depression, daily hassles, etc.) may induce reactions affecting virtually all body systems. In individuals with poor coping mechanisms, these effects are mediated by:

(a) **Stress-induced health-impairing behaviors—**stress-induced behaviors like disturbed sleep patterns affect the secretion of growth hormone, downregulating tissue repair. When this is accompanied by poor nutritional choices, alcohol abuse, and smoking, there is further impairment of wound healing processes.

(b) **Pathophysiological reactions via the hypothalamic–pituitary–adrenal cortex axis and sympathetic–adrenal medullary axis—**pathophysiological mechanisms causing increased levels of cortisol and epinephrine disrupt homeostasis and increase susceptibility to periodontal disease by a wide range of mechanisms: altered inflammatory cytokine profiles, impaired immunity, increased blood glucose levels, and poor wound healing.

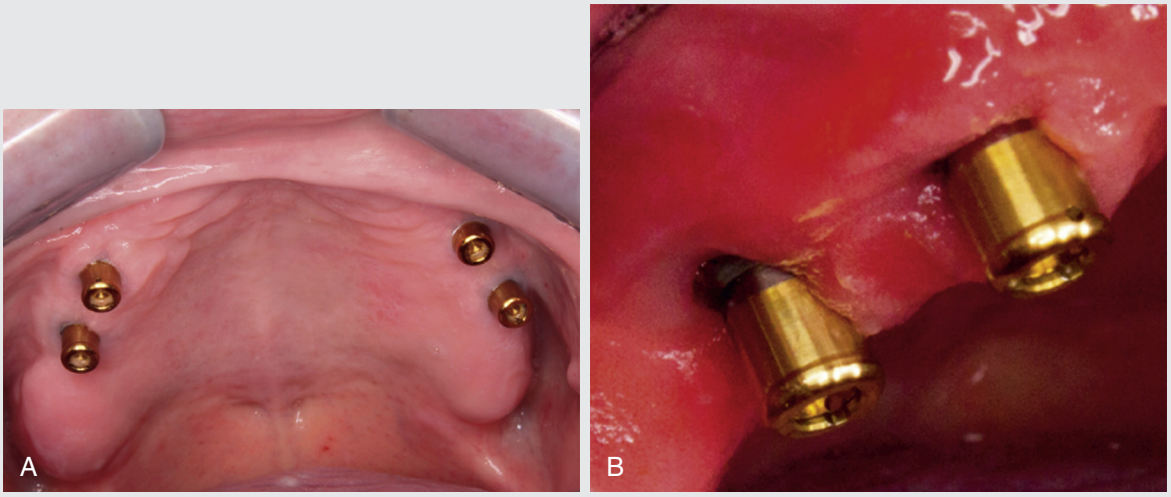
HPA, hypothalamic–pituitary–adrenal cortex axis.

Conclusion

It is now well understood that the medical status of an individual can significantly influence their risk of periodontal disease development as well as their chance of healing after appropriate therapy. For example, smoking can compromise periodontal prognosis following therapy to a large extent. It is vital for the clinician to have up-to-date awareness and understanding regarding the various interactions of key systemic conditions with periodontal health and disease states, so that these factors can be accounted for in periodontal treatment planning and recall for maintenance.

CASE-BASED LEARNING EXERCISE

Scenario: A 62-year-old male presented with the chief complaint, "I have tenderness around dental implants that were placed 1 year ago, and I notice bleeding when I'm brushing them." The patient had a history of hypertension and hypothyroidism. He reported smoking 10–15 hand-rolled cigarettes per day, and had smoked on and off for over 40 years. He managed to quit for a period around the time of implant placement, but recently resumed smoking. The patient was seeing his dentist regularly, had been edentulous for 10 years, and had struggled to wear a complete maxillary denture prior to receiving implants. He reported using a powered toothbrush twice per day to brush around dental implants. Current findings (1 year after implant placement): Generalized probing depths of 5–6 mm around all the implants. BOP score 80%. Evidence of radiographic bone loss 2–4 mm from baseline records. Generally poor oral hygiene.



Clinical images are from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

Questions

1. Smokers who receive dental implants are at increased risk for all of the following EXCEPT:
- **a.** Failure of osseointegration.
- **b.** Peri-implantitis.
- **c.** Wound healing complications.
- **d.** Increased bleeding on probing.

2. If this patient had been a current smoker at the time of dental implant placement, would he have been at increased risk of suffering from a dental implant failure?
- **a.** No increased risk
- **b.** 1.2 times the risk
- **c.** 2 times the risk
- **d.** 5 times the risk

3. If a patient successfully quits smoking, how long should you wait until placing dental implants?
- **a.** 1 week
- **b.** 2 months
- **c.** 2 years
- **d.** Yet to be determined

4. What advice should you give e-cigarette users (former smokers) who are considering dental implant treatment?
- **a.** Stop e-cigarettes immediately.
- **b.** Consider cutting down.
- **c.** Use nicotine-free e-liquids.
- **d.** Change the e-liquid flavor.

This chapter was developed from Chapters 12 and 14 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: d

Explanation: Smoking has myriad effects that will influence the prognosis of a dental implant in both the short and the long term. Smoking has the same effects on the tissues surrounding implants—including its negative effects on the vasculature, immune and inflammatory responses, and microbiology—as on the periodontium (see **Table 9.1**).

2. Answer: c

Explanation: There have been numerous systematic reviews on this topic. Overall, they find that smokers are at approximately double the risk of implant failure compared with nonsmokers. Patients should be made aware of this increased risk and every effort made to support them in their efforts to quit smoking.

3. Answer: d

Explanation: Some early protocols suggested that abstinence for 1 week before and 8 weeks after dental implant placement resulted in significantly improved early outcomes compared with patients who continued to smoke. More contemporary studies have suggested a 2-year cutoff is more useful, with those having dental implant placement earlier than 2 years from the time of cessation having a 2.7 times greater risk of implant failure than those who had been abstinent for longer than 2 years. Therefore a clear timeline has not yet been established.

4. Answer: b

Explanation: There is a growing body of evidence to suggest that e-cigarettes have a negative impact on implant outcomes. Given this early evidence, e-cigarette users considering dental implants should be encouraged to cut down the frequency of use. However, it is important to consider not pressuring the patient to quit immediately, for fear of relapse to regular cigarette smoking.

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10 Genetics of Periodontal Disease: Risk and Treatment

Relevant Terminology

Terminology	Explanation
allele	• Variant form of a gene • Humans have 2 alleles in each genetic locus
autosomal dominant	DNA variation in a gene located on an *autosome* that has a dominant effect over other forms of variation at this location within the gene.
autosomal recessive	DNA variation in a gene located on an *autosome* that has an effect on the gene's function only when the person has inherited both maternal and paternal copies of the same allele.
autosome	Any chromosome that is not a sex chromosome.
candidate gene analysis	• A gene-mapping approach that tests whether a particular allele of a gene occurs more often in patients with the disease than in subjects without the disease • Candidate genes are chosen on the basis of their known or presumed function
case-control study	• Study in which the genetic makeup is compared between patients who have the disease and controls who do not • Confounding factors need to be matched
chédiak–Higashi syndrome	• Autosomal recessive disorder caused by a mutation in a lysosomal trafficking regulator gene • Characterized by decreased phagocytosis in immune cells, making affected individuals significantly prone to infections
chromosome	• A nuclear structure that contains genetic information • Human cells have 22 pairs of autosomes and one pair of sex chromosomes (XX or XY), a total of 46 chromosomes
DNA	• Deoxyribonucleic acid • Forms the hereditary material of all cells
Ehlers–Danlos syndrome	• Autosomal dominant disorder that affects connective tissues supporting the skin, bones, blood vessels, and many other organs and tissues • Joint laxity and skin hyperextensibility, scarring, and bruising are common clinical features • Clinical features in the oral cavity include gingivitis, followed by early onset periodontitis, leading to attachment and tooth loss
epigenetics	Changes in phenotype or gene expression that result from mechanisms other than changes in the underlying DNA sequence.
exon	Protein-coding region of a gene.
frameshift mutation	A mutation that results from the insertion or deletion of one or more nucleotides into a gene, thereby causing the coding regions to be read in the wrong frame and usually causing the protein produced to be defective in function.
gene	• The basic unit of heredity that occupies a specific position (*locus*) on a chromosome and that has one or more specific effects on the *phenotype* of the organism • Consists of enough DNA to code for one protein • Comprises *introns* and *exons*

Relevant Terminology—cont'd

Terminology	Explanation
genetic epidemiology	The study of the role of genetics in the population distribution of a disease and the way in which such genetic factors interact with the environment to underlie individual differences in disease susceptibility.
genome	• The complete set of genes or genetic material present in a cell or organism • The entirety of the organism's DNA
genome-wide association study (GWAS)	• An approach that involves scanning markers across the complete *genomes* of many people to find genetic variations associated with a particular disease • Does not depend on any prior hypothesis about the disease's molecular pathology
genotype	The genetic makeup of an organism or cell, which is distinct from its clinically expressed features or *phenotype*.
haplotype	• A contraction of the term haploid genotype • Refers to a combination of *alleles* at multiple loci which are usually transmitted together on the same region of a chromosome
heterozygous	Having two different *alleles* at a specific position in a gene.
homozygous	Having identical *alleles* at a specific position in a gene.
intron	Noncoding region of a gene, removed from the final coding sequence during RNA splicing.
isoform	• Any of several different forms of the same protein • Can be produced from related genes, or same gene via alternative splicing. Isoforms are frequently caused by single nucleotide polymorphisms
ligand	A molecule that binds to a receptor.
linkage	The tendency for certain genes to be transmitted together from parent to child because they are located close to each other on the same chromosome.
linkage analysis	• A technique used to map a gene responsible for a trait to a specific location on a chromosome • Because many complex diseases are caused by multiple genes of "minor" effect, this method is of limited power for gene identification
linkage disequilibrium	The occurrence of specific alleles at different locations in the DNA that occur relatively close to each other (i.e., are linked) more often than would be expected by chance alone (disequilibrium).
locus (plural: loci)	The physical location that a gene occupies within a chromosome.
mutation	Changes in the DNA sequence of the genome.
next-generation DNA sequencing (high-throughput sequencing)	• Technique allowing sequencing of the entire human genome • Can be used to identify less common genetic variants, predicted to have individual gene effects on disease risk, that cannot be found by either GWAS or linkage analysis
nucleotides (bases)	• Molecules that make up the structural units of RNA and DNA • Composed of a phosphate group; adenine, cytosine, guanine, or thymine; and a pentose sugar • Often referred to as A, C, G, and T • Thymine base is replaced by uracil in RNA
Papillon–Lèfevre syndrome	• Autosomal recessive disorder caused by a mutation in the cathepsin C gene which results in neutrophil dysfunction • Characterized by palmoplantar keratosis and advanced periodontitis affecting deciduous and permanent dentitions, leading to premature tooth loss • Successfully treated with dental implant–supported restorations
penetrance	The proportion of individuals with a particular allele/genotype who express an associated trait (*phenotype*).
phenotype	• The clinically observable characteristics displayed by an organism • Influenced by gene expression and environmental factors
precision dentistry	• Individualized or personalized treatment optimized for the genomic characteristics of each patient • Trial-and-error (nonprecision) approach could cause unnecessary expense and does not provide an optimal quality of care
segregation analysis	• The process of fitting formal genetic models to expressed disease characteristics (*phenotype*) in biologic family members to determine the most likely mode of inheritance for the trait or disease • Useful method for straightforward traits in which mutation in a single gene causes the disease to develop with nearly 100% certainty in carriers (100% *penetrance*)

Continued

Relevant Terminology—cont'd

Terminology	Explanation
sequencing	Determining the linear alignment of *nucleotides* (in RNA or DNA) or amino acids (in proteins).
single nucleotide polymorphism (SNP)	A variation in a gene caused by a change in a single *nucleotide*.
splicing	The removal of *introns* from transcribed RNA.
transcription	RNA synthesis that occurs in the nucleus.
translation	• The first stage of protein synthesis • Occurs in the cytoplasm

Fast Facts

Examples of genetic adaptation to environment	• The sickle-cell hemoglobin variant that protects an individual against the infectious disease malaria • Ability to digest the milk sugar lactose as an adult that evolved in Europeans in conjunction with the domestication of dairy cattle
Techniques for studying the genetics of periodontal disease	• Candidate gene approach • Case-control studies • Twin studies • Familial aggregation and relative risk • Segregation analysis • Linkage analysis • Genome-wide association studies
Syndromic forms of periodontitis	• Chédiak–Higashi syndrome • Ehlers–Danlos syndrome • Papillon–Lefèvre syndrome
Nonsyndromic forms of periodontitis	• Periodontitis (Stage, Grade) with molar incisor involvement (previously called 'aggressive periodontitis') • Strong association of a SNP in the glycosyltransferase gene (*GLT6D1*) with aggressive periodontitis has been shown in one genome-wide association study[1]
Candidate genes possibly related to risk of chronic or aggressive periodontitis	• Interleukin (IL)-1 gene cluster, IL-4, IL-6, and IL-10 • TNFα • Leukocyte receptors for the constant (Fc) part of immunoglobulin (FcγR) • Vitamin D receptor • Pattern-recognition receptor genes (Toll-like receptors, CD-14) • Matrix metalloproteinase (MMP)-1
Genetic test for periodontitis risk prediction	• IL-1 genetic test is commercially available • Limitations exist: studies have shown that of three risk factors—smoking, diabetes, and IL-1 polymorphism—only smoking and diabetes showed significant effects on risks for tooth loss

Core Knowledge

Introduction

Periodontitis is a multifactorial disease. Of the many contributory factors (local, systemic, genetic, and environmental), it is difficult to distinguish the precise influence of genetic factors on the development of periodontal disease. A general trend noted is that older patients with periodontitis are likely to have local, environmental, and lifestyle factors contributing more to disease development, whereas in children and younger individuals, the contribution of genetic factors is greater.[2]

The Genetic Basis of Periodontal Disease

The human genome is estimated to have 20000-25000 genes and the different forms of a particular gene (genetic variants) are called *alleles*. A simplified equation to understand the contribution of a genetic variant to a complex disease like periodontitis would be:[3]

Phenotype = Genotype + Environment + Biologic Interactions

- "Phenotype" is a clinical manifestation—in this case, presence/absence of periodontal disease.

- "Genotype" is the individual's genetic makeup (includes allelic variants and mutations suspected of contributing to periodontal disease).
- "Environment" includes the presence of detrimental lifestyle factors (e.g., smoking or poor oral hygiene).
- "Biologic interactions" includes gene-environment interactions and epigenetic modifications.

Understanding how individual genes may contribute to genetic diseases in general, is vital for appreciating the specific contributions of genetic variants/alleles to periodontal disease development. Genetic diseases are traditionally divided into two broad groups based on the pattern of disease transmission: "simple Mendelian diseases" are caused by single gene mutations, and "complex" diseases are caused by genetic polymorphisms. Therefore the two types of genetic influences that may contribute to the development of periodontal disease are:

- **Single-gene mutations** —here the genetic defect is powerful enough to elicit disease by itself. For example, Papillon–Lefèvre syndrome is caused by mutation of the cathepsin C receptor gene and results in periodontitis.
- **Single nucleotide polymorphisms (SNP)** —when the genetic defect itself does not lead to manifestations of disease, but increases the susceptibility to the disease in the course of time, when many other factors also contribute to disease risk, it is called a genetic polymorphism. A SNP (pronounced "snip") is a genetic polymorphism caused by a change in a single nucleotide in the DNA sequence. Initially this may arise as a rare mutation, but is considered a SNP once it occurs in at least 1% of the population (e.g., IL-1 gene polymorphism).

CLINICAL CORRELATE

What are the effects of gene mutations and gene polymorphisms on proteins implicated in the development of periodontal disease?

Periodontitis is a multifactorial disease in which genetic factors play a role, the exact extent of which is yet unclear. Genes are stretches of DNA on chromosomes, with an order (a start and an end); with the aid of enzyme and other molecules, they direct protein production. Proteins are vital for normal body functioning and formation of healthy structures, including teeth and periodontium. Proteins may also carry signals between cells, be components of the immune system, have enzymatic activity, and/or control biochemical reactions.

Effects of single gene mutations: If a cell's DNA is mutated, a genetic defect results, causing the production of an abnormal quantity and/or form of the encoded protein. This dramatically disrupts normal functioning- enough to result in disease.

Effects of gene polymorphisms (SNP): Changes in a single nucleotide in a DNA(gene) sequence can result in a different version (isoform) of the protein being synthesized. Such a genetic polymorphism can result in a range of changes (from no observable change, to a minor functional change to absolute loss of function) attributed to the protein isoform.[3]

Polymorphisms Versus Mutations

CLINICAL CORRELATE

What is the importance of studying the heritability of a disease? What is the heritability (contribution of host genetic variants) of periodontitis in humans?

Heritability measures the extent to which genetic factors can account for a disease seen in a population. In the era of precision medicine, knowledge of disease heritability can help to:

- Estimate individual disease risk due to a SNP
- Inform prevention and treatment based on individual disease-susceptibility

Some studies estimate that the *heritability for periodontal disease* varies from around 0.3 to 0.5 (30% to 50%), while others reject the hypothesis that heritability may contribute substantially towards the development of periodontitis. However, some consensus exists around certain aspects of heritability:

- Heritability for periodontitis and other chronic inflammatory diseases may share some pathogenesis pathways
- A substantial-portion of the differences in clinical presentation of periodontitis in a population is due to genetics
- The contribution of heritability will be relatively higher for severe periodontitis in younger individuals who are yet to be exposed to environmental risk factors
- Heritability is considerably increased when genome-smoking interactions are taken into account.[4]

Table 10.1 shows the major differences between polymorphisms and mutations.

Genetic Analysis Methods Used to Study Periodontal Disease

To evaluate the quality of genetic studies supporting the association of mutations and polymorphisms with periodontal disease pathogenesis, an understanding of the genetic analytical methods used is required. Of the many methods available, two approaches are very important in studying the genetic basis of periodontal disease: the candidate gene approach, and genome-wide association studies (GWAS). Table 10.2 provides an overview of relevant methods of genetic analysis currently used to study periodontal disease.

Syndromic Forms of Periodontitis

Severe periodontitis can present as part of a number of monogenic syndromes (i.e., those inherited as simple Mendelian traits due to mutations in a single gene). Patterns of inheritance for single-gene disorders can be autosomal dominant, autosomal recessive, or X-linked (inherited on the X

TABLE 10.1 Genetic Polymorphisms Versus Mutations in Periodontal Disease[2]

	Mutations	Polymorphisms
Effect on pathogenesis of periodontal disease	Alteration of a single gene can result in apparent disruption of a protein product (e.g., Mendelian forms of periodontitis in Papillon–Lefèvre syndrome are caused by mutation in the cathepsin C gene)	Alteration of a gene can result in very subtle perturbations of protein function. Consequently, the specific protein products of allelic variants may function abnormally and these abnormalities can be worsened by environmental factors such as, smoking or microbial factors (e.g., IL-1 composite gene polymorphism increases the risk of susceptibility to periodontitis in smokers)
	No compensation in the biologic system can overcome the effect of the underlying genetic defect	Biologic pathways involving immune-inflammatory responses have many compensatory aspects that make it difficult to quantify the effect of any single genetic variant on disease state
Association of genetic alteration with disease phenotype	Strong and predictable	Weak because periodontitis is a complex, multifactorial disease with no single strong cause (Instead, diseased individuals exhibit specific polymorphisms more frequently than unaffected people)
Mode of inheritance	Classic Mendelian (autosomal dominant, autosomal recessive, X-linked)	Complex result of "gene-epigenetic" and "gene-environment" interactions
Population prevalence	Rare (typically < 0.1%)	More common (typically > 1%; sometimes as high as 20%–50%)
Clinical implications	When the gene responsible for the disease has been identified, it is possible to develop a diagnostic test	Identifying a single polymorphism associated strongly with the disease is not enough to develop a diagnostic test. This knowledge must be assessed along with other facets of etiology (environmental, host, microbial risk factors) for a complete diagnosis.

sex chromosome). These Mendelian forms of periodontitis may be grouped as follows:

Periodontitis associated with neutrophil disorders—the clinical phenotype is associated with recurrent bacterial infections, impaired myelopoiesis, aggressive forms of periodontitis, recurrent oral ulcerations, and candidal infections. This may be due to:

- Deficient neutrophil numbers/neutropenia: describes abnormally low numbers of circulating polymorphonuclear leukocytes (PMNs). Inherited neutropenias associated with periodontitis are congenital neutropenia, cyclic neutropenia, infantile agranulocytosis, and familial benign chronic neutropenia.
- Aberrant neutrophil functions: this category includes impaired adhesion to vessel walls (leukocyte adhesion deficiency, LAD), impaired chemotaxis, and impaired bactericidal activity (Chédiak–Higashi syndrome).

Periodontitis associated with metabolic, structural, or immune protein defects:

- Cathepsin C defects—cathepsin C is a neutrophil lysosomal protease, important for bringing about the resolution of inflammation by stopping recruitment of more PMNs into tissues. This is achieved by cleaving macrophage inhibitory protein-1α (which is chemotactic for PMNs) by neutrophil enzymes. When cathepsin C is deficient, excessive PMN pooling within tissues results, leading to inflammation-associated destruction of periodontal tissues. Severe periodontitis happens early in life and is associated with palmar-plantar hyperkeratosis (Papillon–Lefèvre syndrome and Haim–Munk syndrome).
- Collagen defects—poor wound healing is a component of a heterogeneous group of connective tissue disorders characterized by characterized by "joint laxity and skin hyperextensibility, scarring, and bruising". These are collectively known as Ehlers–Danlos syndromes (there are 17 types); severe forms of early-onset periodontitis has been associated with type IV and type VIII subtypes.

TABLE 10.2 Methods of Genetic Analysis Used to Study Periodontal Disease[2]

Method	Objective	Limitations
Familial aggregation	To suggest genetic etiology by studying families and looking for clustering of specific traits	Results are confounded by common environmental factors shared by families (e.g., diet, nutrition, exposure to pollutants like active and passive smoking, exposure to infectious agents)
Twin studies	To assess relative contributions of genes and environment to a disease trait Based on the assumption that if a disease has high heritability, identical (monozygous) twins will be more likely to be either both affected or both unaffected (concordant)	The base assumption is complicated in many situations: • When a genetic mutation does not have complete penetrance, the environmental factors such as smoking may lead to disease development. • This approach may not provide good results in a polygenic disease that is caused by alterations in multiple genes
Segregation analysis	To study the pattern of disease transmission (autosomal, X-linked, dominant, recessive, complex, multi locus, or random environmental)	• Cannot find a specific gene responsible for a disease trait • Segregation analyses are comparisons between two models of transmission and hence the results are only as good as the models compared and tested. If incorrect assumptions of the tested models are made on important aspects, this will undermine quality of the results
Linkage analysis	To localize the gene for a trait to a specific chromosomal location	• Only the first step in determining the approximate location of a gene of interest. Subsequent tests are required to identify the gene mutations/variants responsible for a disease trait • Effective in Mendelian traits, but not so powerful in complex genetic traits that are due to the combined effects of multiple "genes of minor effect" • Expensive
Candidate gene approach; hypothesis-testing approach	• To test whether one allele of a gene occurs more often in patients with the disease than in subjects without the disease • Genes in this approach are chosen based on their known or presumed function (i.e., they have some plausible role in the disease process, such as producing a protein that is important in pathogenesis)	This type of *association analysis* requires some knowledge of the candidate gene in order to look for it
Genome-wide association study (GWAS); hypothesis-generating approach	• To investigate genetic variation across the *entire* genome simultaneously, with the aim of identifying allelic variants associated with a trait or disease of interest • Performed as an open-ended study with no candidate gene in mind	The results of a GWAS need to be further validated by a cohort with a candidate gene approach study

TABLE 10.3 Mendelian Forms of Periodontitis[5]

Periodontitis Associated With Neutrophil Disorders		
Abnormal Numbers (Neutropenia):	**Characteristic Abnormality**	**Mode of Inheritance**
Congenital neutropenia	Neutrophil elastase	AD
Cyclic neutropenia	Neutrophil elastase	AD
Infantile agranulocytosis (Kostmann syndrome)	Cathelicidin LL-37	AR
Familial benign chronic neutropenia	Defect unknown	AD
Abnormal Neutrophil Function:	**Characteristic Abnormality**	**Mode of Inheritance**
Leukocyte adhesion deficiency type 1	Leukocyte chain adhesion molecule (integrin B subunit) CD 18	AR
Leukocyte adhesion deficiency type 2	Glucose diphosphate-fucose transporter-1	AR
Chédiak–Higashi syndrome	Lysosomal trafficking regulator gene	AR
Periodontitis Associated With Metabolic, Structural or Immune Protein Defects		
	Characteristic Abnormality	**Mode of Inheritance**
Papillon–Lefèvre syndrome	Cathepsin C	AR
Haim–Munk syndrome	Cathepsin C	AR
Ehlers–Danlos syndrome type IV	Collagen III	AD
Ehlers–Danlos syndrome type VIII	Unknown	AD
Hypophosphatasia	Alkaline phosphatase	AD or AR

AD, autosomal dominant; AR, autosomal recessive.

CLINICAL CORRELATE

What are the important clinical implications of studying the syndromic forms of periodontitis?

The association of severe periodontitis with some Mendelian conditions carries important implications:

- Because many of these conditions do not respond well to conventional periodontal therapies, identifying the genes responsible and targeting treatment to overcome/compensate for the underlying biologic defects can be more effective (i.e., precision dentistry—using genetics for personalized treatment).
- Everytime a gene mutation is identified to be associated with syndromic periodontitis, it provides a starting point to further understand the exact role of the protein that the gene codes for. This in turn permits clearer understanding of the pathogenesis of periodontal disease, and facilitates the identification of novel therapeutic targets to treat the disease.[5]

- Alkaline phosphatase deficiency—this leads to abnormal mineralization of bone and dental tissues. This condition is called hypophosphatasia and is characterized by premature shedding of primary teeth, presumably secondary to defective cementum, hypoplastic enamel, and aggressive forms of periodontitis.

Examples of monogenic conditions associated with periodontitis are given in Table 10.3.

Nonsyndromic Forms of Periodontitis

A simple cause and effect relationship between a particular SNP (gene polymorphism) and periodontitis is rather difficult to establish as multiple genes may be contributing to overall disease risk in such complex diseases.

Genetic variations can result in changes in:

- tissue structure (innate immunity)
- antibody responses (adaptive immunity)
- inflammatory mediators (nonspecific inflammation)

When trying to prove an association between a disease and a SNP, the following requirements must be kept in mind[3]:

- Any gene polymorphism under study must alter the gene product (protein)
- Selection Bias in cases/control groups must be recognized and reported
- Smoking, socioeconomic status and other confounding factors must be accounted for in the study results.
- The protein encoded for by the candidate gene must play a part in the pathophysiological pathway.

Table 10.4 lists the most promising candidate genetic polymorphisms from the viewpoint of a genetic diagnostic approach to periodontitis.

TABLE 10.4 Genetic Polymorphisms Associated With Periodontitis[2,6]

Category	Gene/Protein Affected by SNP
Inflammatory mediators and cytokine gene polymorphisms	Interleukin (IL)-1 gene cluster (IL-1A, IL-1B, IL-1 receptor antagonist), IL-4, IL-6, and IL-10.
	TNFα
	β-defensin 1
Receptor gene polymorphisms	Leukocyte receptors for the constant (Fc) part of immunoglobulin (FcγR)
	Vitamin D receptor
	Pattern-recognition receptors (TLRs, CD-14)
Enzyme gene polymorphisms	Matrix metalloproteinase 1 (MMP-1)
	Glucosyl transferase
	Cyclooxygenase 2 (COX2)

SNP, single nucleotide polymorphism.

CLINICAL CORRELATE

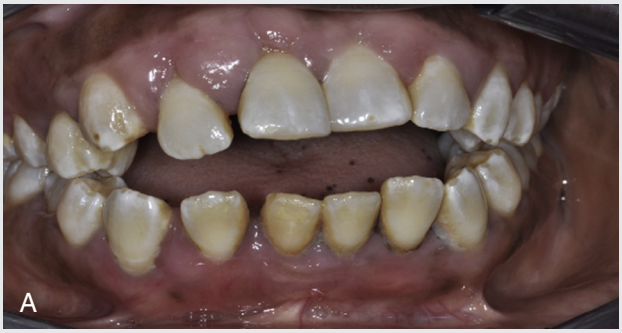
What are the problems in studying genetic polymorphisms in a complex disease like periodontitis, from a diagnostic point of view?

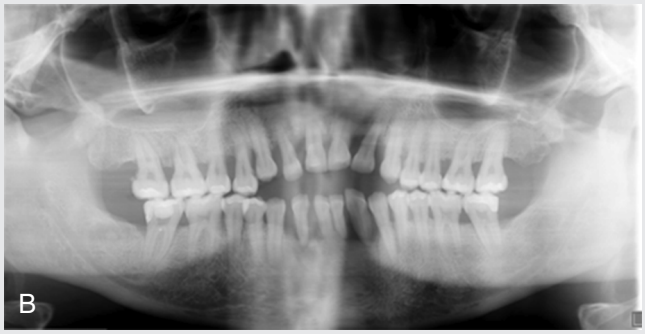
The presence of a disease-associated allele in an individual is not diagnostic for periodontitis because:

- It is difficult to establish causal links.
- Often the only support for a link is the statistical association of an allele with a disease state.
- Disease alleles may also be present in unaffected individuals.
- Individuals with complex disease traits need not have all the alleles associated with the disease.[6]

CASE-BASED LEARNING EXERCISE

Scenario: A 37-year-old female presented with the chief complaint: "My gums are bleeding and tender. My teeth are very loose too." She reported a medical history of hypertension that was initially treated with amlodipine and later switched to lisinopril. Patient had a 10-pack-year cigarette smoking history. Clinical findings: generalized gingival enlargement with deep probing depths, bleeding on probing, mobility, furcation involvement, and deposits of plaque and calculus. Radiographic findings: generalized slight to moderate and localized areas of severe horizontal bone loss, especially in the maxillary and mandibular anterior regions (see images A and B).





Questions

1. Identify the correct equation to understand the contribution of genetics to periodontal disease in the case presented above:
   - **a.** Genotype = phenotype + environment + biologic interactions.
   - **b.** Phenotype = genotype + environment + biologic interactions.
   - **c.** Genotype = phenotype + environment.
   - **d.** Phenotype = genotype + environment.
2. This patient went through genetic testing, which showed that she has a polymorphism in the IL-1 gene. What effect does the IL-1 gene polymorphism, along with her smoking habit, have on the periodontal disease process?
   - **a.** No change in the risk for periodontitis
   - **b.** Increased risk for periodontitis
   - **c.** Decreased risk for periodontitis
3. Identify the syndrome, of which severe periodontal disease is a component, that is associated with a defect in a gene that encodes proteins that play a role in immunity.
   - **a.** Cyclic neutropenia
   - **b.** Kostmann syndrome
   - **c.** Papillon–Lefèvre syndrome
   - **d.** Chédiak–Higashi syndrome
4. What are the possible consequences of a gene polymorphism?
   - **a.** No effect on protein function
   - **b.** Minor effect on protein function
   - **c.** Major effect on protein function
   - **d.** All of the above

This chapter was developed from Chapter 11 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: b

Explanation: A simplified equation to understand the contribution of a genetic variant to a complex disease like periodontitis would be: Phenotype = genotype + environment + biologic interactions.

2. Answer: b

Explanation: Several clinical studies point to an increased risk for periodontitis when an IL-1 gene polymorphism exists in a smoker. This is a good example of **gene-environment** interaction influencing a disease process.

3. Answer: c

Explanation: Severe periodontitis can be a clinical manifestation of a number of monogenic syndromes. These forms of periodontitis may be associated with neutrophil disorders (options a, b, and d) or with metabolic, structural, or immune protein defects (option c) (see Table 10.3). In Papillon–Lefèvre syndome the defect is in the cathepsin C gene, which encodes for a peptidase involved in the resolution of inflammation.

4. Answer: d

Explanation: The effect of a gene polymorphism can range from no effect on protein function to major defects in protein function.

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11

Impact of Periodontal Infection on Systemic Health

Relevant Terminology

Terminology	Explanation
bacteremia	The presence of bacteria in the blood.
chronic obstructive pulmonary disease (COPD)	• Airflow obstruction that results from chronic bronchitis or emphysema • Characterized by enlarged bronchial mucous glands, and accumulation of neutrophils and inflammatory cells within the lung tissue • Smoking is the primary risk factor
community-acquired bacterial pneumonia	• Caused by the inhalation of infectious aerosols or the aspiration of oropharyngeal microorganisms (*Streptococcus pneumoniae* and *Haemophilus influenzae*)
glycated/glycosylated hemoglobin (HbA1c)	• Glucose remains attached to the hemoglobin (glycated Hb) for the entire life span of red blood cells (2–3 months) • The glycosylated hemoglobin test shows what a person's average blood glucose level was for the last 2–3 months
glycemia	The presence of glucose in the blood
glycemic control	• For those with diabetes, glycemic control is a major goal • Tight glycemic control is the clinical practice of controlling blood glucose within an established normal range, aiming to avoid any potential deleterious effects of hyperglycemia
hospital-acquired (nosocomial) bacterial pneumonia	• Most common cause is the aspiration of oropharyngeal contents and dental plaque, which serves as a reservoir of potential respiratory pathogens • Very high morbidity and mortality • Incidence highest among severely ill patients in intensive care units or on ventilator support
low birth weight (LBW)	• Newborns weighing < 2500 g at birth • LBW infants are 40 times more likely to die during the neonatal period than infants with normal birth weight • Increased risk for congenital anomalies, respiratory disorders, and neurodevelopmental disabilities • Caused by preterm labor or premature rupture of membranes; prostaglandins are implicated in this process
lower airways	• Location of gas exchange • Usually maintained free of microorganisms by a combination of immune factors and mechanical clearance through the cough reflex, ciliary transport, and the movement of secretions from the lower airways into the trachea
periodontal medicine	• Field of periodontics that studies the link between periodontal condition and systemic health • Inflammatory periodontal disease may have wide-ranging systemic effects and acts as an independent factor affecting systemic health
selective decontamination	Combines systemic antibiotics with orally administered nonabsorbable antibiotics in an attempt to eradicate potential respiratory pathogens from the digestive tract and the oropharynx, thereby minimizing the risk of nosocomial respiratory infections.

Continued

Relevant Terminology—cont'd

Terminology	Explanation
sulcular epithelium	• Frequently ulcerated and discontinuous in patients with periodontitis • Bacteria and their by-products enter host tissues and blood circulation through ulcerated sulcular epithelium
systemic inflammatory markers	• C-reactive protein (CRP) and fibrinogen are produced in the liver in response to inflammatory or infectious stimuli • CRP induces monocytes and macrophages to produce tissue factors, which contribute to the coagulation pathway • Serum CRP and fibrinogen levels are often elevated in subjects with periodontitis as compared with subjects without periodontitis, indirectly elevating systemic inflammatory response

Fast Facts

Periodontitis	Periodontitis is an infectious disease associated with a small number of predominantly gram-negative microorganisms (present in subgingival biofilm) in a susceptible host
Host susceptibility	• Pathogenic bacteria are necessary but not sufficient to cause disease • In a host who has relatively low susceptibility to disease, bacterial pathogens may have no clinical effect • In a host with relatively high disease susceptibility, marked destruction of periodontal tissues may result • Not all individuals are equally vulnerable to the destructive effects of periodontal pathogens and the inflammatory response to those organisms
Effects of periodontal infection on atherosclerosis and cardiovascular disease	• Periodontal bacteria that disseminate from the oral cavity to the systemic vasculature can live within distant tissues • Low-level bacteremia may initiate host responses that alter coagulability, endothelial and vessel wall integrity, and platelet function, resulting in atherogenic changes and possible thromboembolic events
Periodontal disease and erectile dysfunction (ED)	• ED is associated with endothelial dysfunction • Elevated levels of oxidative stress and systemic inflammation are common to both periodontal disease and ED • The relationship is still speculative; the mechanism of interaction between ED and periodontal disease needs further research
Periodontal disease and stroke	• Periodontal disease is associated with an increased risk of stroke: approximate threefold increased risk has been suggested[1,2] • Periodontal disease provides a persistent bacterial challenge to the arterial endothelium, thereby contributing to the monocyte- and macrophage-driven inflammatory process that results in atheromatosis and narrowing of the vessel lumen (direct mechanism) • Periodontal disease also increases systemic markers such as C-reactive protein (CRP) and fibrinogen (indirect mechanism) • Periodontal pathogens can increase platelet aggregation
Periodontal disease and diabetes mellitus	• Having periodontitis may worsen the glycemic control of diabetic patients • Periodontal treatment may have beneficial effects on glycemic control • Systemic antibiotics as an adjunct to scaling and root planning (SRP) may benefit patients with uncontrolled DM and severe periodontitis • Tetracyclines are known to suppress the glycation of proteins and decrease the activity of tissue-degrading enzymes (e.g., matrix metalloproteinases) and can be useful in these patients
Common complications of diabetes mellitus (DM)	• Retinopathy • Nephropathy • Neuropathy • Macrovascular disease • Altered wound healing • Periodontal disease
Potential mechanism by which periodontal therapy affects HbA1c	• Gram-negative periodontal infection increases insulin resistance, resulting in worsened glycemic control • Reducing systemic inflammation through periodontal treatment results in improved insulin sensitivity, leading to improved glycemic control

Fast Facts—cont'd

Potential mechanism of preterm labor	• Maternal bacterial infection leads to the presence of amniotic bacterial products (e.g., lipopolysaccharides), which stimulates proinflammatory cytokine production, including prostaglandin E_2 (PGE_2) • Premature rise in PGE_2 is characteristic of preterm labor
Periodontal disease and preterm birth	Although not conclusive, some studies have demonstrated that periodontitis was associated with a significantly increased risk of preterm birth and LBW.[3,4] Proposed mechanisms include: • Oral microorganisms disseminate to the fetal-placental unit (direct pathway) • Systemic inflammation with increased inflammatory cytokines (indirect pathway)
Effects of periodontal therapy on pregnancy outcome	• The impact of periodontal therapy on pregnancy outcome is controversial • SRP during the second and third trimester of gestation is safe. Use of local anesthetic is acceptable during such treatment
Periodontal disease and chronic obstructive pulmonary disease (COPD)	• Host inflammatory response in response to chronic challenge by bacteria and effects of smoking is common in both periodontal disease and COPD • Smoking may be a major effect modifier in the relationship between COPD and periodontal disease • In COPD patients with periodontitis, periodontal therapy may impact respiratory health
Periodontal disease and acute respiratory infections	• Oropharynx is the primary site of potential respiratory pathogen colonization • Improving oral hygiene has the potential to decrease the risk of nosocomial pneumonia in high-risk patients (e.g, those in intensive care units and on ventilators)
Periodontal disease and asthma	• Patients with periodontitis may be more likely to have severe asthma than those without periodontitis • Inflammatory response may be the link between periodontitis and asthma

Core Knowledge

Introduction

Periodontitis is a multifactorial disease. While oral care providers can easily recognize periodontal disease conditions, the successful management of these conditions involves an understanding of systemic factors that can be both the cause and effect of periodontal disease. This chapter reviews the various systemic effects associated with periodontal disease and discusses our current understanding of the impact of periodontal treatment on systemic conditions.

Systemic Conditions Influenced by Periodontal Disease

Recent literature suggests that chronic inflammation, such as that triggered in periodontal disease, may influence a number of systemic diseases (Fig. 11.1). It is believed that bacteria and their products from dental plaque enter the general blood circulation through discontinuities in the oral tissues (such as ulcerated sulcular epithelium) and travel via bloodstream to cause infections and propagate inflammation at distant sites.

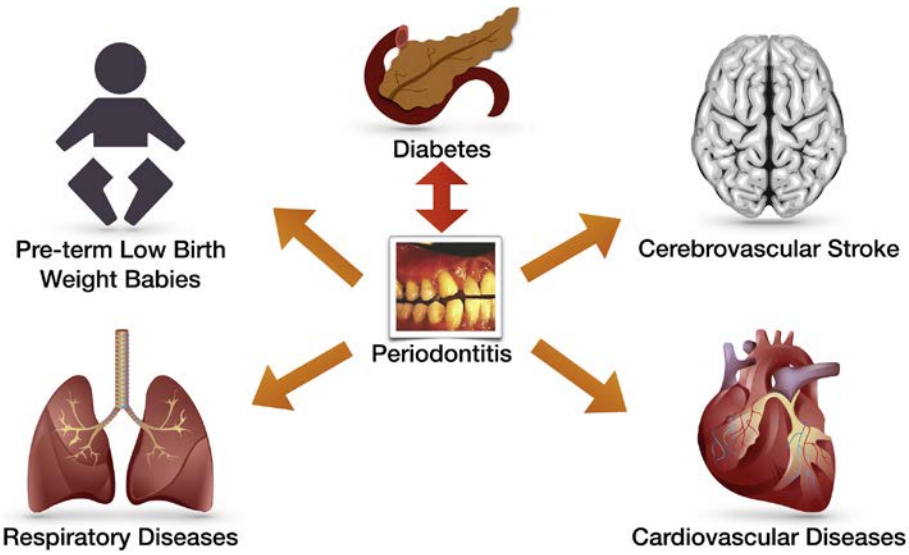
Periodontal Disease: Association With Atherosclerosis, Cardiovascular Disease, and Stroke

There is consistent and strong epidemiological evidence to support the association of periodontitis with increased risk for future cardiovascular and cerebrovascular disease due to thickening of vessel walls that obstructs blood flow (atherosclerosis).

- There is strong evidence that periodontal bacteria disseminated from the oral cavity to the systemic vasculature can be found within distant tissues, and can live within those affected tissues. Atheromas obtained from humans during *endarterectomy* (surgical procedure done to remove the atheromatous plaque material) showed that more than half of the lesions contained periodontal pathogens.
- Low-level bacteremia may result in atherogenic changes and possible thromboembolic events (Fig. 11.2) by having significant effects on:
  - endothelial cells
  - blood coagulation
  - lipid metabolism
  - monocyte- and macrophage-associated inflammatory events.

Periodontal Treatment: Effects on Risk Factors for Cardiovascular and Cerebrovascular Diseases

- Periodontal therapies can improve *surrogate* cardiovascular and cerebrovascular disease outcomes like serum biomarkers and endothelial dysfunction. Any direct effects of periodontal treatment on actual end points such as heart attacks and stroke are yet to be evaluated convincingly.
- Periodontal therapy has been shown to reduce levels of proinflammatory cytokines, CRP, and fibrinogen, and to improve lipid profile, blood pressure, left ventricular mass and pulse-wave velocity (measure of arterial function).



• **Fig. 11.1** Systemic Effects of Periodontal Disease.[5,6] Periodontal infection is thought to have far-reaching consequences on different parts of the body, mainly due to the direct effects of bacteremia and the indirect effects of host immune-inflammatory reactions to periodontal pathogens and their products. Recent evidence focuses on poor oral health, particularly periodontal disease, as an important influence on the initiation and progression of several systemic diseases and conditions, for example:

- diabetes mellitus
- cerebrovascular stroke
- cardiovascular diseases (atherosclerosis and ischemic heart diseases)
- adverse pregnancy outcomes (e.g., preterm and low birth weight babies)
- respiratory diseases (e.g., chronic obstructive pulmonary disease, asthma, and acute respiratory diseases).

(From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

- Increased arterial intima-media thickness (IMT) is a marker for atherosclerosis and is associated with increased risk of future cardiovascular events. Periodontal treatment leads to decreased IMT of the carotid artery and hence leads to improvement in endothelial function.[6]

CLINICAL CORRELATE

What is the association between cardiovascular disease (CVD) and periodontal disease according to current research? Does periodontal therapy decrease the risk for atherosclerosis and ischemic events?

A statistically significant association exists between CVD and periodontal disease, and it is believed that periodontal disease is an *independent* risk factor for atherosclerosis. In other words, common risk factors shared between periodontal disease and CVD (e.g., diabetes, smoking, heredity, obesity) do not explain the association between the two conditions completely; it is thought that periodontal disease itself contributes to the risk for atherosclerosis. However, whether periodontal therapy decreases the risk for atherosclerosis and associated ischemic events of the heart and brain is yet to be established.[7]

Periodontal Disease and Diabetes Mellitus

Elevation in blood glucose levels occurs as part of any inflammatory response; periodontal disease is no exception to this phenomenon. Periodontal infection contributes to various aspects, often in a dose-response manner:

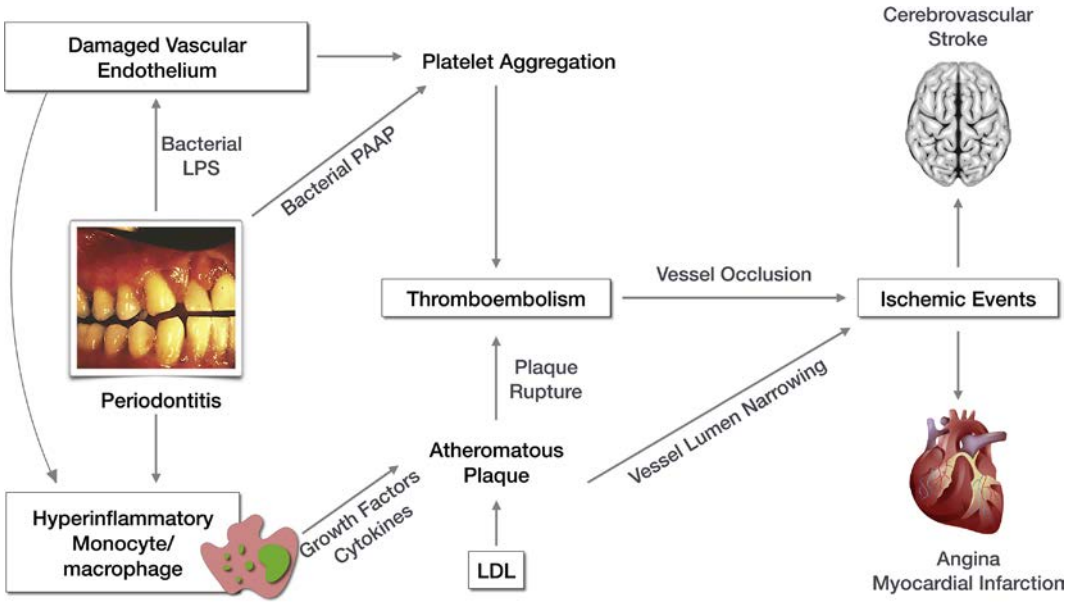
In individuals who are not known to be diabetic:

- Increased blood sugar levels in smokers and so far healthy individuals can occur
- Gestational diabetes and type 2 diabetes can develop
- Impaired glucose tolerance can occur

In known diabetic patients:

- Less effective control of hyperglycemic condition in existing type 2 diabetes can be seen
- Manifestation of some components of metabolic syndrome (a cluster of conditions including increased blood pressure, high blood sugar, excess body fat around the waist, and abnormal cholesterol/triglyceride levels that can lead to diabetes and heart disease) can occur;
- Development of diabetic complications (retinopathy, neuropathy, nephropathy, diabetic foot, subclinical heart disease, kidney and microvascular complications) can occur.

See Fig. 11.3 to understand how periodontal inflammation leads to hyperglycemia.



• **Fig. 11.2** Association of Periodontal Inflammation With Atherosclerosis, Cardiovascular Disease, and Stroke. This figure provides a conceptual model tracing the pathway from periodontal inflammation to ischemic events of heart and brain. A "periodontitis" lesion is a reservoir of bacteria and their products.

Effects on vascular endothelium—bacterial lipopolysaccharides (LPS/endotoxin) can pass into systemic circulation from the periodontium. In cases of severe plaque/calculus accumulation or severe periodontitis, this can happen even with normal activities like chewing, tooth brushing, or during dental procedures like scaling. Bacteremia and release of bacterial toxins within the circulation can cause loss of vessel wall integrity by inducing damage to vascular endothelium.

Effects on blood coagulability—some strains of periodontal pathogens (e.g., *Streptococcus sanguis* and *Porphyromonas gingivalis*) express platelet aggregation–associated protein (PAAP). Platelets bind to these bacteria and clump together; the aggregation of circulating platelets causes thromboembolism. Damage to vascular endothelium also causes platelet aggregation.

Effects on monocytes/macrophages—monocytes in the circulation adhere to damaged vascular endothelium via adhesion molecules (e.g., ICAM-1, ELAM-1,VCAM-1), which are upregulated by LPS, prostaglandins, and proinflammatory cytokines. Monocytes from inside vessel lumen enter the damaged vessel wall under the arterial intima layer and become macrophages (hyperinflammatory phenotype). They then ingest and become engorged with low-density lipoproteins from the circulation, forming "foam cells." The hyperinflammatory macrophages release growth factors that cause vessel wall thickening by stimulating collagen and muscle fiber growth. This thickening forms a cholesterol-rich atheroma or atheromatous plaque, which narrows the blood vessel lumen and decreases blood flow, leading to ischemia. Inflammatory markers shared by both cardiovascular diseases and periodontal disease (IL-1, IL-6, IL-18, CRP, TNFα) contribute to atheromatous plaque rupture.

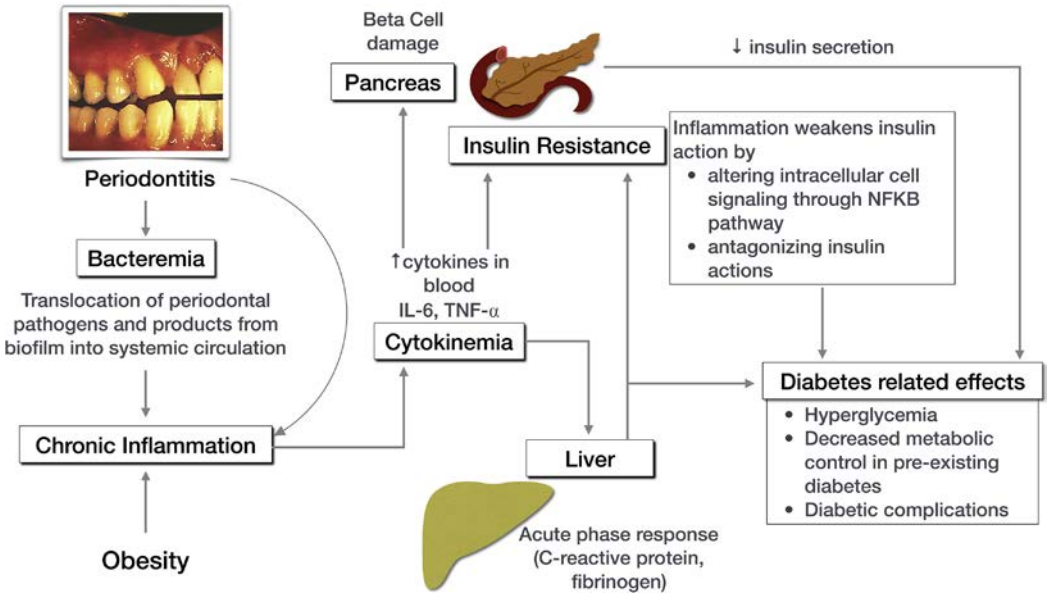
All these events together contribute to *embolus* formation that can cause blockage or occlusion of the blood vessel; ischemia results because of the loss of blood supply and oxygenation. If atheromatous plaques and emboli build up significantly enough to clog brain arteries, 'stroke' results; a similar occurence in coronary arteries results in myocardial infarction. CRP, C-reactive protein; ELAM, endothelial leukocyte adhesion molecule; ICAM, intercellular cell adhesion molecule; IL, interleukin; *LDL*, low-density lipoprotein; *LPS,* lipopolysaccharide; *PAAP*, platelet aggregation–associated protein; TNF, tumor necrosis factor; VCAM, vascular cell adhesion molecule.

(From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

Periodontal Treatment in Diabetes Mellitus: Effects on Glycemic Control

It seems logical to assume that treating periodontal infection would diminish inflammatory responses and thus reduce elevated blood glucose levels. This is because periodontal treatment reduces IL-6 and TNFα, the inflammatory mediators that can interfere with insulin production and function. There is evidence that nonsurgical periodontal treatment achieves the following effects in diabetic patients:[6]

- Decreased systemic inflammation;
- Decreased levels of glycated hemoglobin (HbA1c) by a mean value of 0.4% and improved glycemic control in the short term (3 months);
- Smaller impact on glycemic control in patients with type 1 diabetes than in those with type 2 disease. (Type 1 diabetes is not strongly associated with insulin resistance, so reduced inflammation after periodontal therapy may not have a major effect on insulin sensitivity in patients with type 1 disease; this would minimize the impact of periodontal treatment in Type 1 diabetics).



• **Fig. 11.3** Role of Inflammation in Inducing Hyperglycemia.[6] This figure provides a conceptual model tracing the pathway from periodontal inflammation to hyperglycemia. It also integrates inflammation stemming from central/visceral obesity within this pathway to demonstrate the hypothetical role of common events leading to the development of hyperglycemia as a part of the pathogenesis of diabetes and atherosclerotic cardiovascular disease. The main steps leading from periodontal infection to diabetic events are:

1 Untreated periodontitis leads to chronic inflammation with resultant release of inflammatory mediators and cytokines into the systemic circulation (cytokinemia).
2 Bacteria in deep periodontal pockets also easily penetrate ulcerations in inflamed pocket epithelium and enter the bloodstream (bacteremia). Once in the blood, they travel to inviting locations (e.g., atherosclerotic plaque) and multiply in those sites. The frequent, consistent dissemination of periodontal pathogens via the bloodstream causes chronic inflammatory responses. In cases of untreated periodontitis, this can happen even due to daily activities like chewing hard food, tooth brushing, or flossing.
3 Increased inflammation reduces the production of insulin in the pancreas and also weakens the action of insulin, creating "insulin resistance" (i.e., host cells become resistant to the action of insulin, causing reduced uptake of glucose from the blood by cells).
4 When host cells are insulin-resistant, sugar builds up in the blood (hyperglycemia).
5 Increased concentration of inflammatory mediators in the general circulation also causes the liver to release acute-phase proteins (C-reactive protein, fibrinogen, etc.) that can themselves aggravate hyperglycemia.

Finally, lack of insulin, insulin resistance, and release of acute-phase proteins by the liver all contribute to diabetes-related effects within the body: development of new type 2 diabetes in previously nondiabetic persons, decreased metabolic control in preexisting diabetes, increased risk of diabetic complications, and perhaps even gestational diabetes. *IL,* interleukin; *TNF,* tumor necrosis factor; *NF-κB*, nuclear factor kappa beta pathway of intracellular signaling. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

Note: Periodontal therapy is most likely to result in short-term improvement in glycemia in diabetic patients with severe periodontitis and poor metabolic control, who also demonstrate marked reduction in periodontal inflammation after treatment. Conversely, individuals with well-controlled diabetes and periodontitis show less reduction in inflammation and demonstrate only minimal changes in glycemic control following periodontal treatment.

Conclusion

The goal of periodontal therapy, often overlooked, is to improve or maintain a patient's quality of life; this is achieved by ensuring they have a functional set of teeth surrounded by a healthy periodontium. This in turn prevents the initiation and propagation of several systemic chronic diseases by eliminating a focus of infection. Clinicians and patients should be knowledgeable about consistent associations between periodontal disease and systemic diseases as well as about the potential preventive benefits of periodontal interventions.

CLINICAL CORRELATE

Is the reduction in HbA1c achieved by periodontal treatment (a decrease of 0.4%) clinically meaningful?

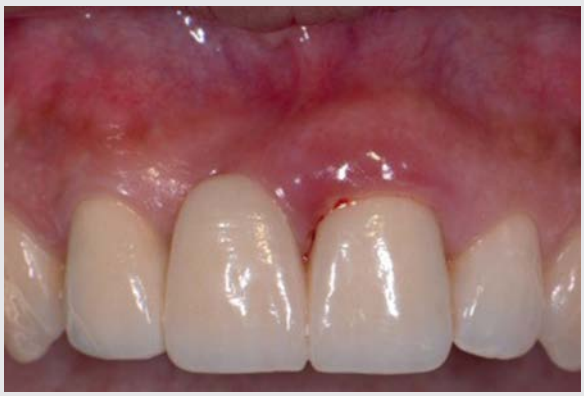
On diagnosis of type 2 diabetes mellitus, typical treatment begins with dietary modifications and exercise, usually supplemented by an oral hypoglycemia agent like metformin. The expected effect of metformin on HbA1c is a reduction of about 1%. Also to be kept in mind:

- The mean reduction in HbA1c values after initial periodontal therapy is similar to estimated reductions achieved with α-glucosidase inhibitors (AGIs; e.g., acarbose, miglitol, voglibose), which are widely used in the treatment of patients with type 2 diabetes. AGIs reduce HbA1c levels by 0.5%.
- Every 0.2% decrease in HbA1c implies a 10% decrease in mortality rate.
- Every 1% decrease in HbA1c is estimated to cause a 35% decrease in the rate of complications.

Therefore the 0.4% decrease in HbA1c that occurs due to nonsurgical periodontal therapy may be clinically significant in managing individuals with poorly controlled diabetes and periodontitis.[6,7]

CASE-BASED LEARNING EXERCISE

Scenario: A 38-year-old female presented to the clinic with the chief complaint "I have swelling and pain in my gums." Background information: Patient was a nonsmoker with poorly controlled DM (HbA1c = 8.4) and was overweight, with a BMI of 27. She reported brushing one to two times per day but did not floss. She reported infrequent dental visits. Her last professional dental cleaning was at least 3 years ago. Current findings: Probing depths were generally in the range of 2–4 mm with localized 5–7 mm (maxillary anterior) and BOP of 43% (image A).



Questions

1 Which of the following diseases have NOT been shown to be adversely affected by periodontal disease?
   a. Coronary heart disease.
   b. Diabetes.
   c. Hypothyroidism.

2 Periodontal therapy has _____ on glycemic control.
   a. A favorable effect.
   b. No effect.
   c. An unfavorable effect.

3 For this clinical scenario, it is recommended to have the HbA1c value recorded every _____ months:
   a. 2.
   b. 3.
   c. 4.
   d. 6.

4 Metabolic syndrome includes which of the following?
   a. High blood pressure.
   b. High glucose.
   c. High abdominal fat with abnormal cholesterol levels.
   d. All of the above.

Clinical image is from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

This chapter was developed from Chapters 14 and 15 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1 Answer: c

Explanation: Periodontal disease has been associated with coronary heart disease and diabetes, but not hypothyroidism; however, causal studies still need to be performed. Proving an association may be difficult, because these disease processes share many of the same risk factors; thus the association may be among the risk factors rather than the diseases themselves (see **Fig. 11.1**).

2 Answer: a

Explanation: Patients who improved periodontal health with treatment also experienced improvements in glycemic control. This effect is pronounced in patients with poor glycemic control and with more advanced periodontal destruction.

3 Answer: b

Explanation: Hyperglycemia leads to glycosylation of proteins, including hemoglobin, resulting in glycosylated hemoglobin formation. Because the life span of red blood cells is 120 days, the HbA1C value should be recorded every 3 months in order to assess the glycemic state of patients.

4 Answer: d

Explanation: Metabolic syndrome by definition includes all of the listed conditions.

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12

Gingiva: Defense Mechanisms and Inflammation

Relevant Terminology

Terminology / Abbreviation	Explanation
alpha and beta defensins	Low-molecular-weight antimicrobial peptides produced by a number of host cells.
anoxemia	Bluish hue on the reddened gingiva caused by impaired blood flow and low oxygen levels; seen at inflamed sites.
diapedesis	The passage of blood cells through the intact walls of capillaries during inflammation.
gingival crevicular fluid (GCF)	• Fluid in the gingival sulcus that is considered more of an inflammatory exudate than a continuous serum transudate • Fluid that contains an array of biologic mediators (biomarkers), cells, and bacteria • Good potential for use in diagnostics and disease management
lactoperoxidase-thiocyanate system in saliva	• Bactericidal to some strains of *Lactobacillus* and *Streptococcus*
lysozyme	Hydrolytic enzyme found in saliva that cleaves structural components of the cell wall of certain bacteria.
myeloperoxidase	Enzyme produced by leukocytes that is bactericidal to *Actinobacillus* species.
orogranulocytes	Living polymorphonuclear cells (PMNs) in saliva.
stasis	Slowing of blood flow, with vasodilation and fluid exudation.
xerostomia	Dry mouth condition due to reduced or absent salivary flow.

Fast Facts

Gingival defense mechanisms	• Gingival health is maintained by the epithelial barrier, immune response, crevicular fluid, and saliva
Methods of GCF collection	Paper/absorbent cellulose strips, micropipette tips, intracrevicular washings and preweighed, twisted threads.
Brill technique of GCF collection	Involves inserting a paper strip into the pocket until resistance is encountered (intrasulcular method). This method produces a degree of irritation of the sulcular epithelium that by itself can trigger the flow of fluid.
Composition of GCF	• Proteins, metabolites, specific antibodies (IgG predominant), antigens, and enzymes of several specificities • Cellular elements: bacteria, desquamated epithelial cells, and leukocytes • Electrolytes: potassium, sodium, and calcium • Organic compounds: carbohydrates, proteins, glucose hexosamine, and hexuronic acid

Continued

Fast Facts—cont'd

Leukocyte levels in healthy gingiva	• Small numbers of leukocytes (predominantly PMNs) are seen in clinically healthy gingival sulci • 58% B lymphocytes, 24% T lymphocytes, 18% mononuclear phagocytes in GCF in gingival health • T cells:B cells = 3:1 in peripheral blood • T cells:B cells = 1:3 in GCF
Functions of saliva	• Lubrication • Physical protection • Cleansing • Buffering • Maintenance of tooth integrity • Antibacterial action
Reduced saliva	• A risk factor for caries and periodontal disease • Xerostomia is defined as dry mouth resulting from reduced or absent saliva flow (flow rate < 0.1mL/min) • Xerostomia may result from sialolithiasis, sarcoidosis, Sjögren syndrome, Mikulicz disease, irradiation, surgical removal of salivary glands, and side effects of medications
Salivary antibodies	• Predominantly IgA (IgG predominant in GCF) • Major and minor salivary glands contribute to all the secretory IgA • IgA impair the ability of bacteria to attach to mucosal and dental surfaces
Salivary enzymes	• Amylase (from parotid gland) is the major enzyme • Concentrations of hyaluronidase, lipase, β-glucuronidase, chondroitin sulfatase, aspartate aminotransferase, alkaline phosphatase, amino acid decarboxylases, catalase, peroxidase, and collagenase increase in periodontal disease
Salivary buffers and coagulation factors	• Bicarbonate–carbonic acid system is the antacid buffering system • Saliva contains coagulation factors (factors VIII, IX, and X, plasma thromboplastin antecedent, and Hageman factor) that hasten blood coagulation and protect wounds from bacterial invasion
Leukocytes in saliva	• Main leukocytes in saliva are polymorphonuclear leukocytes (PMNs) • The migration rate of orogranulocytes (living PMNs in saliva) is referred as the orogranulocytic migratory rate, and can be used as an index for the assessment of gingivitis; increased rate is associated with more severe inflammation
Saliva stimulants	• Pilocarpine • Cevimeline • Anethole trithione • Artificial saliva is NOT a stimulant, but a substitute
Gingival inflammation: the initial lesion	• 2–4 days • Vascular dilation • Predominant infiltration by neutrophils • Degradation of junctional epithelium and perivascular connective tissue • Increased exudation of fluid from gingival sulcus
Gingival inflammation: the early lesion	• 4–7 days • Vascular proliferation • Predominant infiltration by lymphocytes (predominance of T cells) • Atrophic rete pegs • Further loss of perivascular connective tissue • Erythema and bleeding on probing
Gingival inflammation: the established lesion	• 14–21 days • Vascular proliferation and stasis • Predominant immune infiltration by plasma cells • Continued loss of connective tissue • Changes in size, color, texture etc. • Some established lesions last for months or years, while others may become more active and convert to progressively destructive lesions
gingival inflammation: the advanced lesion	• Phase of periodontal breakdown (periodontitis) • Occurs only in a susceptible host • Predominance of plasma cells in connective tissue and neutrophils in junctional epithelium

Core Knowledge

Introduction

Gingival inflammation (gingivitis) is usually caused by plaque bacteria, and defense mechanisms exist within the gingiva to counteract the detrimental effects of periodontal pathogens and their noxious products. While epithelial surfaces, immune cells, and mediators provide oral immunity, the most significant gingival defense mechanisms are mediated by:

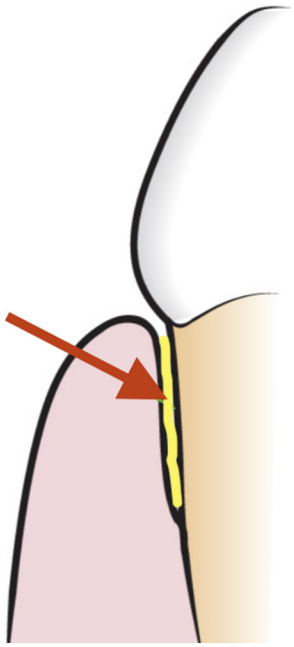
- gingival crevicular fluid (GCF)
- leukocytes in the dentogingival area
- saliva.

Gingival Crevicular Fluid (GCF)

Gingival crevicular fluid (also called sulcular fluid) is considered to be an inflammatory exudate, not a continuous serum transudate. In a strictly normal gingiva (pristine gingiva), little or no fluid can be collected from the sulcus (Fig. 12.1). During inflammation, however, blood vessels in the gingiva dilate, leading to the passage of materials and fluid from the blood vessels through connective tissue, via junctional epithelium into the sulcus.

Composition of GCF:

- ***Enzyme component:*** β-glucuronidase, lactate dehydrogenase, collagenase, phospholipase, etc.
- ***Non-enzyme components:***
  1. Cellular elements—bacteria, desquamated epithelial cells, leukocytes (neutrophils, monocytes, and lymphocytes);
  2. Electrolytes—potassium, sodium, calcium;
  3. Organic compounds—glucose (3–4 times higher concentration in GCF than in serum), proteins (lower concentration than in serum), tissue breakdown products, antibodies (immunoglobulins IgG, IgA, IgM), complement protein components (e.g., C3, C4), plasma proteins (e.g., albumin), endotoxins.



• **Fig. 12.1** Diagram of the Gingival Crevice and Gingival Crevicular Fluid (in yellow). (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

Increase in GCF flow: About 0.43–1.56 µL of GCF is secreted per day. The amount of fluid increases with inflammation and certain other conditions:

- Circadian periodicity: gradual increase from 6 am to 10 am and a decrease afterwards;
- Influence of sex hormones: increased during pregnancy, ovulation, and intake of hormonal contraceptives, probably because female sex hormones enhance vascular permeability;
- Chewing on coarse foods, gingival massage, and vigorous tooth brushing;
- Post–periodontal surgery healing;
- Smoking causes an immediate, transient, and marked increase in GCF flow;
- Use of prosthetic appliances.

Note: Gingival fluid is *not* increased by trauma from occlusion.

GCF Function: Crevicular fluid plays a protective role, achieved by the following mechanisms:

- Cleansing action: the flow rate and direction of GCF help in flushing out bacteria and "bacteria-invaded and shed" epithelial cells from within the gingival sulcus into the oral cavity.
- Antibacterial properties: GCF is composed of complement components, antibodies, enzymes, and viable leukocytes that act against bacteria.

CLINICAL CORRELATE

What are the advantages of using gingival crevicular fluid (GCF) over serum or saliva as a diagnostic fluid for periodontal disease?

GCF may be better than serum or saliva for monitoring changes occurring during the development of periodontal disease for the following reasons;

1. Advantage over blood collection: It can be collected noninvasively, just like saliva.
2. Advantage over saliva: Its composition and dynamics are more closely related to the environment of the periodontal tissues than saliva. While saliva is made up of components derived from multiple sources (e.g., salivary glands, serum, bacteria, sloughed oral epithelial cells, foreign substances introduced into the oral cavity, and GCF itself), GCF contains products of the host, plaque, and also products of host-microbe interactions which are specific to periodontal environment and hence may more accurately reflect the underlying periodontal disease condition.[1]

Methods of collection of GCF:

- *Absorbent paper/cellulose strips:* GCF is collected using a blotter (Periopaper) and measured electronically using a device called a Periotron. The wetness of the paper strip affects the flow of an electric current and provides a digital readout.
- *Micropipettes:* permit collection of GCF by capillary action (adhesion of liquid to the vessel walls causes an upward force on the liquid at the edges, allowing collection of fluid within the pipette). This fluid is later centrifuged and analyzed for its components.
- *Intracrevicular washings:* A peristaltic pump or "ejection-collection needle" assembly flushes out the gingival sulcular contents and collects them for further analysis.
- *Preweighed twisted threads:* after GCF is collected by absorption, the threads are weighed again and the difference is calculated.

Saliva

Saliva is a *biofluid* mainly produced by three pairs of major salivary glands—the submandibular, parotid, and sublingual glands—along with contributions from many minor salivary glands. Previously, saliva was thought to protect against infection through its immunoglobulins only; however, it is now clear that enzymes, cytokines, nucleotides, and live cells are all part of the host armamentarium that exists in saliva. The protective functions (Table 12.1) are carried out by its organic (e.g., lysozyme) and inorganic (e.g., bicarbonate, phosphate) components.

CLINICAL CORRELATE

What is the influence of saliva on the oral microbiota?

Saliva influences the composition and actions of the oral microbial colonies, via a variety of mechanisms:

1. **Pellicle formation:** Molecules, mainly from saliva, cover oral surfaces, forming a conditioning film that provides receptors for bacterial attachment. The attached bacteria use salivary components (e.g., glycoproteins) as their primary nutrient source for growth.
2. **Commensal bacterial colonization:** Oral bacteria sequentially break down structurally complex molecules from saliva. Saliva also buffers biofilm pH to near neutral values, thus creating a friendly environment for commensals to grow in.
3. **Immune function:** Components of the adaptive and innate host defenses from saliva act at sublethal concentrations, leading to a complex relationship between the host and the resident microflora.
4. **Homeostatic regulation:** Saliva favors the establishment of a highly diverse yet balanced microflbiota (which protects against overgrowth by pathogenic species) rather than a sterile environment. Dysbiosis (microbial imbalance favoring pathogenic species over commensal species) can occur rapidly if salivary flow is disrupted.[2]

TABLE 12.1 Role of Saliva in Oral Defense Mechanisms

Function	Salivary Components	Probable Mechanism
Lubrication and physical protection	Glycoproteins, mucoids	Coating on surfaces similar to gastric mucin
Cleansing	Physical flow	Clearance of debris and bacteria
Buffering	Bicarbonate and phosphate	Act as antacids
Tooth integrity maintenance	Minerals	Maturation, remineralization
	Glycoprotein pellicle	Mechanical protection
Antibacterial action	Immunoglobulin A	Control of bacterial colonization
	Lysozyme	Breaking of bacterial cell walls
	Lactoperoxidase	Oxidation of susceptible bacteria
	Myeloperoxidase	Bactericidal (in susceptible species); inhibits bacterial colonization
	Antimicrobial peptides (human alpha and beta defensins)	Combat bacterial infections; important for homeostasis
Antiproteases	TIMP (against MMP), antileukoprotease (against elastase)	Combat proteolytic enzymes (that destroy host tissue) secreted by host and oral bacteria
Coagulation factors	Factors VIII, IX, and X; plasma thromboplastin antecedent; and Hageman factor	Hasten blood coagulation and protect wounds from bacterial invasion

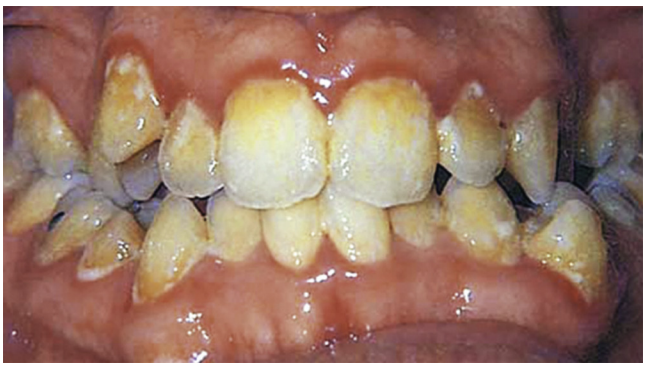
MMP, matrix metalloproteinase; TIMP, tissue inhibitor of metalloproteinase.
Adapted from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

Gingival Inflammation

Inflammation of gingiva is called **gingivitis** (Fig. 12.2). It is a common, reversible condition confined to the gingival epithelium and connective tissue, with *no* destruction of other periodontal structures (such as periodontal ligament or alveolar bone).

The four (histologic) stages of gingival inflammation are:

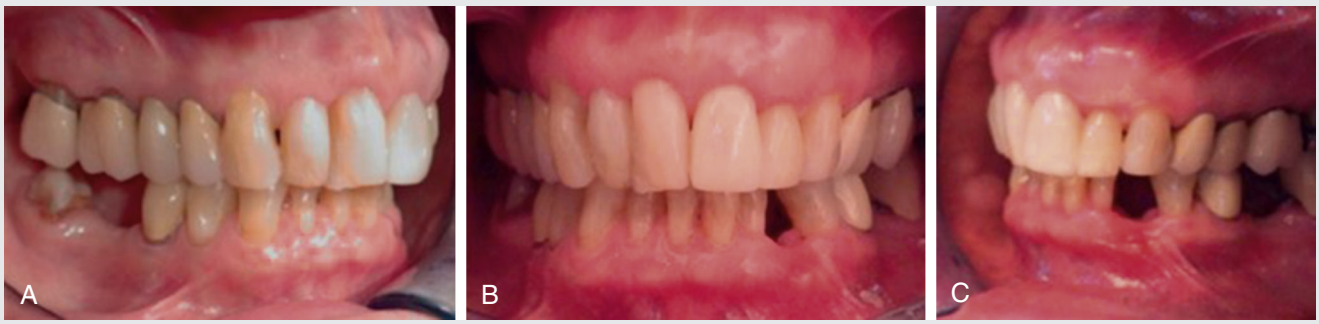
- **Stage I (initial lesion):** Subclinical gingivitis; first response involves vascular changes (dilation of capillaries, increased blood flow) and leukocyte migration (mainly PMNs) into the gingival sulcus.
- **Stage II (early lesion)**: Early gingivitis; clinical signs of erythema and bleeding on probing are evident. Lymphocytes predominate in gingival connective tissue.
- **Stage III (established lesion):** Chronic gingivitis; enlarged and congested blood vessels with impaired venous blood drainage. Red blood cells are extravasated into connective tissue and hemoglobin breakdown occurs. Plasma cells predominate in gingival connective tissue.
- **Stage IV (advanced lesion):** Transition to periodontitis; phase of periodontal breakdown.



• **Fig. 12.2** Generalized Plaque-Induced Gingivitis. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

CASE-BASED LEARNING EXERCISE

Scenario: A 52-year-old female presented to the clinic with the chief complaint: "I have trouble going to sleep because my mouth and throat are extremely dry" (images A to C). Background Information: The patient was a heavy smoker who reported difficulty sleeping, sleeping only for 4 hours per night, and drinking minimal alcohol socially. The patient had a history of allergic rhinitis, for which she was taking over-the-counter antihistamines and decongestants. Current Findings: Probing depths were in the range of 3–4 mm, bleeding on probing was 38%, and the patient had poor oral hygiene.



Clinical image is from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

Questions

1. Which of the following is not a probable consequence of xerostomia?
- **a.** Caries.
- **b.** Gingivitis.
- **c.** Squamous cell carcinoma.
- **d.** Candidiasis.

2. All of the following are saliva stimulants except:
- **a.** Pilocarpine.
- **b.** Cevimeline.
- **c.** Anethole trithione.
- **d.** Artificial saliva (Oralube).

3. Identify the mechanism(s) that play a major role in saliva's influence on the composition of oral microbiota:
   a. Pellicle formation.
   b. Immune function.
   c. Homeostatic regulation.
   d. All of the above.
4. In a patient who is a mouth breather, gingival inflammation is typically seen in which part of the oral cavity?
   a. Maxillary anterior (buccal aspect).
   b. Maxillary anterior (lingual aspect).
   c. Mandibular anterior (buccal aspect).
   d. Mandibular anterior (lingual aspect).

This chapter was developed from Chapters 16 and 17 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: c

Explanation: The rest are potential consequences of xerostomia. Patients with dry mouth or xerostomia lack the cleansing function of saliva, which can result in the conditions listed.

2. Answer: d

Explanation: Artificial saliva (Oralube) is a saliva substitute, not a stimulant. Artificial saliva or saliva substitutes can be used to replace moisture and lubricate the mouth, whereas saliva stimulants such as pilocarpine act as cholinergic parasympathomimetic agents with predominantly muscarinic M3 action that causes stimulation of residual functioning exocrine glands.

3. Answer: d

Explanation: Saliva plays a major role in determining the composition and activity of the oral microbiota, via a variety of mechanisms: pellicle formation, commensal bacterial colonization, immune function, and homeostatic regulation. More details regarding each mechanism are presented in the second "Clinical Correlate" box.

4. Answer: a

Explanation: In mouth breathers, due to the constant exposure of the buccal aspect of maxillary anterior gingiva to air and the dryness that results from it, the inflammation is typically confined only to this location.

References

1. Curtis, M. A., Gillett, I. R., Griffiths, G. S., Maiden, M. F., Sterne, J. A., Wilson, D. T., et al. (1989). Detection of high-risk groups and individuals for periodontal diseases. *Journal of Clinical Periodontology*, *16*, 1–11.
2. Marsh, P. D., Do, T., Beighton, D., & Devine, D. A. (2016). Influence of saliva on the oral microbiota. *Periodontology 2000*, *70*(1), 80–92.

13 Gingivitis and Acute Gingival Infections

Relevant Terminology

Terminology / Abbreviation	Explanation
communicability	Ability of an infectious agent to be maintained and spread between patients through direct or indirect contact.
gingival recession	The exposure of root surface due to apical migration of the gingival margin in relation to the cementoenamel junction.
gingivitis	Inflammation of the gingiva.
pericoronitis	Inflammation of the gingiva around the incompletely erupted crown (the gingival flap is called "operculum"). Can be acute, subacute, or chronic.
primary herpetic gingivostomatitis	Acute inflammatory disease caused by herpes simplex virus (HSV) type 1 infection.
stippling	• Pitted "orange-peel" appearance of the healthy attached gingiva • It reflects the underlying connective tissue projections (rete ridges) into the epithelium • The disappearance of stippling signifies edematous gingiva (a sign of gingival inflammation)

supracrestal tissue attachment (biologic width)	• Supracrestal tissue attachment (previously called "biologic width") means the apicocoronal dimensions of the supracrestal attached tissues, including
	  • junctional epithelium
	  • connective tissue around the tooth

traumatic lesions of the gingiva	Gingival lesions may be caused by physical (brushing), chemical (etching), or thermal (burning) trauma.
Tzanck cells	Virus-infected epithelial cells exhibit ballooning degeneration characterized by acantholysis, nuclear clearing, and nuclear enlargement. These acantholytic epithelial cells are called Tzanck cells. Histologically, they can be seen in biopsies of primary herpetic gingivostomatitis.

Fast Facts

bleeding on probing	• Commonly presented as percentage of sites that bleed at time of gingival probing; used typically to diagnose gingivitis • Its absence in the clinical examination is an excellent negative predictor of disease progression (attachment loss) • According to the 2017 World Workshop, >10% bleeding sites is considered to be the cutoff for diagnosing gingivitis[1]
Etiology of gingivitis	Gingivitis is primarily induced by dental plaque biofilm accumulation that stimulates inflammatory responses.

Continued

Fast Facts—cont'd

Clinical features of gingivitis	Redness of gingival tissue, bleeding on provocation (bleeding on probing, BOP), gingival contour change (swollen and enlarged), loss of stippling, and the presence of calculus or plaque with no radiographic bone loss.
Prevalence of gingivitis	In the general adult population, the prevalence may be higher than 50%. The reported prevalence depends on the case definition used for gingivitis in different studies.
Severity of gingivitis	According to the 2017 World Workshop, there is no robust and solid evidence to clearly define mild, moderate, and severe gingivitis. However, the severity of gingivitis could be mentioned for clinical communication.[1]
Extent of gingivitis	According to the 2017 World Workshop, gingivitis can be defined as localized or generalized based on the BOP sites: • localized gingivitis: 10%–30% bleeding sites • generalized gingivitis: >30% bleeding sites[1]
Local contributing factors	Any local factor contributing to plaque retention that leads to inflammation can induce gingivitis. These factors include overhangs of restorations, use of prosthetic or orthodontic devices, open contacts, lack of keratinized gingiva, malpositioned teeth, mouth breathing, and anatomic tooth variations.
Impact of smoking on gingivitis	Smoking may induce a chronic, significant, and dose-dependent suppression of gingival bleeding. However, smoking is generally detrimental to periodontal health.
Systemic contributing factors of gingivitis	Hemorrhagic disorders, diabetes, and pregnancy may exacerbate gingivitis because of clotting abnormalities, significant inflammation, and hormonal imbalance, respectively.
Acute gingival infections	Acute gingival infections include, but are not limited to: necrotizing ulcerative gingivitis (NUG), primary herpetic gingivostomatitis, and pericoronitis. Patients presenting with acute gingival infections usually need urgent treatment to relieve pain and control inflammation.
Clinical Characteristics of necrotizing gingivitis (NG)	• Punched-out, crater-like lesions at the interdental papillae and the marginal gingiva • Lesion is usually covered by pseudomembranous slough demarcated by a distinct linear erythema
Characteristics of primary herpetic gingivostomatitis	• Frequently occurs in children younger than 6 years of age • Characterized by diffuse and erythematous lesions and vesicles on the gingiva and oral mucosa • The course of the disease is usually 7–10 days
Characteristics of pericoronitis	• Painful condition which occurs commonly in relation to the partially impacted third molar area in the mandible • The gingival inflammation around the crown is induced by the accumulation of food debris and bacteria, and can be exacerbated by trauma • Complications include trismus, fever, leukocytosis, cellulitis, or Ludwig's angina

Core Knowledge

Introduction

The most common gingival disease is *chronic gingivitis*—a simple inflammatory response to a variety of stimuli, the most common stimulus being dental plaque bacteria. Gingivitis is initiated if dental plaque accumulates near gingival margins over days or weeks without disruption or removal. This may occur even in a treated and healthy reduced periodontium, and is characterized by the return of bacteria-induced inflammation to the gingival margin with no evidence of progressive attachment loss (i.e., no indication of active disease).

Classification of Plaque-Induced Gingivitis

The intensity of the clinical signs and symptoms of plaque-induced gingivitis varies among individuals and among sites within a dentition. Hence, gingivitis may be classified as follows[1,2]:

Based on extent (number of gingival sites exhibiting inflammation):
- Localized—when <30% of teeth are affected;
- Generalized— when >30% of teeth are affected.

Based on severity (intensity of inflammation):
- Mild gingivitis—minor change in color and texture;
- Moderate gingivitis—erythema, edema, enlargement and bleeding on probing (BOP) seen;
- Severe gingivitis—overt erythema and edema with tendency to bleed when touched rather than only when probed.

Based on location:
- Marginal gingivitis—involves the gingival margin;
- Papillary gingivitis—involves the interdental papillae, often extends into the adjacent portion of the gingival margin;

- Diffuse gingivitis—affects the gingival margin, the attached gingiva, and the interdental papillae;

Based on causative and modifying factors:

- A. Associated with bacterial dental biofilm only;
- B. Potential modifying factors of plaque-induced gingivitis:
  - Systemic conditions:
    a) sex steroid hormones (puberty, menstrual cycle, pregnancy, oral contraceptives)
    b) hyperglycemia
    c) leukemia
    d) smoking
    e) malnutrition.
  - Oral factors enhancing plaque accumulation:
    a) prominent subgingival restoration margins*
    b) hyposalivation.
- C. Drug-influenced gingival enlargements.

* Other local plaque-retentive factors (e.g., calculus, orthodontic brackets, overhanging margins) can also influence plaque accumulation.

Based on whether plaque-induced gingival inflammation occurs on an intact or reduced periodontium:

- Gingivitis on an intact periodontium;
- Gingivitis on a reduced periodontium in a non-periodontitis patient (e.g., in cases of recession, crown lengthening).
- Gingival inflammation on a reduced periodontium in a successfully treated periodontitis patient (note that recurrent periodontitis cannot be ruled out in this case)

CLINICAL CORRELATE

What are the clinical features common to all plaque-induced gingival conditions?

The clinical characteristics present in all dental plaque–induced inflammatory gingival conditions include:

- Clinical signs and symptoms of inflammation confined to the gingiva (bleeding, erythema, ulceration);
- Reversibility of gingival inflammation by biofilm removal/disruption
- High bacterial plaque burden that initiates inflammation;
- Systemic factors (e.g., hormones, systemic disorders, drugs) that modify the severity of the plaque-induced inflammation;
- Stable (i.e., nonchanging) attachment levels on a periodontium with/without previous loss of attachment or bone loss.[2]

The current classification also proposes addition of the term *incipient gingivitis*, a condition defined as being characterized by only a few sites being affected by mild inflammation and mild erythema, and/or displaying a delayed and broken line of bleeding. Incipient gingivitis, regarded as a condition of "clinical health", may rapidly progress to localized gingivitis if left untreated.

Clinical Features of Gingivitis

Chronic gingivitis, a nonspecific dental plaque–induced inflammatory condition, can be characterized by any of the following clinical signs of inflammation that are confined to the free and attached gingiva and do not extend beyond the mucogingival junction (Fig. 13.1):

- Bleeding and ulceration;
- Erythema/redness;
- Changes in contour and consistency;
- Presence of calculus or plaque with no radiographic evidence of crestal bone loss.

BASIC SCIENCE CORRELATE

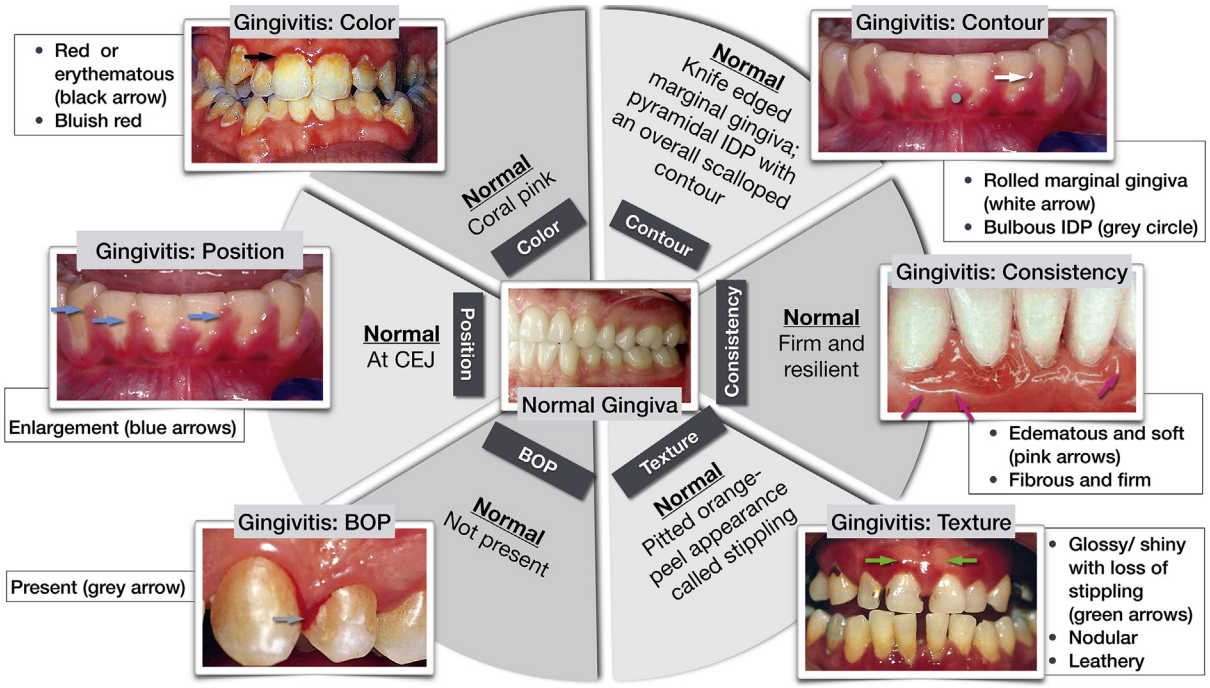
What are the histologic features and molecular characteristics common to all plaque-induced gingivitis conditions?

- Histopathologic changes include inflammation of vasculature adjacent to the junctional epithelium, progressive destruction of the collagen fiber network, elongation of rete pegs into the gingival connective tissue, and a progressive inflammatory-immune cellular infiltrate in subepithelial connective tissue (See Chapter 12 and Fig. 4.1 for a detailed description of the four histopathological stages of gingival inflammation).[2]
- In plaque-induced gingival inflammation, molecular changes involve the gingival *transcriptome* (refers to the sum total of all mRNA molecules expressed by genes in the gingiva), leading to disruption of several components of the host response including:
  - microbial pattern recognition molecules
  - chemotaxis
  - phagocytosis
  - cytokine signaling
  - T lymphocyte response
  - angiogenesis
  - epithelial immune response.

Acute Gingival Infections

Diseases of short duration, recent or rapid onset, and which present with a certain degree of discomfort and pain are termed "acute." While gingivitis in its classical form is quite common, it is chronic in nature and not really associated with pain. Acute gingival infections are not especially common and are more often associated with pain at the time of presentation. They include:

- necrotizing gingivitis (NG)—formerly called acute necrotizing ulcerative gingivitis (ANUG) and necrotizing ulcerative gingivitis (NUG)
- primary herpetic gingivostomatitis
- pericoronitis.



• **Fig. 13.1** Classical Clinical Signs of Inflammation in Gingivitis. The figure lists the clinical features of normal gingiva (within the segments of the circle) and those of gingival inflammation (images outside the circle). A systematic approach for the evaluation of gingivitis requires an orderly examination of the gingiva for color, contour, consistency, texture, gingival margin position, and ease and severity of bleeding: **Color**—While normal gingiva is coral pink in color (with some melanin pigmentation), inflamed gingiva turns red or erythematous due to increased vascularity. Venous stasis contributes a bluish hue in chronic gingivitis. The changes start in the interdental papillae and gingival margin, then spread to the attached gingiva. **Contour**—Normal gingiva shows a scalloped contour such that it firmly encases the necks of teeth, with gingival margins following the course of their cementoenamel junctions. When healthy, the gingival margin appears knife-edged in cross section; during inflammation, it loses this sharp edge and becomes "rolled" when in proximity to plaque or calculus. Changes in gingival contour are primarily associated with gingival enlargement, which causes the normally triangular interdental papillae ("pyramidal" shape in the anteriors) to become bulbous in contour. **Consistency**—Healthy gingiva is firm and resilient. In patients with chronic gingivitis, destructive (i.e., edematous) and reparative (i.e., fibrotic) changes coexist, and the consistency of the gingiva is determined by their relative predominance. **Texture**—In chronic inflammation, the gingival surface is smooth and shiny or firm and nodular, depending on whether the dominant changes are exudative or fibrotic, respectively. Hyperkeratosis results in a leathery texture. Loss of stippling occurs in patients who normally exhibit stippling. **Bleeding on probing (BOP)**—BOP appears earlier and is a more objective sign of gingivitis than color changes. While clinically healthy gingiva does not usually exhibit BOP, in gingivitis the severity of bleeding and the ease of its provocation depend on the intensity of inflammation. **Position**—Normally, the gingival margin is seen positioned at the level of the cementoenamel junction. Both gingival recession (exposure of the root surface by an apical shift in the position of the gingiva) and enlargement (coronal shift in the position of the gingiva) can be found in gingival inflammation, though the latter is more common as a direct consequence of gingivitis. *BOP*, bleeding on probing; *CEJ*, cementoenamel junction; *IDP*, interdental papilla. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

Necrotizing Gingivitis (NG)

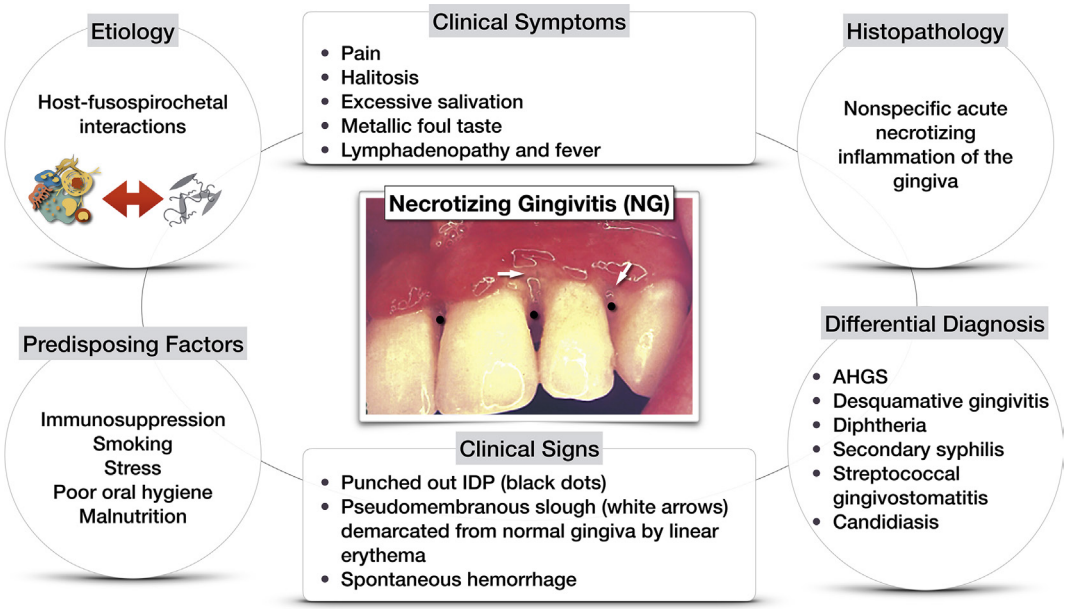
Although these lesions are not directly caused by plaque, their clinical course may be impacted by plaque accumulation and subsequent gingival inflammation. Key characteristics differentiating this acute condition from chronic gingivitis are:[3]

- Presence of pain;
- Central necrosis of the interdental papillae, resulting in considerable tissue destruction with formation of a crater-like soft tissue defect in proximal areas between teeth;
- Presence of a clear bacterial etiology, most likely a fusospirochetal infection showing a constant finding of microflora primarily containing *Treponema* spp., *Selenomonas* spp., *Fusobacterium* spp., and *Prevotella intermedia.* See Fig. 13.2 for a detailed description of NG.

CLINICAL CORRELATE

What are the reasons for the change in terminology from acute necrotizing ulcerative gingivitis (ANUG) to necrotizing gingivitis (NG)?

Despite necrotizing diseases often running a rapidly destructive course, the term "acute" has not been included in the diagnoses since 1999 because:[4]



• **Fig. 13.2** Necrotizing Gingivitis. Necrotizing gingivitis (NG) is a microbial disease of the gingiva that most often occurs in an impaired host with the characteristic clinical signs of necrosis and sloughing of the gingival tissues. **History:** NG is characterized by a sudden onset of symptoms, sometimes occurring after an episode of debilitating disease or acute respiratory tract infection. **Etiology and predisposing factors:** Although certain bacteria (e.g., fusospirochetal complex) are likely responsible for the lesions observed in necrotizing ulcerative gingivitis, immunocompromise appears to be a necessary predisposing condition for the disease. **Clinical symptoms:** The lesions are extremely sensitive to touch, and the patient often complains of a constant radiating, gnawing pain that is intensified by eating spicy or hot foods and chewing. There is a metallic foul taste, and the patient is conscious of an excessive amount of pasty saliva. **Clinical signs:** Characteristic punched-out, crater-like depressions covered by a pseudomembranous slough at the crest of the interdental papillae that subsequently extends to the marginal gingiva. **Histopathology:** The microscopic appearance of tissues in a NG lesion is nonspecific: (1) The surface epithelium is destroyed and replaced by a mesh of fibrin, necrotic epithelial cells, polymorphonuclear leukocytes (PMNs), and various microorganisms. This is the zone that appears clinically as the surface *pseudomembrane*. (2) The underlying connective tissue is markedly hyperemic, with numerous engorged capillaries and a dense infiltration of PMNs. This acutely inflamed zone appears clinically as the *linear erythema* beneath the surface pseudomembrane. (3) The layer between the necrotic and living tissue contains enormous numbers of fusiform bacilli and spirochetes, in addition to leukocytes and fibrin. Spirochetes and other bacteria invade the underlying living tissue. *AHGS*, acute herpetic gingivostomatitis; *IDP*, interdental papilla. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

- Studies have suggested that NG and necrotizing periodontitis (NP) may represent different stages of the same disease; they display similar etiology and clinical manifestations. They also respond to similar clinical interventions. Since they may progress to more severe forms such as necrotizing stomatitis and noma (cancrum oris), in 1999, both conditions were classified as necrotizing periodontal diseases (NPD). If the lesion is indeed capable of progression from gingivitis to periodontitis, the lesion cannot technically remain exclusively '*acute*'.
- NPD may frequently recur and cause it to become a chronic condition, albeit with a slower rate of destruction.

Because superficial necrosis always involves an ulcer, the use of the term "ulcerative" may be somewhat superfluous. Additionally, the terminology was eliminated because the ulceration was considered to be secondary to the necrosis.

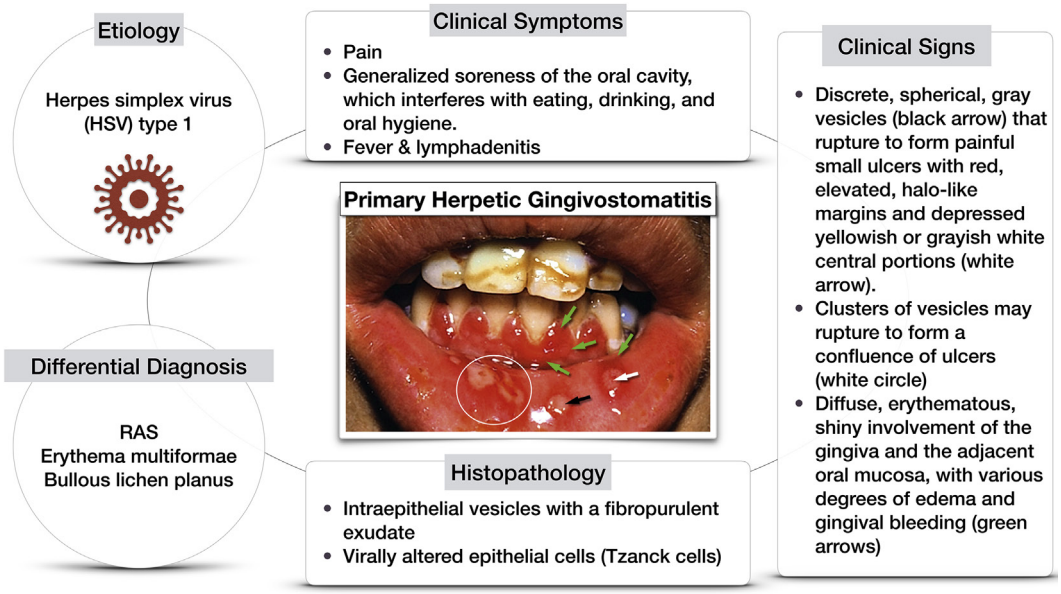
Note the additional former terminology:

- NUG, necrotizing ulcerative gingivitis, former terminology for NG, necrotizing gingivitis;
- NUP, necrotizing ulcerative periodontitis, former terminology for NP, necrotizing periodontitis.

Primary Herpetic Gingivostomatitis

Gingivitis caused by herpes simplex virus type 1 (HSV-1), is typically seen in infants with an incubation period of 1 week. In early childhood, it may also result in an asymptomatic infection or severe gingivostomatitis. These lesions are characterized by vesicle formation followed by their rupture and coalescence, leaving irregular, fibrin-coated ulcers.[3] Note that:

- Recurrent intraoral herpes simplex lesions are often mistaken for aphthous ulcerations. Aphthous ulcers however, do not typically affect keratinized mucosa.
- Secondary manifestations result from various stimuli (e.g., sunlight, trauma, fever, stress) and include herpes labialis, herpetic stomatitis, herpes genitalis, ocular herpes, and herpetic encephalitis. Secondary herpetic stomatitis can occur on the palate or gingiva, or on the mucosa as a result of dental treatment that traumatizes or stimulates the latent virus in the ganglia innervating the



• **Fig. 13.3** Primary Herpetic Gingivostomatitis. Acute herpetic gingivostomatitis usually occurs in infants and children because most adults have developed immunity to herpes simplex virus from childhood exposure, often with mild or no symptoms. **Etiology:** Primary herpetic gingivostomatitis is the result of an acute infection by HSV-1. **History:** There is an acute onset of symptoms. As part of the primary infection, the virus ascends through the sensory and autonomic nerves, where it persists as latent HSV in the neuronal ganglia that innervate the site. **Clinical signs and symptoms:** During its initial stage, it is characterized by discrete, spherical, gray vesicles, which can occur on the gingiva, labial and buccal mucosa, soft palate, pharynx, sublingual mucosa, and tongue. The ruptured vesicles are the focal sites of pain; they are particularly sensitive to touch, thermal changes, foods such as condiments and fruit juices, and the action of coarse foods. In infants, the disease is marked by irritability and refusal to take food. The course of the disease is limited to 7–10 days. Cervical lymphadenitis, fever as high as 101°–105°F (38°–40.6°C), and generalized malaise are common. **Histopathology:** The virus targets the epithelial cells, which become large, rounded keratinocytes with an area of acantholysis around them. These are called *Tzanck cells* after fusing together to form multinucleated cells. The intercellular edema leads to the formation of intraepithelial vesicles that rupture and develop a secondary inflammatory response with a fibropurulent exudate. *RAS, recurrent aphthous stomatitis*. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

area. It may manifest as pain away from the site of treatment 2–4 days later.

See Fig. 13.3 for a detailed description of primary herpetic gingivostomatitis.

See Table 13.1 for a summary of the differentiating features of NG and primary herpetic gingivostomatitis.

Pericoronitis and Pericoronal Abscess

The term *pericoronitis* refers to inflammation of the gingiva in relation to the crown of an incompletely erupted tooth. It occurs most often in the mandibular third molar area and can be acute, subacute, or chronic. When complicated by purulence, it can lead to pericoronal abscess with pus discharge. See Fig. 13.4 for details regarding this condition.

CASE-BASED LEARNING EXERCISE

Scenario: An 18-year-old female presented to clinic with a Chief Complaint "My gums bleed sometimes and my orthodontist asked me to see you before she starts her treatment." Background Information: Patient has no underlying medical condition that would affect her oral health. Her last dental cleaning was done 3 years ago. Patient reports brushing at least once daily but denies flossing. Patient reports sensitivity and bleeding of the gingiva in the mandibular anterior sextant while brushing. Visual exam revealed inflammation of the marginal gingiva, especially in the mandibular anterior sextant (image A).

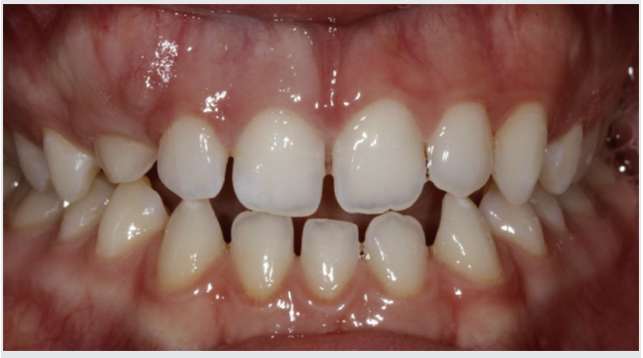
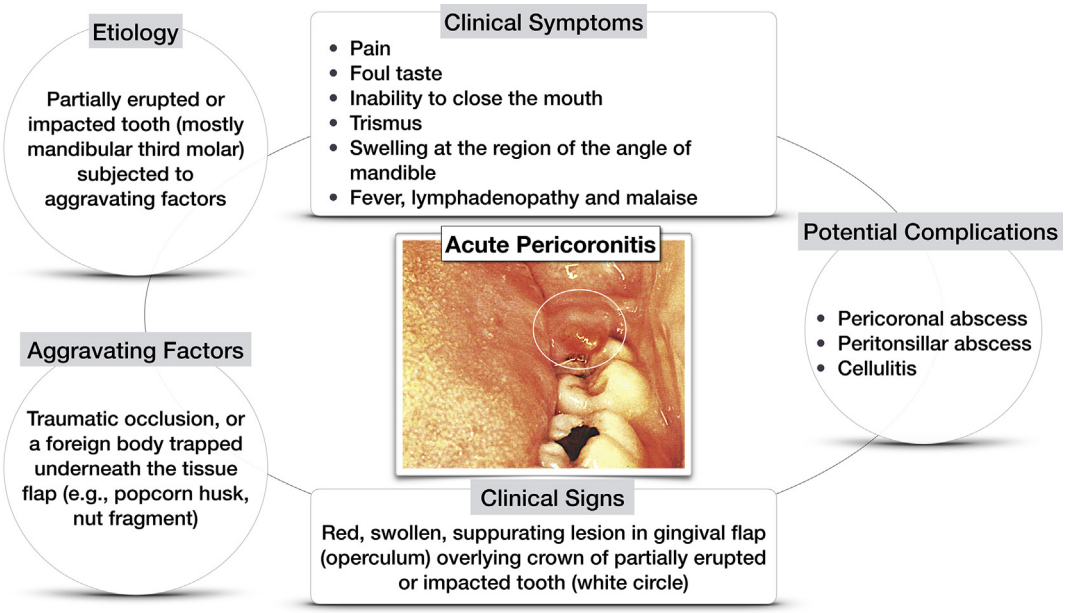


TABLE 13.1 Differences Between Necrotizing Gingivitis and Primary Herpetic Gingivostomatitis

	Necrotizing Gingivitis	Primary Herpetic Gingivostomatitis
Etiology	Caused by interaction between host and bacteria, most often fusospirochetes	Caused by specific viral infection (HSV-1)
Characteristic clinical feature	Necrosis	Diffuse erythema and vesicular eruption
Description of ulcer	Punched-out IDP and gingival margin; pseudomembrane that peels off and leaves raw areas	Vesicles that rupture and leave slightly depressed oval or spherical ulcer
Affected mucosa	Marginal gingiva affected; other oral tissues rarely affected	Diffuse involvement of gingiva; may include buccal mucosa and lips
Population	Uncommon in children	Occurs more frequently in children
Duration	No definite duration	Duration 7–10 days
Immune response	No conclusive demonstration of serum antibody response to implicated pathogens	Acute episode results in some degree of serum immune response
Disease spread	Contagion not demonstrated	Contagious

IDP, interdental papilla; HSV-1, herpes simplex virus type 1
Adapted from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). Newman and Carranza's Clinical Periodontology *(13th ed.). Philadelphia: Elsevier.*



• **Fig. 13.4** Acute Pericoronitis. Acute pericoronitis is identified by various degrees of inflammatory involvement of the pericoronal flap (operculum) and adjacent structures, and by systemic complications. The inflammatory fluid and cellular exudate increase the bulk of the flap, which can interfere with complete closure of the jaws. It also can be traumatized by contact with the opposing dentition, thereby aggravating the inflammatory involvement. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

Questions

1 The early signs of gingival inflammation that precede established localized gingivitis include which of the following?
a. Increased gingival crevicular fluid production.
b. Bleeding from the gingival sulcus on gentle probing.
c. Suppuration on gentle probing.
d. All of the above.
e. (a) and (b) only.

2 Localized marginal gingivitis usually:
a. Affects unattached terminal gingiva that surrounds the teeth like a collar in limited areas.
b. Affects unattached terminal gingiva that surrounds the teeth like a collar in the entire mouth.
c. Extends only to interpapillary areas.
d. Extends on the lingual surfaces only.

3 What is the most probable cause of this gingival condition?
   a. Extensive restorations.
   b. Dental plaque.
   c. Chemical injury.
   d. Uncontrolled diabetes.
4 Which of the following is not an established systemic modifier of plaque-induced gingivitis?
   a. Puberty.
   b. Malnutrition.
   c. Hypertension.
   d. Pregnancy.

This chapter was developed from Chapters 18 and 20 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: e

Explanation: Suppuration (i.e., discharge of purulence) on gentle probing is an uncommon early sign of gingivitis. Suppuration indicates active infection and is a common sign of gingival or periodontal abscess.

2. Answer: a

Explanation: Localized marginal gingivitis is typically restricted to marginal gingiva, the unattached terminal edge of gingiva that surrounds the teeth (like a collar) in localized areas of the dentition.

3. Answer: b

Explanation: In this patient with no known underlying systemic conditions and presenting with plaque and poor oral hygiene, dental plaque seems to be the primary cause.

4. Answer: c

Explanation: Except for hypertension, the listed conditions are modifying factors for plaque-induced gingivitis.

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14

Gingival Enlargement and Desquamative Gingivitis

Relevant Terminology

Terminology/Abbreviation	Explanation
acantholysis	Loss of cohesion between keratinocytes due to the breakdown of their intercellular desmosomal bridges. The cells remain intact but are no longer attached to each other; they tend to become rounded up (Tzanck cells), losing their normal prickly appearance, resulting in intraepithelial clefts, vesicles, and bullae. *(Greek: akantha, a thorn or prickle; lysis, loosening.)*
atrophic lesion	Characterized by thinning of epithelium, resulting in erythema
conditioned gingival enlargement	The existing biofilm-induced inflammation causes gingival enlargement that is exaggerated by systemic conditions (e.g., hormonal change during pregnancy, vitamin C deficiency).
desmosome	• A cell structure specialized for *cell-to-cell* adhesion. Because they also link intracellularly to the cytoskeleton, desmosomes provide mechanical strength to tissues. • Pemphigus is a severe autoimmune blistering disease affecting the gingiva and causing desquamative gingivitis. It is caused by autoantibodies that primarily target the desmosomal adhesion molecules desmogleins (Dsgs 1 and 3), which are required for intercellular adhesion between keratinocytes. "Intraepithelial splits" are seen histologically because of autoantibodies targeting the desmosomes between cells.
desquamative gingivitis (DG)	Desquamative gingivitis is not a specific disease entity. It is characterized by desquamation, ulceration, and intense erythema of free and attached gingiva. In addition to the gingiva, approximately 50% of patients may have lesions at other intraoral and extraoral sites.
direct immunofluorescence (DIF)	Laboratory technique used to diagnose certain oral pathologic diseases. Antibody-fluorophore conjugate molecules are used to target abnormal depositions or distributions of proteins in the biopsied tissue. The fluorophore emits light when it is stimulated under a fluorescence microscope.
enlargement	Any increase in size, irrespective of the underlying pathologic process.
epulis	Any growth on the gingiva, irrespective of its etiology.
fibroma	Benign tumor composed of fibrous or connective tissue.
fibrosis	A pathologic process in which disrupted wound healing is associated with defective fibroblastic cell proliferation, cell-to-cell interactions, cell-to-matrix interactions, and matrix deposition, and with an impaired immune system response.
gingival enlargement	A pathologic condition affecting gingiva, during which hyperplastic, hypertrophic, and fibrotic changes can be observed. The two terminologies "gingival enlargement" and "gingival overgrowth" are sometimes used interchangeably.
gingival fibromatosis	Form of gingival enlargement that affects gingival margin, interproximal gingiva, and attached gingiva. It is usually hereditary and sometimes linked to impaired physical development.
hemidesmosome	Cell structure forming one half of the desmosome (on the cell side) creating cell-to-basal lamina adhesions.
hyperplasia	A pathologic process that results in an increase in tissue volume due to an increase in cell numbers.

Continued

Relevant Terminology—cont'd

Terminology/Abbreviation	Explanation
hypertrophy	A pathologic process that results in an increase in tissue volume due to an increase in cell size
immune complex	• Antigen-antibody (Ag-Ab) complexes • Immune complex reactions (e.g., erythema multiforme) are caused by the activation of complement in response to Ag-Ab complexes that are deposited in tissues
indirect immunofluorescence (IIF)	Laboratory technique used to evaluate the presence of specific antibodies circulating in the patient's blood. An animal tissue (e.g., monkey esophagus) is incubated with the patient's serum, then antibody-fluorophore conjugate molecules are applied to detect specific antibodies binding to the animal tissue.
leukemia-associated gingival overgrowth	Leukemic gingival enlargement/overgrowth is characterized by diffuse enlargement of the gingiva and mucosa. The gingiva looks bluish-red and shiny. Connective tissues are infiltrated with mature, immature, and proliferating leukocytes.
Nikolsky sign	Separation of the superficial epidermal layer from the underlying dermal layer while a horizontal tangential force is applied. It indicates a cleavage of the dermal-epidermal junction and is often identified in vesiculobullous disorders (e.g., pemphigus vulgaris).
overgrowth	Usually refers to a situation where *hyperplasia* and *hypertrophy* occur simultaneously and when cellular involvement in hyperplasia most likely triggers the overgrowth.
papilloma	Epithelial tumor growing as a finger-like or wart-like protuberance from the gingival surface.
pregnancy tumor	A hormonally conditioned gingival inflammatory response to local irritation—not a neoplasm. Appearing during pregnancy, it is also called angiogranuloma.
pyogenic granuloma	A tumor-like gingival enlargement that is an exaggerated, nonspecific conditioned response to minor trauma.
stomatitis medicamentosa	Eruptions in the oral cavity resulting from sensitivity to drugs that have been taken by mouth or parenterally.
stomatitis venenata	Local reaction to the use of a medication in the oral cavity (e.g., stomatitis as a result of topical penicillin use); also called *contact stomatitis.*
vesiculobullous lesions	Characterized by fluid-filled blisters of varying sizes that rupture to leave surface erosions or ulcerations.

Fast Facts

Inflammatory enlargement due to gingivitis	Primarily caused by gingival inflammation without other contributing factors. The inflammation may be induced by trauma, impaction of a foreign substance, or biofilm. This enlargement can be observed in interproximal, marginal, or attached gingiva. It can possibly lead to periodontal abscess if inflammation is not controlled in the early stage.
Drug-induced gingival overgrowth (DIGO)	Anticonvulsants (e.g., phenytoin), calcium channel blockers (e.g., nifedipine) and immunosuppressants (e.g., cyclosporine) are the most common medications causing DIGO. The first sign of enlargement could be observed as early as 1–3 months. Gingival enlargement is often observed at the maxillary or mandibular anterior buccal area. Changing medications may effectively reverse gingival enlargement.
Histology of DIGO	Thick epithelial layers, elongated rete pegs, increased collagen matrix, and inflammatory cell infiltration are observed. Cyclosporin A–induced gingival overgrowth (GO) has more inflammatory infiltration and vascularization than other DIGO.
Cytokines and growth factors in GO lesions	Increased levels of interleukin-6 (IL-6), interleukin-1β (IL-1β), platelet-derived growth factor subunit B (PDGFB), fibroblast growth factor 2 (FGF2), transforming growth factor-β (TGF-β), and connective tissue growth factor (CTGF) are identified in GO lesions. The TGF-β1-CTGF axis that regulates fibroblast proliferation, differentiation, and extracellular matrix synthesis is involved in the pathogenesis of DIGO.
Treatment for DIGO	Withdrawal or substitution of medications is the most effective treatment. It is also important to receive periodontal maintenance, improve oral hygiene, and control local factors that induce inflammation. In cases where extensive GO affects esthetics or function, surgical excision (gingivectomy) is performed.
Pathogenesis of pregnancy-associated GO	Higher levels of progesterone and estrogen result in increased vascular permeability, leading to gingival edema and inflammatory responses, which cause GO.

Fast Facts—cont'd

Etiology of desquamative gingivitis (DG)	Lichen planus, cicatricial pemphigoid (i.e., mucous membrane pemphygoid) and pemphigus vulgaris are the most common causes of DG.
Clinical features of DG	• Pemphigus vulgaris—epidermal and mucous membrane blisters in the oral cavity, soft palate, and buccal mucosa are most frequent • Cicatricial pemphigoid—subepithelial blistering, involves the oral cavity, conjunctiva, other mucosa, and skin • Lichen planus—bilateral white striae on the buccal mucosa. Atrophic or erosive forms are associated with pain and burning sensation.
Treatment of DG	Rigorous plaque control is a necessary treatment. Using topical or systemic steroids may reduce symptoms. Based on the diagnosis, a dentist may refer the patient to other specialists (e.g., an ophthalmologist to treat the ocular lesion in cicatricial pemphigoid).

Core Knowledge

Introduction

The spectrum of periodontal diseases includes two distinctive gingival diseases with unique clinical presentations—gingival enlargement and gingival desquamation—that, like fever, cannot be a regarded as a "diagnosis" in the true sense of the word. This is because both conditions can be the clinical manifestation of a number of underlying local and systemic pathologic processes; they require careful history taking and evaluation before a diagnosis or treatment plan can be made. This chapter reviews the etiology, classification, pathogenesis, and clinical presentation of these two conditions.

Gingival Enlargement

Inflammation within the periodontal tissues may result in three types of outcomes:

1. Complete resolution of inflammation and restoration of tissue integrity (i.e., homeostasis)
2. Destruction of periodontal tissues and loss of attachment (i.e., periodontitis)
3. Fibrosis (a component of the defense mechanism that works against the progression of periodontal inflammation)

"Fibrosis" is a characteristic finding in in gingival enlargements, and may or may not be accompanied by edema and underlying osseous defects (e.g., periodontitis) or bony lesions (e.g., exostoses). It can be defined as a pathologic *lesion*, whereas hyperplasia and hypertrophy can be viewed as pathologic *processes*. In this chapter, for the sake of consistency, the term *gingival enlargement* will be mostly used to describe this clinical presentation (though *gingival overgrowth,* GO, is still an acceptable nomenclature and is also used occasionally).

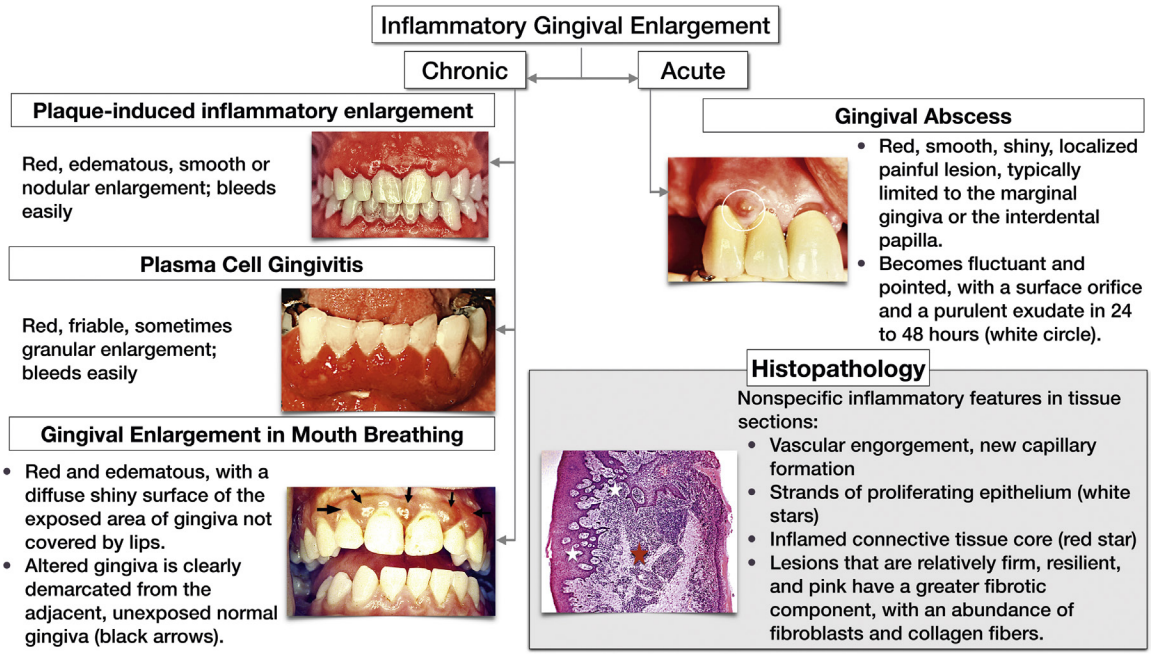
Classification of Gingival Enlargements

Classification of these conditions is based on their underlying etiology:

- **Inflammatory enlargements:**
  - chronic (e.g., plaque-induced inflammatory response, plasma cell gingivitis)
  - acute (e.g., gingival abscess due to trauma from toothbrush bristle)
- **Fibrotic enlargements:**
  - drug-influenced: due to use of anticonvulsants (phenytoin), calcium channel blockers (nifedipine) and immunosuppressants (cyclosporine)
  - idiopathic or hereditary gingival fibromatosis
- **Combined enlargements:**
  - (inflammatory + fibrotic)
- **Enlargements associated with systemic diseases/conditions:**
  - enlargements influenced by systemic conditions (e.g., pregnancy, puberty, scurvy)
  - enlargements caused by systemic diseases (e.g., granulomatous diseases like Wegener's granulomatosis and sarcoidosis)
- **Neoplastic enlargements:**
  - benign enlargements (e.g., due to fibroma, papilloma, giant cell granuloma, gingival cysts)
  - malignant enlargements (e.g., leukemia, squamous cell carcinoma)
- **False enlargements:**
  - due to underlying osseous lesions (e.g., tori, exostoses, Paget disease, osteosarcoma)
  - due to underlying developmental conditions (e.g., erupting tooth crown)

Diagnosis of Gingival Enlargements

- Enlargements may be localized (restricted to interdental papilla), generalized (includes marginal gingiva and interdental papilla), discrete (tumor-like, sessile or pedunculated), or diffuse (involves marginal, papillary, and attached gingiva).
- Indices are important for quantifying the extent and severity of gingival enlargements. The following is an example of an index scoring the degree of gingival enlargement:[1]
  - Grade 0: no signs of gingival enlargement
  - Grade I: enlargement confined to interdental papilla
  - Grade II: enlargement involves papilla and marginal gingiva
  - Grade III: enlargement covers three-fourths or more of the crown



• **Fig. 14.1** Inflammatory Gingival Enlargement: Clinical Presentation and Histopathologic Features. Inflammatory gingival enlargement originates as a slight ballooning of the interdental papilla and marginal gingiva, producing swelling around the involved teeth, which can increase in size until it covers part of the crowns. The enlargement progresses slowly and painlessly unless it is complicated by acute infection or trauma. Inflammatory enlargement can be acute (gingival abscess due to trauma) or chronic (dental biofilm–induced inflammatory enlargement, plasma cell gingivitis, gingival enlargement associated with mouth breathing habit and incompetent lips), but the common histologic presentation is a picture of nonspecific inflammation, with only plasma cell gingivitis showing the underlying connective tissue infiltrated densely with plasma cells that also extend to the oral epithelium. From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

Types of Gingival Enlargement

As discussed in the classification section, there are many types of gingival enlargement. In general, the degree of inflammation, fibrosis, and cellularity depends on many factors, the most important of which are:

- oral hygiene
- drug duration, dose, and chemistry
- individual susceptibility based on genetic and environmental influences

The following review is restricted to the most important and distinctive types of the condition.

Inflammatory Enlargement of the Gingiva

While all changes in gingival tissues manifest with some degree of inflammation, in some cases gingival enlargement is a direct outcome of gingivitis without any complicating factors or involvement of systemic conditions. See Fig. 14.1 for a review of the common clinical features and histopathology of this condition.

Drug-Influenced Gingival Enlargement

This condition is also called drug-induced gingival overgrowth (DIGO). It is a tissue-specific lesion with gingival enlargement that occurs in approximately one-third to one-half of the people who take the drugs phenytoin (anticonvulsant), nifedipine (calcium channel blocker), or cyclosporin A (immunosuppressant) on a regular basis. Phenytoin-influenced lesions are the most fibrotic, cyclosporine-influenced lesions are highly inflamed and exhibit little fibrosis, while nifedipine-influenced lesions are mixed.

Table 14.1 lists the most common drugs that influence gingival enlargement and describes their differentiating features.

Pathogenesis of Drug-Influenced Gingival Enlargement

The complex pathogenesis of drug-influenced gingival enlargements (Fig. 14.2) is mainly mediated through defective functioning of the gingival fibroblasts responsible for matrix deposition within gingival tissues. The functional defects include:

- Increasing the production of matrix proteins
- Decreasing collagenase activity, so that less destruction of collagen fibers occurs during tissue renewal and turnover, allowing their accumulation within connective tissue

CLINICAL CORRELATE

What is the need to study the cellular and molecular mechanisms underlying the pathogenesis of drug-influenced gingival enlargements?

There is a critical need for better understanding of the cellular and molecular mechanisms that drive the tissue specificity of drug-influenced gingival enlargement because:[2]

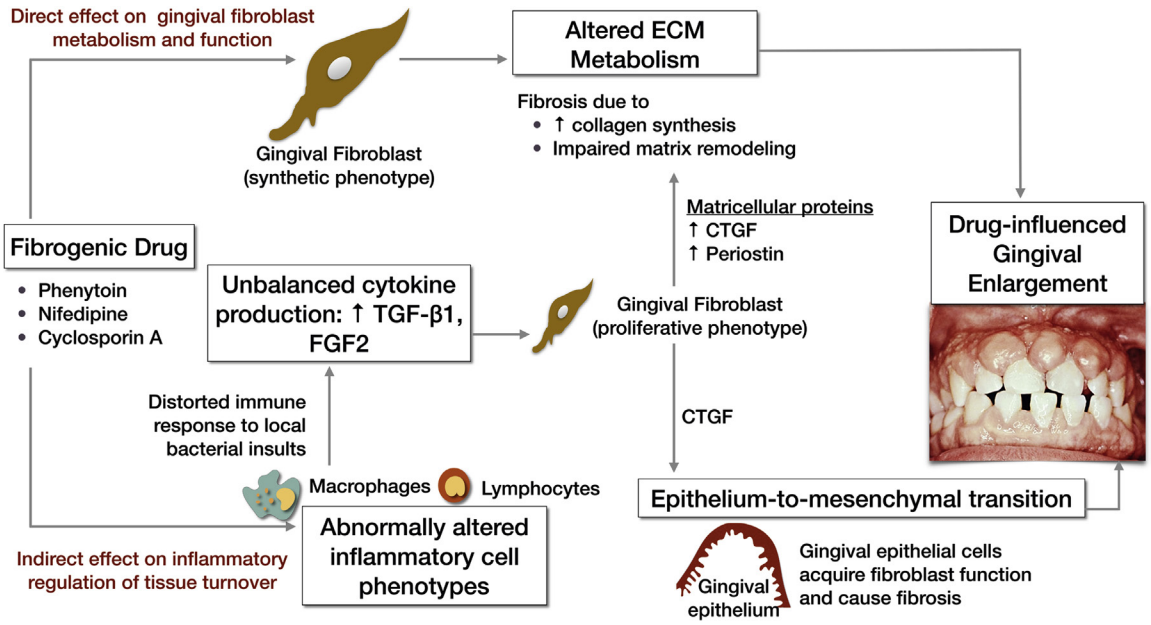
- Gingival enlargement interferes with proper oral hygiene maintenance and subsequent plaque retention. It increases risk of systemic inflammatory complications. This is particularly problematic in cases of epilepsy, cardiovascular disease or organ transplantation. When drug substitution is not feasible in such individuals, enlarged lesions require repeated surgical interventions due to the high recurrence rate caused by continued use of the drug.
- Gingival enlargement limits quality of life and gaining insights into novel cellular/molecular pathways unique to this condition may allow conservative management of this condition in future as well as enhance knowledge regarding other connective tissue disorders affecting the oral cavity [e.g. neurofibromatosis, metastatic oral cancer, etc] Current knowledge points to interference of TGF-β induction of CTGF as potential therapeutic target to reduce fibrotic overgrowth.

DIGO, drug-induced gingival overgrowth; *TGF*, transforming growth factor; *CTGF* (also called CCN2), connective tissue growth factor.

TABLE 14.1 Drug-Influenced Gingival Enlargements: Different Forms and Differentiating Features

	Drug Categories Commonly Influencing Gingival Enlargements		
	Anticonvulsants or Antiepileptics	**Antihypertensives (Calcium Channel Blockers)**	**Immunosuppressants**
Drug name	Phenytoin	Nifedipine	Cyclosporin A
Drug indications	• Drug of choice for the treatment of grand mal, temporal lobe, and psychomotor seizures	• Hypertension, angina pectoris, coronary artery spasm, and cardiac arrhythmia	• Drug of choice for preventing rejection of transplanted solid organs and bone marrow • Autoimmune conditions
Prevalence of gingival enlargements in drug users	50%	6%–83%	30% (higher in children)
Other drugs in same group capable of influencing gingival enlargement	• Phenobarbital, valproic acid	• Benzodiazepine derivatives (diltiazem) • Phenylalkylamine derivatives (verapamil) • Dihydropyridines (amlodipine, felodipine, etc.)	–
Clinical presentation	• Rapid clinical onset, as early as one month of drug usage • Anterior maxillary/ mandibular regions affected most • Enlarged interdental papilla with pebbly surface • Thickened marginal gingiva	• Anterior maxillary/mandibular segments affected most • Enlarged interdental papillae • Tissue overgrowth restricted to marginal and attached gingiva, not extending beyond mucogingival junction	• Enlargement commonly restricted to buccal gingival surfaces • More inflamed than other forms of DIGO • Gingiva bleeds more readily than in other forms of DIGO
Effect on bone loss	Less bone loss seen compared to other forms of DIGO due to "resistance" to further bone destruction	Can coexist with periodontitis and attachment loss that is different from other forms of DIGO	–
Histopathology	• Thick, stratified squamous epithelium with long, thin rete pegs extending deep into the connective tissue • Fibrosis and expanded connective tissue components	• Thick, stratified squamous epithelium with long, thin rete pegs extending deep into the connective tissue • Fibrosis and expanded connective tissue components	• Thickened epithelium, rete peg formation, and irregular collagen fibers • Characterized by more inflammatory infiltration and vascularization compared with phenytoin and calcium channel blockers
Inflammation	Moderate	Moderate	High
Fibrosis and CTGF in DIGO lesions[3]	High	Moderate	Low

DIGO, drug-induced gingival overgrowth; CTGF, connective tissue growth factor.



• **Fig. 14.2** Drug-Influenced Gingival Enlargement: Proposed Pathogenic Pathways [2,3]. Drug-influenced gingival enlargement is a consequence of interactions between gingival fibroblasts, cellular and biochemical inflammatory mediators, and the drug or its metabolites. Fibroblast heterogeneity (variations in fibroblast distribution and phenotype) may contribute to drug-influenced and inherited forms of gingival enlargements. A decrease in gingival fibroblasts undergoing apoptosis (along with perhaps a preponderence towards synthetic/proliferative phenotypes) allows their accumulation within tissues in DIGO. Fibrogenic drugs (phenytoin, nifedipine and cyclosporin A) may work in two possible ways to induce GO:

1. **Via direct effects on gingival fibroblasts' metabolism and function:** Here the drug (e.g., cyclosporin A) directly affects collagen synthesis by gingival fibroblasts with a concomitant rise in the levels of type I collagen.
2. **Via indirect effects on inflammatory regulation of tissue turnover:** Here the fibrogenic drugs (phenytoin, nifedipine and cyclosporin A) are said to alter the phenotypes of inflammatory cells (mainly lymphocytes and macrophages), resulting in distorted immune responses to periodontal bacterial insults. This leads to secretion of a subset of cytokines that influence ECM metabolism and proliferation. Increased FGF2 stimulates gingival fibroblast proliferation. The TGF-β1–CTGF axis pathway directly regulates fibrosis, gingival fibroblast lysyl oxidase (an enzyme that catalyzes the final step in cross-linking collagen and elastin in the synthesis of a functional extracellular matrix), and collagen generation. CTGF is a reliable marker of fibrosis and contributes to the development of fibrosis initiated by TGF-β1. Periostin, like CTGF, is a matricellular protein that contributes to fibrosis and is upregulated in nifedipine-induced GO. In addition, human gingival epithelial cells undergo functional and genetic epithelium-to-mesenchymal transition in response to TGF-β1. This conversion is regulated by CTGF and results in fibrosis due to epithelial cells additionally performing the function of fibroblasts. *CTGF* (also called CCN2), connective tissue growth factor; *DIGO*, drug-induced gingival overgrowth; *ECM*, extracellular matrix; *FGF2*, fibroblast growth factor; *GO*, gingival overgrowth; *TGFβ1*, transforming growth factor. *Matricellular proteins* are a family of nonstructural matrix proteins that regulate a variety of biologic processes in normal and pathologic situations. From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

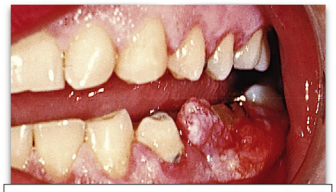
Other Clinically Distinctive Forms of Gingival Enlargement Associated With Systemic Conditions and Diseases

This section briefly describes the clinical and histopathologic features of the gingival enlargements associated with systemic and/or hereditary conditions and diseases. Though there are many underlying causes for gingival enlargements in these categories, the review is restricted to the more clinically distinctive types (Figs. 14.3 and 14.4).

Desquamative Gingivitis

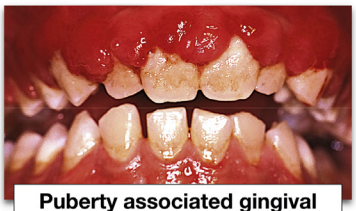
Desquamative gingivitis is not strictly a diagnosis, but rather a clinical presentation that may be due to a number of underlying reasons. The diagnostic procedure following the discovery of a desquamative gingivitis lesion must involve:

- Thorough medical history
- Clinical examination
- Histopathology (hematoxylin and eosin staining) and immunofluorescence (direct or indirect) of biopsied tissues from lesions



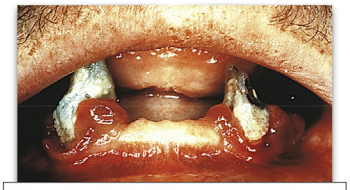
Pregnancy associated gingival enlargement

Clinical Features
- Single mass (pregnancy tumors) or multiple tumor-like masses at the gingival margin, sessile or pedunculated
- Bright red or magenta, soft, friable, with bleeding on slight provocation
- Flattened appearance due to pressure from the tongue and the cheek
- Usually painless unless ulcerated due to debris collection or traumatic occlusion



Puberty associated gingival enlargement

Clinical Features
- Prominent bulbous interproximal papillae
- Often, only the facial gingivae are enlarged, and the lingual surfaces are relatively unaltered.



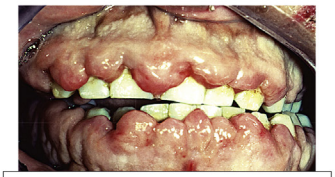
Vitamin C deficiency associated gingival enlargement

Clinical Features
- Not very common anymore
- Bluish red marginal gingival enlargements with smooth, shiny surface
- Spontaneous hemorrhage or bleeding on slight provocation
- Surface necrosis with pseudomembrane formations

Histopathology

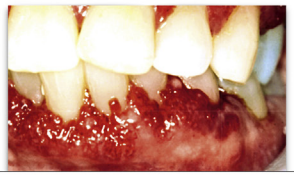
Non-specific chronic inflammatory features are common in all 3 types of lesions, with certain characteristic features for each condition:
- Prominent vascular changes in pregnancy (called *angiogranuloma*)
- Prominent edema in puberty
- Prominent collagen degeneration in vitamin C deficiency

• **Fig. 14.3** Conditioned Gingival Enlargements. Conditioned enlargements include lesions associated with hormonal and nutritional etiologic factors. Gingival inflammation due to microbial factors is a requirement that is further influenced by nutritional and hormonal changes—and some researchers classify these lesions as gingivitis-associated pathologies. From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.



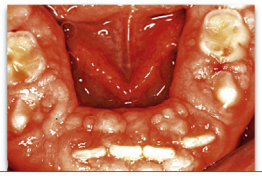
Leukemic gingival enlargement

Clinical Features
- Diffuse enlargement or discrete tumor-like interproximal mass
- Moderately firm with spontaneous hemorrhage
- Acute painful necrotizing ulcerative inflammatory involvement can occur in the crevice formed at the junction of the enlarged gingiva and the contiguous tooth surfaces.
- True leukemic enlargement often occurs with acute leukemia, but seldom with chronic leukemia



Wegener's Granulomatosis

Clinical Features
- Granulomatous papillary enlargement
- Reddish purple and bleeds easily on stimulation



Gingival Fibromatosis

Clinical Features
- Pink, firm, and almost leathery in consistency, with a characteristic minutely pebbled surface
- Teeth are almost completely covered
- Can be hereditary or idiopathic

Histopathology

Nonspecific chronic inflammatory features are common in all 3 types of lesions, with certain characteristic features for each condition:
- Leukemia: Various degrees of leukocytic infiltration; isolated areas with with a pseudomembranous meshwork of fibrin, necrotic epithelial cells, polymorphonuclear leukocytes, and bacteria are often seen.
- Wegener's granulomatosis: scattered giant cells, small blood vessels and microabscesses covered by a thin, acanthotic epithelium.
- Gingival fibromatosis: relatively avascular connective tissue with densely arranged collagen bundles and numerous fibroblasts. The surface epithelium is thickened and acanthotic, with elongated rete pegs

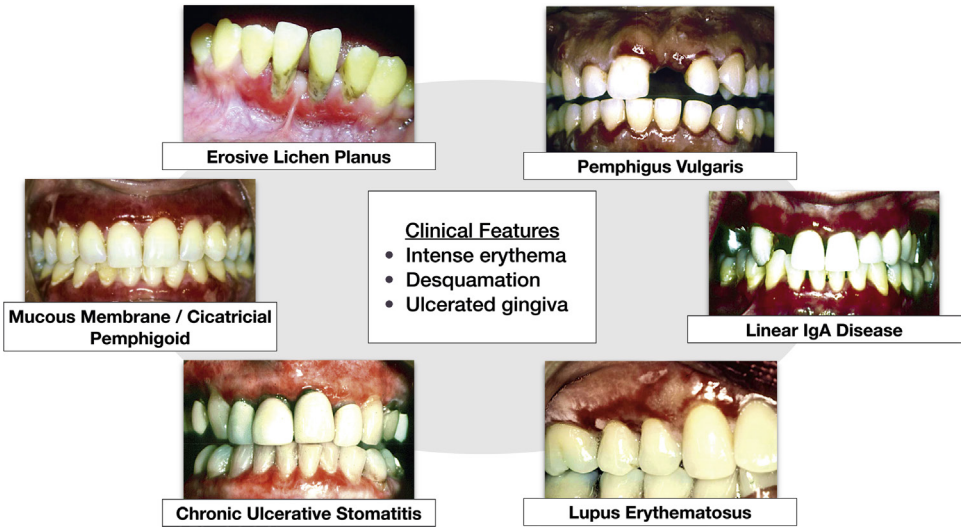
• **Fig. 14.4** Gingival Enlargements Associated With Systemic Diseases. From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

BASIC SCIENCE CORRELATE

What is the "immunopathology" of oral desquamative gingival diseases?

Mucosal integrity and continuity is maintained by adhesion proteins that bind a cell-to-cell or a cell-to-matrix. Immune-inflammatory diseases that destroy epithelial cells [keratinocytes] or adversely affect cell adhesion to the subjacent basement membrane-connective tissue complex (or even to another cell) lead to discontinuities in mucosal structure that present as erosions, ulcerations or desquamations. The disease processes that cause such immunologically mediated tissue destruction include:[4]

- T cell–mediated hypersensitivity (lichen planus)
- Humoral-mediated immunity to cadherin intercellular adhesion molecules (a component of the desmosomal junctions between epithelial cells) that is important in the process of acantholysis (pemphigus vulgaris)
- Genetic defects and antibody-mediated processes that give rise to junctional separation (epidermolysis bullosa and mucous membrane pemphigoid, respectively)
- An immune complex mechanism (erythema multiforme)



• **Fig. 14.5** Clinical Features of Desquamative Gingivitis. The figure shows some of the more common systemic diseases that may manifest as desquamative gingivitis; most may or may not be accompanied by pain and extraoral lesions. As all the clinical photos show, all conditions share the same nonspecific features of intense erythema, desquamation, and ulceration of the gingiva, so a biopsy of affected tissue followed by histopathologic and immunofluorescence studies is extremely important to establish the correct diagnosis and prognosis, and to plan therapy. Most of the conditions are treated by helping the patient maintain good oral hygiene and prescribing topical steroids; severe cases are managed by interdisciplinary care that involves medical specialists (e.g., dermatologists, opthalmologists). From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

Underlying Etiology

Approximately 75% of desquamative gingivitis cases have a dermatologic origin, of which more than 95% are accounted for by *cicatricial (mucous membrane) pemphigoid* or *lichen planus*. Other conditions that may cause desquamative gingivitis and must be included in the differential diagnosis are:

- pemphigus vulgaris
- chronic ulcerative stomatitis
- bullous pemphigoid
- linear immunoglobulin A (IgA) disease
- lupus erythematosus
- erythema multiforme
- dermatitis herpetiformis
- drug eruptions
- graft-versus-host disease

Clinical Presentation

- This peculiar condition is characterized by nonspecific features like intense erythema and desquamation (sometimes preceded by vesiculobullous lesions that slough off to produce raw, ulcerated surfaces) of the free and attached gingiva.
- Patients may be asymptomatic; when symptomatic, however, their complaints range from a mild burning sensation to an intense pain.
- Extraoral lesions may also be present (Fig. 14.5).

Biopsy

It is of great importance to identify the disease responsible for desquamative gingivitis in order to establish the appropriate therapeutic approach. To this end, the medical history and clinical examination must be supplemented with histologic and immunofluorescence studies—although even with biopsy examinations, up to one-third of desquamative gingivitis lesions remain unexplained by any clear underlying etiology.

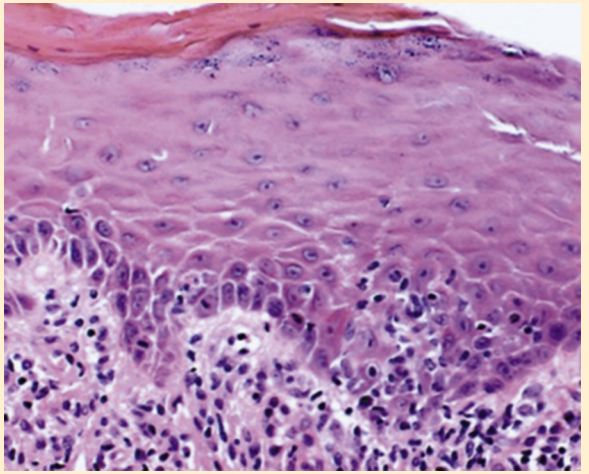
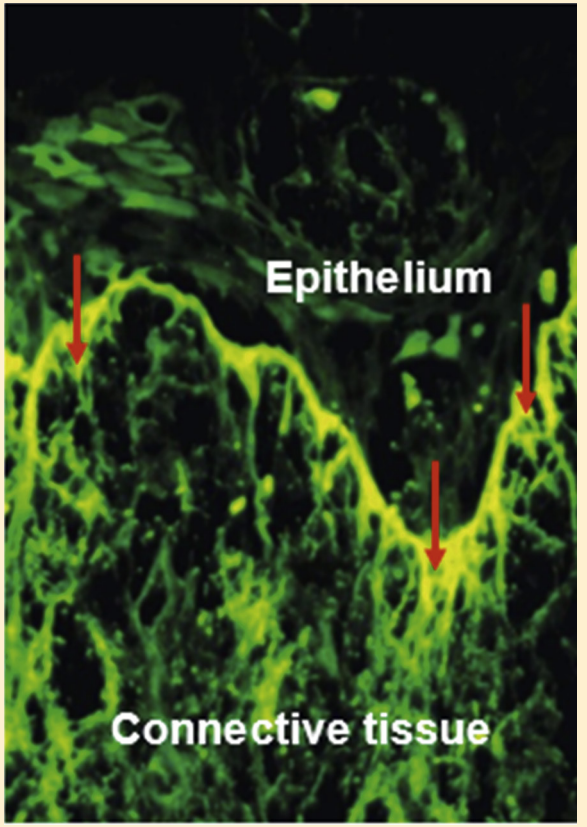
Biopsy procedure guidelines:

- Selection of the biopsy site—a perilesional incisional biopsy should be done to avoid areas of ulceration, because necrosis and epithelial denudation severely hamper the diagnostic process.
- Evaluation of biopsied tissue—after the tissue is excised from the oral cavity, the specimen can be bisected and submitted for microscopic examination by two methods:
  1. **Histologic evaluation:** buffered formalin (10%) should be used to fix the tissue for conventional hematoxylin-eosin (H&E) evaluation
  2. **Immunofluorescence studies:** includes both direct (DIF) and indirect (IIF) studies:
     - DIF is performed on biopsied specimens that are placed in a special medium (not formalin) called Michel's buffer (ammonium sulfate buffer, pH 7.0) for transport. It is a cell imaging technique that relies on the use of antibodies to label a specific target antigen in the biopsied tissue with a fluorescent dye. DIF uses a single antibody directed against the antigen of interest.
     - IIF is not performed on the biopsied tissue but uses serum from the patient's blood specimen. IIF detects *circulating* antibodies against target antigens.

Note: Here, the term "target antigens" refers to specific structural proteins in the gingival tissue targeted by the patient's own immune system (antibodies).

Table 14.2 describes the histopathologic and immunofluorescence (direct and indirect) findings in select conditions that may present clinically as desquamative gingivitis.

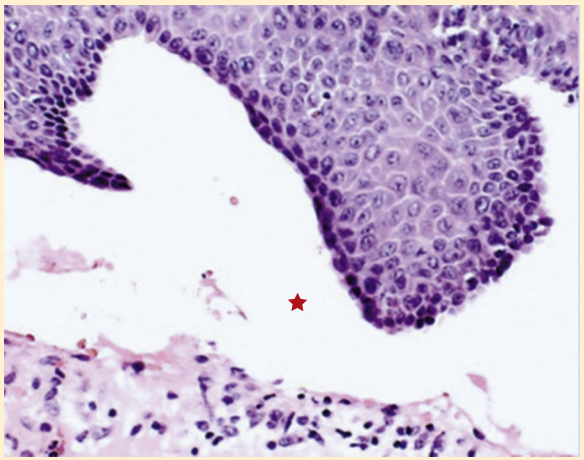
TABLE 14.2 **Histopathologic and Immunofluorescence Findings in Tissue and Blood Specimens of Patients With Desquamative Gingivitis**

Disease	Histopathology Findings	DIF Findings	IIF Findings
Lichen planus (LP)	• Hydropic/liquefaction degeneration of the basal layer • Sawtooth rete pegs • Dense, bandlike infiltrate, primarily of T lymphocytes in lamina propria • Colloid bodies (Civatte bodies) are present at the epithelium–connective tissue interface 	• Linear, fibrillar ("shaggy") deposits of fibrin at the dermal-epidermal junction (red arrows) • Immunoglobulin staining of scattered cytoid bodies in the upper areas of the lamina propria 	• Negative

Continued

TABLE 14.2 **Histopathologic and Immunofluorescence Findings in Tissue and Blood Specimens of Patients With Desquamative Gingivitis—cont'd**

Disease	Histopathology Findings	DIF Findings	IIF Findings
Cicatricial / mucous membrane pemphigoid	• Subepithelial clefting (red star) • An intact basal cell layer remains attached to the epithelium	• Linear deposits of C3 (red arrows) with or without IgG at the basement membrane zone in almost all cases	• Basement membrane zone (IgG) antibodies in 10% of cases



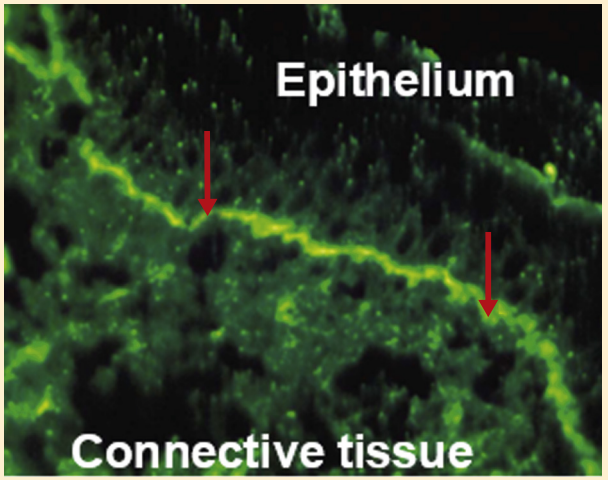
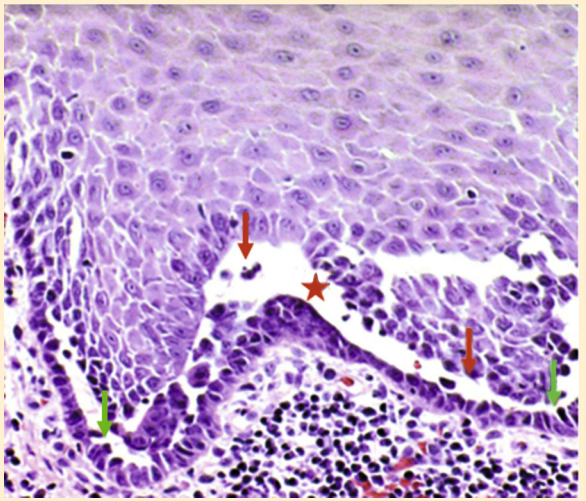
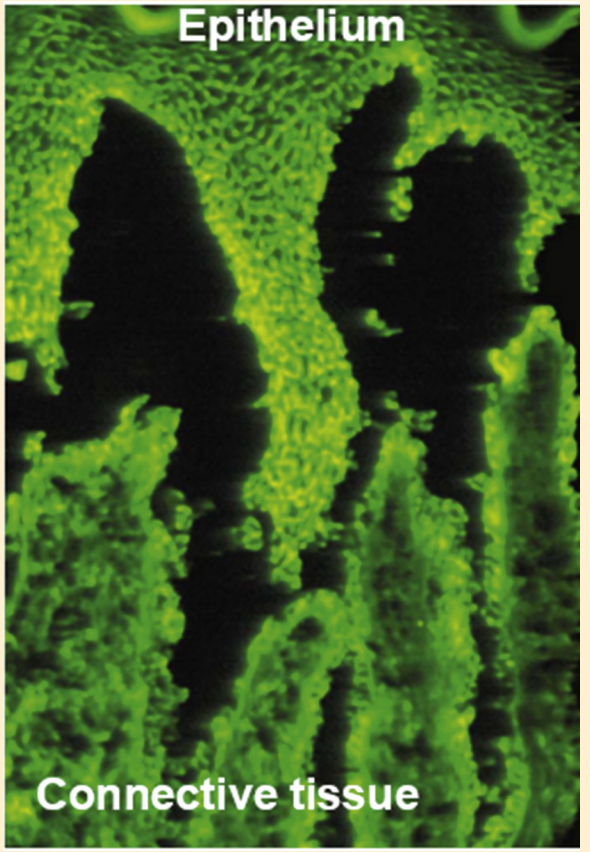


TABLE 14.2 **Histopathologic and Immunofluorescence Findings in Tissue and Blood Specimens of Patients With Desquamative Gingivitis—cont'd**

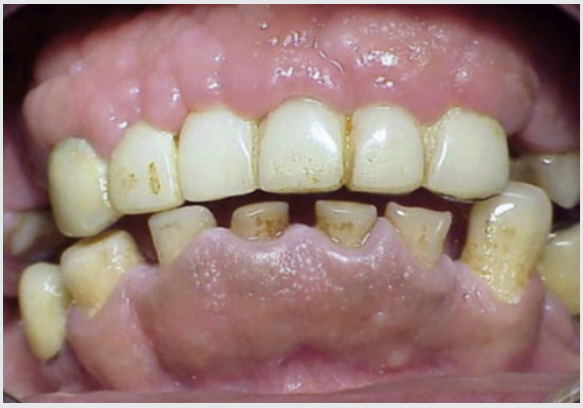
Disease	Histopathology Findings	DIF Findings	IIF Findings
Pemphigus vulgaris (PV)	• Intraepithelial clefting (red star) above the intact basal cell layer which remains attached to the connective tissue • Basal cells have a characteristic "tombstone" appearance (green arrows) • Acantholysis is present, with Tzanck cells in the cleft (red arrow) 	• Intercellular deposits of IgG in all cases, and C3 in most cases, within epithelium 	• Intercellular (IgG) antibodies in ≥90% of cases

C3, complement 3 deposits; DIF, direct immunofluorescence; IgG, immunoglobulin G; IIF, indirect immunofluorescence; Tzanck cells, rounded epithelial cells, (different from the usual polyhedral keratinocytes), that have lost their intercellular bridges.

Adapted from Table 22.1 in Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). Newman and Carranza's Clinical Periodontology *(13th ed.). Philadelphia: Elsevier. Figures from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019).* Newman and Carranza's Clinical Periodontology *(13th ed.). Philadelphia: Elsevier.*

CASE-BASED LEARNING EXERCISE

Scenario: A 29-year-old female patient presented with the chief complaint "My gums are swollen and my teeth move." Patient was not taking any medications. Intraoral examination revealed moderate to severe gingival overgrowth (GO), particularly in the mandibular anterior areas. Generalized significant periodontal probing depths (5–6 mm) and generalized slight horizontal bone loss were observed in the radiographs. Patient reported that her mother and younger brother both had similar GO.



Questions

1. Based on the history and clinical exam, what is your preliminary diagnosis?
   - **a.** Drug-induced gingival overgrowth
   - **b.** Hereditary gingival fibromatosis
   - **c.** Idiopathic gingival overgrowth
2. What would be the most likely treatment for phase 2 to address the periodontal condition?
   - **a.** Resective therapy
   - **b.** Guided tissue regeneration
   - **c.** Free gingival graft
   - **d.** Implant treatment
3. Based on the clinical presentation, this is a _____ degree of gingival enlargement on the buccal aspect of mandibular central incisors.
   - **a.** Grade 0
   - **b.** Grade I
   - **c.** Grade II
   - **d.** Grade III
4. What is the most common form of gingival overgrowth?
   - **a.** Drug-induced gingival overgrowth
   - **b.** Hereditary gingival fibromatosis
   - **c.** Idiopathic gingival overgrowth

Clinical images are from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

This chapter was developed from Chapters 19 and 22 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Solutions

1. Answer: b

Explanation: Hereditary gingival fibromatosis is an induced fibrotic form of gingival overgrowth that is not related to medications but has strong familial linkage.

2. Answer: a

This is a complex case of hereditary gingival fibromatosis. Treatment planning should be rendered by a multidisciplinary approach. Of the options presented, resective therapy (gingivectomy) will most likely be recommended to correct the gingival fibromatosis.

3. Answer: c

Explanation: The degree of gingival enlargement can be scored as follows:[1]

- Grade 0: no signs of gingival enlargement
- Grade I: enlargement confined to interdental papilla
- Grade II: enlargement involves papilla and marginal gingiva
- Grade III: enlargement covers three-fourths or more of the crown

4. Answer: a

Explanation: Most common forms of gingival enlargement result from systemic use of various medications, particularly anticonvulsants, calcium channel blockers, and immunosuppressants.

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1. Buchner, A., & Hansen, A. S. (1979). The histomorphologic spectrum of the gingival cyst in the adult. *Oral Surgery, Oral Medicine, and Oral Pathology*, *48*, 532.
2. Trackman, P. C., & Kantarci, A. (2015). Molecular and clinical aspects of drug-induced gingival overgrowth. *Journal of Dental Research*, *94*, 540–546.
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15 Periodontal Pocket, Bone Loss, and Patterns of Bone Loss

Relevant Terminology

Terminology / Abbreviation	Explanation
Cytokines	• Proteins secreted by cells leading to specific cellular responses • Can be proinflammatory (e.g., IL-1, TNF-α) or antiinflammatory (e.g., IL-4, IL-10) in nature
gingival pocket	Also called pseudopocket. It is formed by gingival enlargement and sulcus elongation without periodontal tissue destruction, and is usually induced by inflammation or medications (as in drug-induced gingival enlargements).
gingival sulcus	The shallow fissure between the circumferential gingiva and the tooth surface. When sulcus deepens due to the apical migration of junctional epithelium, it becomes a periodontal pocket. Therefore, a pocket is a pathologic counterpart of sulcus.
horizontal bone loss	The most common bone loss pattern in periodontitis. The bone level is reduced and remains approximately perpendicular to the tooth surface.
intrabony periodontal pocket	Also called infrabony, subcrestal, or intraalveolar periodontal pocket. The base of the pocket and the associated inflammatory responses are located apical to the underlying alveolar bone crest.
matrix metalloproteinases (MMPs)	Group of proteinases (enzymes that degrade proteins) involved in degradation of extracellular matrix that is composed primarily of collagen.
osseous craters	Specific type of two-wall defect characterized by concavities in the interproximal bone crest. Craters make up about one-third of all bony defects.
periodontal abscess	• A localized inflammation with suppuration in the periodontal tissues • Also known as a lateral abscess or a parietal abscess • Usually caused by the extension of infection from the periodontal pocket • Microorganisms that colonize the periodontal abscess have been reported to be primarily gram-negative anaerobic rods
periodontal pocket	A pathologically deepened gingival sulcus with underlying periodontal tissue destruction. It can be classified into *suprabony pocket* or *intrabony pocket*, based on the apicocoronal location of the base of the sulcus in relation to the alveolar crest.
radius of action	• Refers to a range of effectiveness of about 1.5–2.5 mm in which bacterial plaque can induce loss of bone • If bacterial biofilm is more than 2.5 mm away from the alveolar bone, there might be little or no bone loss • Interproximal angular defects can appear only in spaces that are wider than 2.5 mm, because narrower spaces would be destroyed entirely
reversed (negative) architecture	Situation in which the level of interdental bone is more apical than the level of radicular bone. It is the reversed pattern of the normal architecture.

Relevant Terminology—cont'd

Terminology / Abbreviation	Explanation
site specificity of periodontal disease	Characteristic phenomenon of periodontal disease in which: • Periodontal destruction does not occur in all parts of the mouth at the same time • Sites of periodontal destruction are often found right next to sites with little or no destruction
suprabony periodontal pocket	Also called supracrestal or supraalveolar periodontal pocket. The base of the pocket and the associated inflammatory responses are located coronal to the underlying alveolar bone.
tissue inhibitors of metalloproteinases (TIMPs)	Proteins that inhibit the activity of *MMPs*. The balance between MMPs and TIMPs is very important for extracellular matrix remodeling. Excessive MMPs in periodontal tissues may result in attachment loss and pocket formation.
vertical (angular) defects	Vertical defects have bone loss in an oblique direction and are surrounded by bony walls. Angular defects usually have intrabony periodontal pockets.

Fast Facts

Pocket Formation	The periodontal pocket forms as a result of deepening of the gingival sulcus due to apical migration of sulcus base and/or coronal movement of the gingival margin. These changes are usually induced by inflammation.
Microbial changes in the transition from normal gingival sulcus to the pathologic periodontal pocket	• Healthy gingival sulcus is associated with few microorganisms—mostly coccoid cells and straight rods • Periodontal pocket is associated with increased numbers of spirochetes and motile rods • However, the microbiota of diseased sites cannot be used as a predictor of future attachment or bone loss
Two mechanisms associated with collagen loss during pocket formation	• Enzymatic destruction of extracellular collagen by collagenases and other enzymes • Phagocytosis of collagen fibers by fibroblasts via cytoplasmic processes that extend into the ligament-cementum interface and degrade the inserted collagen fibrils and the fibrils of the cementum matrix
Microtopography of gingival pocket wall	Scanning electron microscopy has identified several areas in the soft tissue (gingival) wall of the periodontal pocket. There are areas of: • Relative quiescence • Bacterial accumulation • Emergence of leukocytes • Leukocyte-bacteria interaction • Intense epithelial desquamation • Ulceration • Hemorrhage
Surface morphology of tooth wall of the pocket	The following zones can be found in the periodontal pocket as we move in a coronoapical direction along the tooth wall of the pocket: 1. Cementum covered by calculus 2. Zone of attached plaque, which covers calculus 3. Zone of unattached plaque 4. Zone of attachment of the junctional epithelium to the tooth 5. Zone of semidestroyed connective tissue fibers
Periodontal pocket contents	• Periodontal pockets contain debris that primarily consists of bacteria and their products (e.g., endotoxins), gingival fluid, salivary mucin, food remnants, desquamated epithelial cells, and leukocytes • Purulent exudate, if present, consists of living, degenerated, and necrotic leukocytes, living and dead bacteria, serum, and a scant amount of fibrin
Periodontal disease activity	Periodontal pockets go through periods of exacerbation (enhanced loss of collagen and alveolar bone, deepened pockets) and quiescence (a reduced inflammatory response and decreased rate of tissue loss).
Bone loss in periodontitis	Bone loss is the ultimate consequence of periodontitis and is the result of the extension of gingival inflammation into the periodontal tissues.
Rate of bone loss	In patients with untreated periodontitis, the bone loss rate can be categorized into[1] • Rapid (approximately 8% of subjects) • Moderate (approximately 81% of subjects • Minimum or no progression (remaining 11% of subjects)

Continued

Fast Facts—cont'd

Characteristics of periods of bone destruction during active periodontal disease	• Clinical: subgingival ulceration and rapid loss of alveolar bone • Histopathology: coincides with the conversion of a predominantly T lymphocyte lesion to one with a predominantly B lymphocyte–plasma cell infiltrate • Microbiology: associated with an increase in the loose, unattached, motile, gram-negative, anaerobic pocket flora
Bone destruction caused by trauma from occlusion	Bone destruction caused by trauma from occlusion can occur in the absence or presence of inflammation. In the absence of inflammation, persistent occlusal trauma can increase osteoclast activity, resulting in bone loss, which is reversible. In the presence of inflammation, persistent occlusal trauma can exacerbate bone loss.

Core Knowledge

Introduction

Irrespective of the specific combination of etiologic and risk factors leading to the occurrence of periodontitis, certain pathogenic features are common to all forms of this disease. These common clinical presentations involve periodontal pocket formation due to attachment loss and bone loss. Different patterns of bone loss are also seen. This chapter provides a comprehensive review of commonly found types of pockets and bone loss patterns.

Periodontal Pocket

The periodontal pocket is a pathologically deepened gingival sulcus.

Pathogenesis of Pocket Formation

The transformation of a healthy gingival sulcus into a periodontal pocket involves the following steps:

- Plaque accumulation and consequent gingival inflammation destroy dentogingival connective tissue fibers just below the junctional epithelium (JE) at the base of the pocket.
- This allows viable JE cells to migrate apically along the root surface into collagen-depleted areas to maintain continuity with the tooth surface.
- This apical migration, combined with concomitant coronal separation of JE cells from the tooth surface at the sulcus base (due to increased neutrophil infiltration between JE cells and the resultant loss of tissue cohesiveness), leads to a pathologic deepening of the gingival sulcus called "pocket." The separated JE cells at the coronal end may become part of the pocket-lining epithelium at the base of the sulcus. See Fig. 15.1 for classification of pockets.

Please refer to Chapter 4, Figs. 4.1, 4.3 and Clinical correlates for more details on the pathogenesis of pocket formation and attachment loss.

CLINICAL CORRELATE

What is the rationale behind aiming for pocket reduction in periodontal therapy?

The rationale for pocket reduction is based on the need to eliminate areas of plaque accumulation. This is because the transformation of a gingival sulcus into a periodontal pocket creates a niche from which plaque removal becomes very difficult. This establishes a vicious cycle of plaque accumulation, gingival inflammation, consequent deepening of existing pockets, leading to more plaque accumulation. In order to restore health and homeostasis of both tissues and commensals, this cycle needs to be broken; hence pocket reduction is a vital goal of periodontal therapy.

Relationship of Attachment Loss and Bone Loss to Pocket Depth

- **Attachment loss** refers to the pathologic detachment of collagen fibers of the gingiva and periodontal ligament from the cemental surface, with the concomitant apical migration of the junctional or pocket epithelium on the root surface. In periodontitis, it usually *precedes* alveolar bone loss by a few (6–8) months.
- The severity of attachment loss and bone loss is generally but not always correlated with pocket depth (Fig. 15.2).

Clinical Features Associated With Periodontal Pocket Formation

The only reliable method of locating periodontal pockets and determining their extent is careful probing of the gingival margin along each tooth surface. A pocket may be characterized by (Fig. 15.3):

- a bluish-red thickened marginal gingiva;
- a bluish-red vertical zone from the gingival margin to the alveolar mucosa;
- gingival bleeding and suppuration;
- tooth mobility;

BASIC SCIENCE CORRELATE

What are the clinically significant root surface wall changes during pocket formation?

The root surface wall of periodontal pockets often undergoes changes that are significant because they may perpetuate the periodontal infection, cause pain, and complicate periodontal treatment.

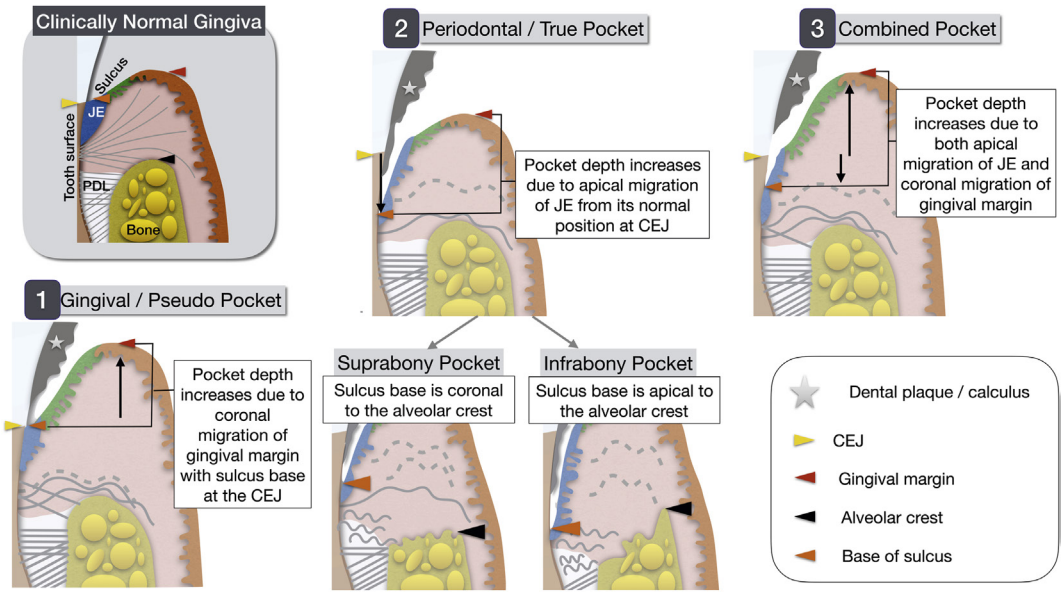
1. **Perpetuation of infection by providing a nutritionally favorable environment for pathogens on periodontally diseased, noncarious cementum surfaces:** As the pocket deepens, collagen fibers embedded in the cementum are destroyed, and cementum becomes exposed to the oral environment. Collagenous remnants of Sharpey fibers in the cementum undergo degeneration, creating an environment favorable to the penetration of bacteria.
2. **Perpetuation of pain:** Degeneration of collagen fibers manifests clinically as a softening of the cementum surface; this is usually asymptomatic, but it can be painful when a probe or explorer penetrates the area.
3. **Perpetuation of posttherapy complications:** Exposure of root cementum (a common consequence of periodontal therapy) to oral fluid and bacterial plaque results in proteolysis of the embedded remnants of Sharpey fibers; the softened cementum may undergo fragmentation and cavitation, leading to root caries and dentin hypersensitivity; it may constitute a possible reservoir for reinfection of the area after treatment.[2]

- diastema formation (due to pathological migration and/or secondary trauma from occlusion);
- symptoms such as localized pain or pain "deep in the bone."

Bone Loss and Bone Loss Patterns

Destruction of the supporting alveolar bone leads to tooth loss. The height and density of the alveolar bone are normally maintained by an equilibrium between bone formation and bone resorption, regulated by local and systemic influences. When resorption exceeds formation, both bone height and bone density may be reduced.

- The most common cause of bone destruction in periodontal disease is the extension of inflammation from the marginal gingiva into the supporting periodontal tissues.
- Resistance of the host to this inflammation extension includes:
  - host immune response;
  - width of the attached gingiva;
  - reactive fibrogenesis and osteogenesis that occur *peripheral* to the inflammatory lesion.



• **Fig. 15.1** Classification of Pockets. The left upper box in the figure shows clinically normal gingiva with a healthy gingival sulcus. Here the sulcus base (represented by the coronal end of JE) is usually at or near the cementoenamel junction (CEJ). The marginal gingiva and tooth surface form the walls of the sulcus. The sulcus may deepen due to:

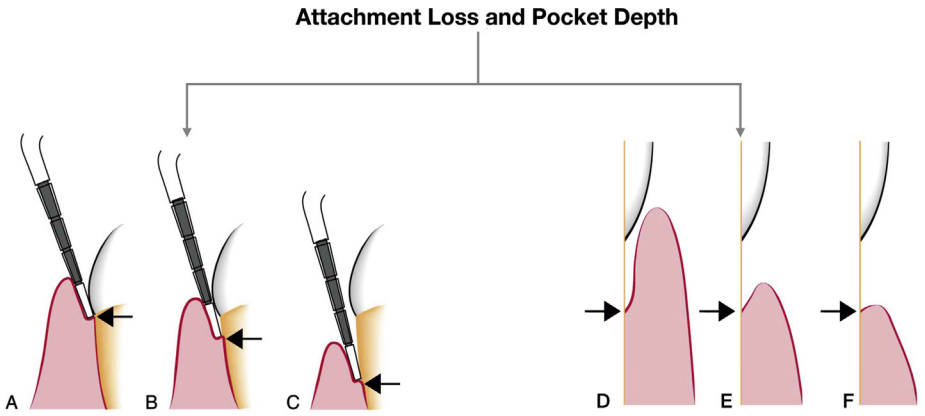
1. Coronal migration of the gingival margin to form **gingival/pseudo/false pocket**;
2. Apical migration of the JE to form **periodontal/true pocket** (may further be classified as suprabony or infrabony/intrabony pockets);
3. A combination of both the aforementioned situations to form a **combined pocket**

Another way of classifying periodontal pockets (not illustrated) is based on the number of tooth surfaces involved and the complexity of pocket configuration:

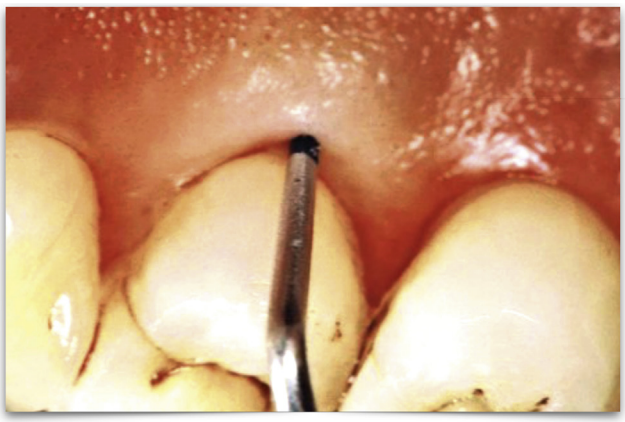
Simple pocket—involves one tooth surface;

Compound pocket—involves two or more surfaces;

Complex pocket—for example, a spiral pocket (more common in furcation areas) that originates with the pocket base on one tooth surface; the pocket then winds around to involve additional surfaces with the sulcus opening into the oral cavity on another tooth surface. *CEJ*, cementoenamel junction; *JE*, junctional epithelium; *PDL*, periodontal ligament.



• **Fig. 15.2** Relationship of Attachment Loss, Bone Loss, and Pocket Depth. Attachment loss is a phenomenon that occurs in the connective tissues of mainly the periodontal ligament and gingiva, and is different from bone loss. The degree of attachment loss depends on the location of the base of the pocket on the root surface (black arrows), whereas pocket depth is the distance between the base of the pocket and the crest of the gingival margin. The severity of attachment loss (and bone loss) is generally, but not always, correlated with pocket depth. For example, extensive attachment and bone loss may be associated with shallow pockets (Figs. C, E, and F) if the attachment loss is accompanied by recession of the gingival margin. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)



The entire length of the periodontal probe has been inserted to the base of the pocket in the palatal surface of the first premolar.

• **Fig. 15.3** Clinical Appearance of a Periodontal Pocket. Correlations exist between some clinical and histopathologic features of the periodontal pocket. These include:

- Bluish-red discoloration—caused by circulatory stagnation.
- Gingival flaccidity—caused by destruction of gingival fibers and surrounding tissues.
- Smooth, shiny surface—caused by atrophy of the epithelium and edema.
- Pitting on pressure—caused by edema and degeneration of connective tissue fibers.
- Pink and firm gingival walls—caused by fibrotic changes that predominate over exudation and degeneration, particularly in relation to the outer surface of the pocket wall. However, despite the external appearance of health, the inner wall of the pocket invariably presents some degeneration and is often ulcerated.
- Bleeding on probing—caused by increased vascularity, the thinning and degeneration of the epithelium, and the proximity of engorged vessels to the inner surface.
- Pain on probing—caused by the ulceration of the inner aspect of the pocket wall (probing healthy gingival sulcus elicits minimal to no discomfort).
- Pus discharge on digital (finger) pressure—caused by suppurative inflammation of the inner wall. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

CLINICAL CORRELATE

What is the reason for the lack of correlation between the level of alveolar bone and pocket wall changes?

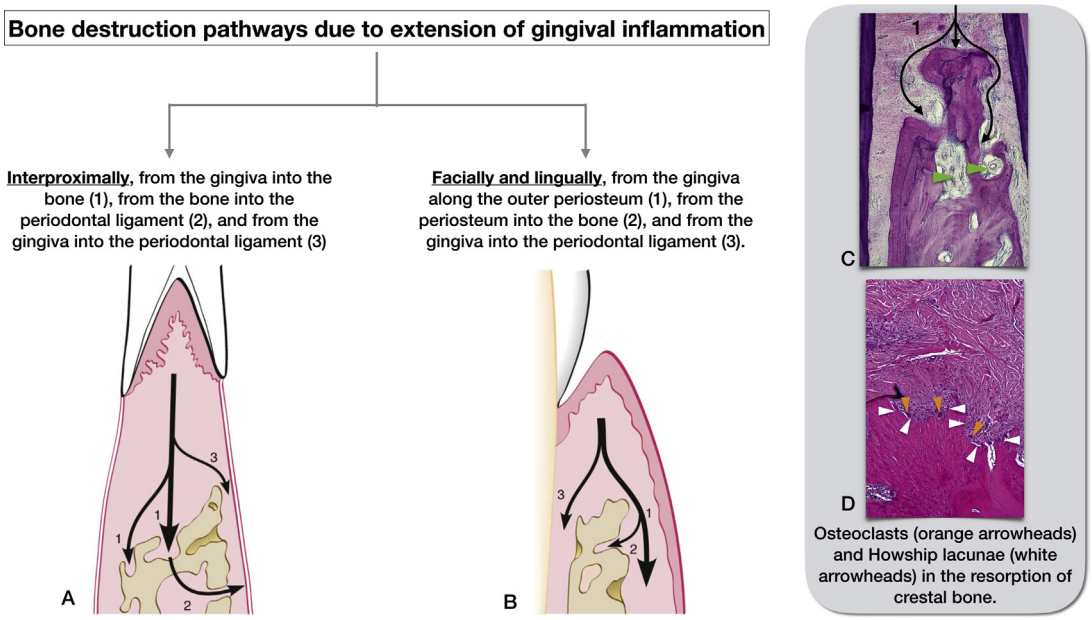
The level of bone is the consequence of *past* pathologic experiences, whereas changes in the soft tissue of the pocket wall reflect the *present* inflammatory condition. Therefore the degree of bone loss is not necessarily correlated with:

- the depth of periodontal pockets;
- the severity of ulceration of the pocket wall;
- the presence or absence of pus.

Pathways of Inflammation in Bone Destruction

Studying the pathways of spread of inflammation (Fig. 15.4) is important, because the way in which it spreads affects the pattern of bone destruction. The characteristic patterns of spread are:

- Gingival inflammation extends along the collagen fiber bundles and follows the course of the blood vessels into the alveolar bone.
- The inflammatory infiltrate often reaches the bone and elicits a response before clinical or radiographic evidence of crestal resorption or loss of attachment can exist.
- After the inflammation reaches the bone, it spreads into the marrow spaces and replaces the marrow with a leukocytic and fluid exudate, new blood vessels, and proliferating fibroblasts.



• **Fig. 15.4** Pathways of Inflammation From the Gingiva Into the Supporting Periodontal Tissues in Periodontitis. Inflammation may enter the bone through more than one channel. This figure shows two diagrammatic representations for interproximal (Fig. A) and facial/lingual (Fig. B) spread of inflammatory infiltrate, and two actual histologic sections (C and D). The diagrammatic representations show three possible pathways of inflammation extension leading to bone destruction (numbered 1, 2, and 3 in Figs. A and B).

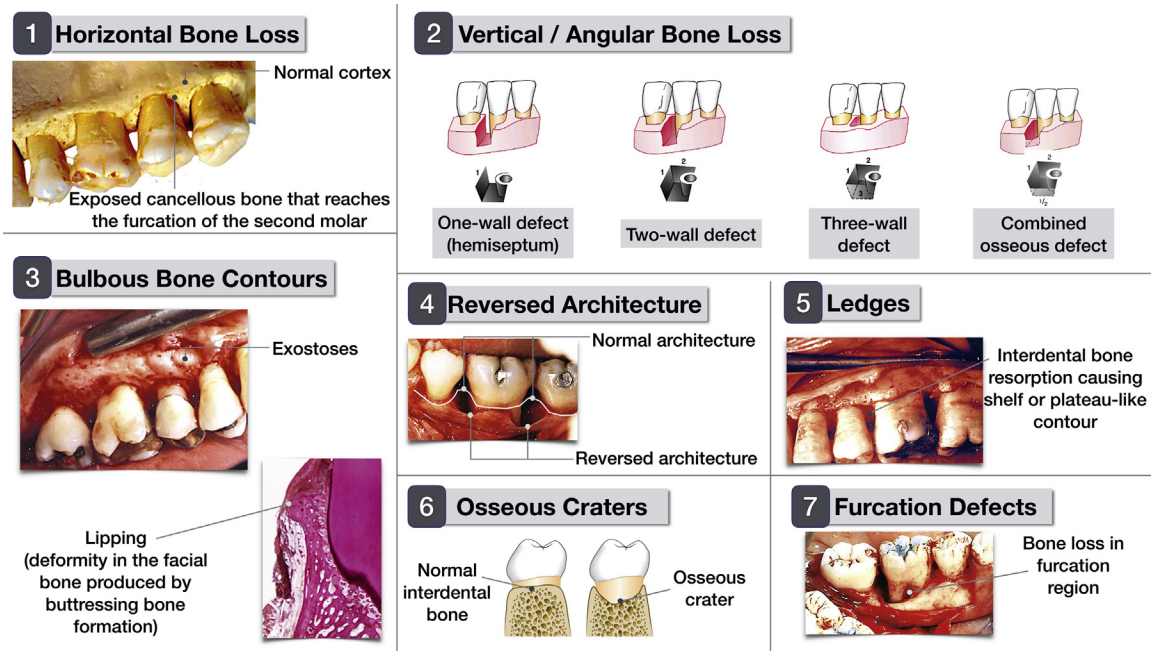
A. **Interproximal pathways:**
   1. From gingiva into bone;
   2. From bone into periodontal ligament;
   3. From gingiva directly into periodontal ligament.

B. **Facial and lingual pathways:**
   1. From gingiva along the outer periosteum;
   2. From periosteum into bone;
   3. From gingiva into the periodontal ligament.

In both A and B, direct extension of inflammation from gingiva to periodontal ligament (3) occurs less frequently than pathways 1 and 2.[3]

C. Histologic section of an interdental septum. Extensive inflammatory infiltrate has invaded the marrow spaces, entering from both the mesial and distal aspects of crestal bone. Fatty bone marrow has been replaced by inflammatory cells and fibrous marrow (green arrowheads).

D. Histologic section of crestal bone destruction with osteoclasts lining Howship lacunae. Unlike in necrotizing periodontal diseases, bone destruction in periodontal disease is not a process of bone necrosis; it involves the activity of living cells (osteoclasts) along viable bone. When tissue necrosis and pus are present in periodontal disease, they occur in the soft tissue walls of periodontal pockets rather than along the resorbing margin of the underlying bone. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)



• **Fig. 15.5** Bone Loss Patterns in Periodontal Disease. Understanding the nature of various bone loss patterns induced by periodontal disease is vital for effective diagnosis and treatment planning. Some of the common patterns of bone destruction are:

(1) **Horizontal bone loss**—the most common pattern of bone loss in periodontal disease; bone is reduced in height, but the bone margin remains approximately perpendicular to the tooth surface.

(2) **Vertical or angular bone loss**—occurs in an oblique direction to the tooth surface; the base of the defect is located apical to the surrounding bone. This type may be classified as one-wall (hemiseptum), two-wall, three-wall, and combined defects based on the remaining number of osseous walls; the remaining number of osseous walls in the apical portion of the defect is often greater than in its occlusal portion.

(3) **Bulbous bone contours**—bony enlargements caused by exostoses, adaptation to function, or buttressing bone formation (e.g., *lipping* refers to peripheral buttressing bone formation along the external surface of the facial bony plate and at the crest).

(4) **Reversed architecture**—produced by a loss of interdental bone, including the facial plates and the lingual plates, without a concomitant loss of radicular bone, thereby reversing the normal architecture (where interdental bone is always coronal to radicular bone).

(5) **Ledges**—plateau-like bone margins that are caused by the resorption of thickened bony plates.

(6) **Osseous crater**—concavities in the crest of the interdental bone confined within the facial and lingual walls; may be considered a two-wall defect.

(7) **Furcation defects**—bone loss due to the invasion of the bifurcation and trifurcation of multirooted teeth by periodontal disease. See Chapter 35 for detailed discussion. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier; and Newman M.G., Takei H.H., Klokkevold P.R., et al. (2015) *Carranza's Clinical Periodontology*, (12th ed.). Philadelphia: Elsevier.)

- In the marrow spaces, resorption proceeds from within, causing a thinning of the surrounding bone and an enlargement of the marrow spaces, followed by the destruction of the bone and a reduction in bone height.

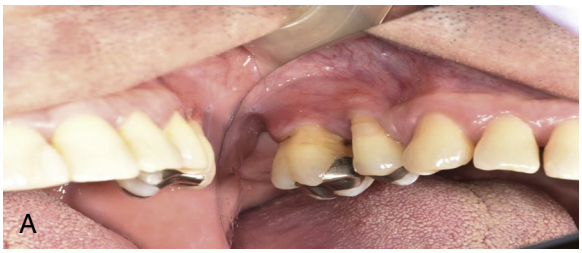
Factors Determining Morphology of Bone Defects in Periodontal Disease

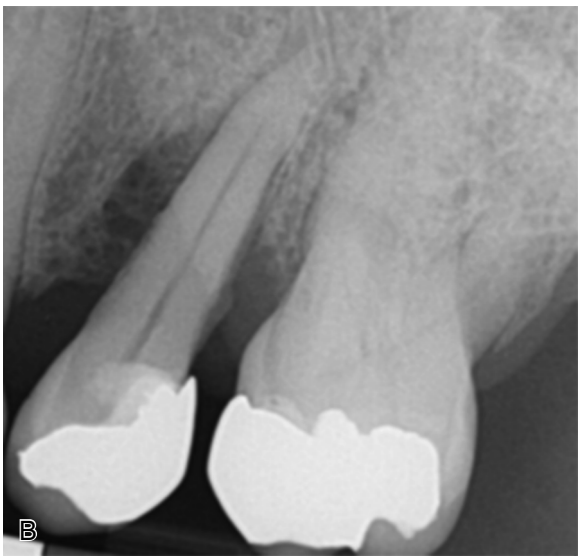
- Thickness and crestal angulation of the interdental septa;
- Thickness of the facial and lingual alveolar plates;
- Presence of fenestrations and dehiscences;
- Alignment of the teeth and root position within the alveolar process;
- Root trunk anatomy;
- Proximity with another tooth surface.

See Fig. 15.5 for a review of various patterns of bone loss commonly found as a result of periodontal disease.

CASE-BASED LEARNING EXERCISE

Scenario: A 53-year-old male patient presented with the chief complaint: "My gums constantly bleed when I brush and I want a healthy mouth." His last dental exam had been 6 months earlier. Prior to that, he had infrequent care for 5 years. Around 5 years ago, he was diagnosed with hypertension and hyperlipidemia. Oral hygiene was poor, with a plaque and bleeding index of 70%. Generalized heavy plaque with moderate to heavy supragingival and subgingival calculus was noted.





Questions

1. The bone loss pattern observed on the maxillary left bicuspid mesial surface (Fig. B) is:
a. Angular.
b. Horizontal.
c. Normal.

2. To assess furcation involvement for #14, what will be the ideal method?
a. Radiographic evaluation.
b. Clinical evaluation with a Nabers probe.
c. Surgical exposure and examination.

3. Based on the clinical and radiographic presentation (Figs. A and B), which furcation is most probably affected for tooth #14?
a. Distal.
b. Mesial.
c. Buccal.
d. Palatal.

4. The first molar and bicuspid present with premature contacts on excursive mandibular movements. If performed as monotherapy, what effect should occlusal adjustments alone have on the progression of clinical attachment and bone loss?
a. Mild.
b. Significant.

This chapter was developed from Chapters 23 and 24 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: a

Explanation: The average distance between the cementoenamel junction and bone crest is 2 mm, although substantial variation exists. The radiograph shows the distance to be more than 5 mm on the mesial surface. It is clear from the radiograph that the pattern of bone loss is angular or vertical.

2. Answer: b

Explanation: The insertion of a Nabers probe into the furcation area is the least invasive and most ideal method to diagnose a furcation involvement, which is graded by evaluating the destruction of the hard tissue and clinical attachment in the horizontal direction, using the entrance of the furcation as reference.

3. Answer: b

Explanation: Based on the radiograph, it is clear that the extent of bone loss on the mesial aspect of #14 will potentially expose the mesial furcation of the first molar.

4. Answer: a

Explanation: Mild at most—if any. The primary factor in the progression of clinical attachment and bone loss is inflammation caused by the bacterial biofilm. Therefore therapy should focus on eliminating local factors and controlling biofilm formation. Without nonsurgical periodontal therapy, occlusal treatment is likely to have little effect on disease progression.

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16

Occlusal Forces and Masticatory System Disorders That Influence the Periodontium

Relevant Terminology

Terminology /Abbreviation	Explanation
bruxism	Involuntary habit of grinding the teeth. The force generated may damage both tooth structure and periodontal tissues.
closed lock	Happens when the articular disc remains anterior to the head of the condyle during jaw movements. It is also called disc displacement, or disc displacement (dislocation) without reduction.
excessive occlusal force	Occlusal force that exceeds the adaptive or reparative capacity of the periodontal attachment apparatus, which results in occlusal trauma and/or excessive tooth wear.[1]
masticatory system	Consists of the temporomandibular joints, the masticatory muscles, the occluding teeth, and the neurovascular supplies for all those structures.
orthopedic instability of temporomandibular joints	Occurs when one or both condyles are not seated into the fossa of the temporal bone while elevator muscles are contracted to achieve stable occlusion in the maximal intercuspal position.
pathologic migration	Displacement of the tooth position caused by periodontal inflammation and altered by (not necessarily excessive) forces. Reduced periodontal support increases the risk of pathologic migration.
primary trauma from occlusion	Occurs when a tooth with normal periodontal support displays adverse changes within periodontal tissues due to excessive occlusal forces.
reciprocal click	Clicking sound in TMJ produced in the case of anterior disc displacement (the articular disc is displaced from the condylar head during jaw movement) with reduction (disc slips back into correct position over condylar head).
secondary trauma from occlusion	Secondary trauma from occlusion occurs when a tooth with reduced periodontal support is unable to withstand even normal occlusal forces leading to pathological changes within periodontal tissues.
temporomandibular joint	Formed by the condylar head of the mandible that fits into the articular fossa of the temporal bone. There is an articular disc consisting of dense connective tissues between the condyle and the temporal bone. This joint can provide both hinging (rotation) and gliding (translation) movements.
trauma from occlusion	Alterations (injuries) of periodontal tissues—including periodontal ligament, alveolar bone, and cementum—caused by occlusal forces that are beyond the adaptive capacity of the periodontium. The common clinical feature of trauma from occlusion is tooth mobility. Also called occlusal trauma.

Fast Facts

Pathogenesis of trauma from occlusion	Increase in magnitude, duration, and frequency, and altered direction of the occlusal force, may damage periodontium and result in tooth mobility.
Tissue responses to increased occlusal forces	Tissue responses have three stages: 1. Injury (excessive occlusal forces can cause periodontal tissue damage); 2. Repair (establishment of new connective tissue attachment); 3. Adaptive remodeling of the periodontium (periodontal tissues are remodeled to adapt to the forces, leading to widening of the periodontal ligament; may lead to development of intrabony defect).
Reversibility of traumatic lesions	Trauma from occlusion is reversible if the excessive occlusal force can be controlled. However, the damaged periodontal tissues may not fully recover, especially when inflammation cannot be controlled.
Relationship between plaque-induced periodontal diseases and trauma from occlusion	In general, it is believed that periodontal tissue damage caused by excessive occlusal forces is reversible if periodontal inflammation is absent; however, when both excessive occlusal forces and periodontal inflammation are present, faster progression of periodontal tissue destruction occurs. Trauma from occlusion does not initiate periodontal disease, but it is a risk factor for progression.
Clinical and radiographic signs of trauma from occlusion	Increased tooth mobility is the most common clinical sign of trauma from occlusion. Widening of the periodontal ligament space, thickening of lamina dura, and root resorption are common radiographic signs.
Muscles and nerves of the masticatory system	The muscles can be categorized into elevator muscles (e.g., masseter, medial pterygoid, temporal is muscles) and depressor muscles (e.g., geniohyoid, mylohyoid, inferior lateral pterygoid muscles). The motor and sensory innervation of the temporomandibular joints are provided by the trigeminal nerve.
Biomechanics of the masticatory system	Movement of the condyle can occur in the presence or absence of any tooth-to-tooth contact. The intensity of muscle activity and the steepness of teeth inclines can influence the position and the movement of the condyle-disc assemblies within the temporomandibular joint.
Masticatory system dysfunctions	Dysfunctions can be caused by acute (e.g., accident to the face) or chronic trauma (e.g., parafunctional occlusal habits).
Differential diagnosis of orofacial pain	Intracranial pain disorders (e.g., aneurysm, hematoma), primary headache disorders (e.g., migraine, cluster headache), neurogenic pain disorders (e.g., paroxysmal neuralgias), intraoral pain disorders (e.g., pulpitis), temporomandibular disorders, disorders of associated structures (e.g., lymph nodes), and mental disorders.
Comprehensive evaluation of masticatory system disorders	Comprehensive patient history, interview, and clinical examinations are essential to diagnosing masticatory system disorders. For example, a range-of-motion analysis can be performed to evaluate the health and function of the temporomandibular joint; in general, less than 40 mm of opening suggests limited opening.

Core Knowledge

Introduction

Excessive occlusal forces may:

- Injure the periodontium (causing changes in the periodontal ligament and alveolar bone);
- Disrupt the function of the masticatory musculature and cause painful spasms;
- Injure the temporomandibular joints (TMJs);
- Produce excessive tooth wear.

In this chapter, the effects of excessive forces on the periodontium, TMJs, and masticatory muscles will be reviewed.

Trauma From Occlusion

As the role of microbes and the host response in the development of periodontitis became clearer, the role of traumatic occlusal forces—once thought to be "causative" in periodontal destruction—was slowly relegated to that of a 'plausible' *cofactor* (a factor that can potentially modify the course/expression of a disease process but by itself cannot cause the disease) in the progression of periodontitis.[2] Based on evidence accumulated since then, occlusal trauma has been called a *risk factor* in the progression of periodontitis.[3]

Note: *Occlusal trauma* or *trauma from occlusion* are terms that refer to the tissue injury (effect) seen in the periodontium as a result of a traumatic occlusion (cause) that may cause tooth mobility (symptom) (Fig. 16.1).

Adaptive Capacity of the Periodontium

The effect of occlusal forces on the periodontium is influenced by their magnitude, direction, duration, and frequency:

- Magnitude of occlusal force—when increased, the periodontium responds with:
  - a widening of the periodontal ligament space;

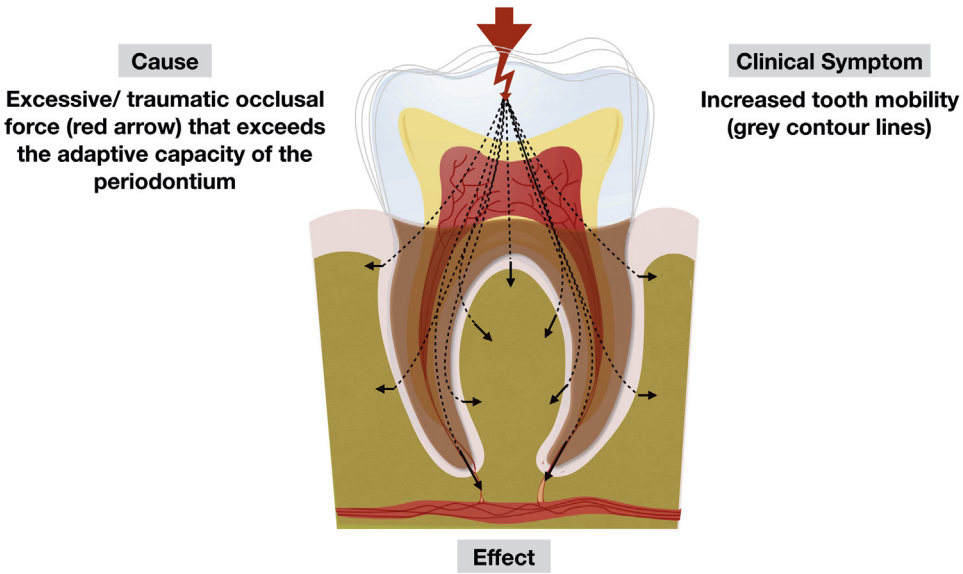
- an increase in the number and width of periodontal ligament fibers;
- an increase in the density of alveolar bone.

- Direction of occlusal force—a change in the direction of forces causes a reorientation of the stresses and strains within the periodontium.
- Duration of occlusal force—constant pressure on the bone is more injurious than intermittent forces.
- Frequency of occlusal force—the more frequent the application of an intermittent force, the greater the injury to the periodontium.

CLINICAL CORRELATE

What is the current state of evidence regarding the role of occlusal trauma and tooth mobility in periodontitis progression?

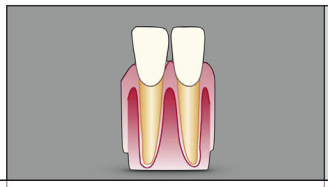
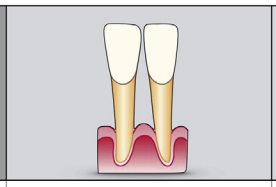
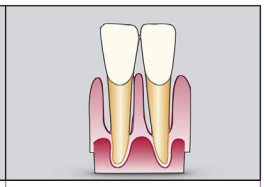
- Occlusal trauma (a lesion in the periodontium) may result from a traumatic occlusion; it is considered a *risk factor* in the progression of periodontal disease. For example, occlusal discrepancies like balancing interferences are associated with accelerated periodontal breakdown during periodontal maintenance.
- Although tooth mobility and progressing periodontitis show some relationship, (for example, mobility by itself can affect prognosis, because grade 2 tooth mobility is usually assigned a "questionable prognosis," which affects subsequent treatment options) such a relationship neither implicates nor defends occlusion as a co-factor in causing progressive periodontitis.
- Both occlusal trauma and tooth mobility may threaten periodontal longevity and impede successful therapy. Hence there is sufficient evidence to warrant consideration of *appropriate* occlusal therapy (along with inflammation control) in the management of periodontal disease. The clinician's decision whether or not to use occlusal therapy should be based on an evaluation of the patient's comfort during function, not on an assumption that occlusal adjustment is necessary to stop the progression of periodontitis.
- The impact of occlusal trauma on periodontal bone loss seems to be affected by the presence of certain systemic co-morbidities (eg.smoking, diabetes, estrogen deficiency).[2,3]



• **Fig. 16.1** Trauma From Occlusion, Traumatic Occlusion, and Tooth Mobility. The three interrelated phenomena of trauma from occlusion, traumatic occlusion, and tooth mobility may be thought of in these terms:[3]

- Occlusal discrepancies/traumatic occlusion—not the pathology, but the cause for pathology. Traumatic occlusal relationships are also referred to by such terms as *occlusal disharmony*, *functional imbalance*, and *occlusal dystrophy*. An increased occlusal force is not traumatic if the periodontium can accommodate it.
- Trauma from occlusion/occlusal trauma/occlusal traumatism—the (potential) resultant lesion of tissue injury when occlusal forces exceed the adaptive capacity of the periodontium; considered as the actual pathology (effect).
- Tooth mobility—considered as the symptom of the pathology. Note that not all occlusal discrepancies cause tooth mobility.

TFO, trauma from occlusion (or occlusal trauma). The stress lines in the figure are meant only to convey the concept.

			
Clinical Presentation	Traumatic forces on normal periodontium with normal height of bone.	Normal forces on normal periodontium with reduced height of bone.	Normal forces on teeth with periodontitis with reduced height of bone.
Type of TFO	Primary	Secondary	
Etiology	• High filling • Drifting or extrusion of teeth into edentulous spaces	Reduced ability of the tissues to resist occlusal forces	
Changes in supracrestal fibers / JE and clinical implications	Nil. Hence no loss of connective tissue attachment or pocket formation.	Marginal inflammation reduces the periodontal attachment area and alters the leverage on the remaining tissues. The periodontium becomes more vulnerable to injury, and previously well-tolerated occlusal forces become traumatic.	

• **Fig. 16.2** Primary Versus Secondary Trauma From Occlusion. When trauma from occlusion is the result of alterations in occlusal forces, it is called primary trauma from occlusion. When it results from the reduced ability of the tissues to resist the occlusal forces, it is known as secondary trauma from occlusion. *TFO*, trauma from occlusion (or occlusal trauma); *JE*, junctional epithelium. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

Classification

The criterion that determines a traumatic occlusion is whether it produces periodontal injury, irrespective of how the teeth occlude. Hence, *any* occlusion (even one that looks normal) that produces periodontal injury is traumatic; similarly, malocclusion does not necessarily always cause tissue injury. TFO can be:

- **Acute**—resulting from an ***abrupt occlusal impact*** such as that produced by biting on a hard object (e.g., small stone in soft food, restorations or prosthetic appliances that interfere with or alter the direction of occlusal forces); it results in tooth pain, sensitivity to percussion, and increased tooth mobility, and may also produce cementum tears.
- **Chronic**—most often developing from ***gradual changes in occlusion*** produced by tooth wear, drifting movement, and extrusion of the teeth in combination with parafunctional habits (e.g., bruxism, clenching) rather than as a sequela of acute periodontal trauma; it is more common than the acute form and is of greater clinical significance. It may be further classified as (Fig. 16.2):
  - Primary TFO—caused by altered occlusal forces on normal periodontium;
  - Secondary TFO—caused by normal occlusal forces on reduced periodontium.

Clinical and Radiographic Features in TFO

TFO lesions within the periodontium, with or without concurrent inflammatory periodontal disease, display certain characteristic clinical and radiographic features (though such features are not considered pathognomonic of or exclusive to this condition) (Fig. 16.3). It is necessary to use supplementary diagnostic procedures, such as pulp vitality tests, evaluation of parafunctional habits, and so on, to establish a proper differential diagnosis.

CLINICAL CORRELATE

What is the correct timing of definitive occlusal adjustments during periodontal therapy?

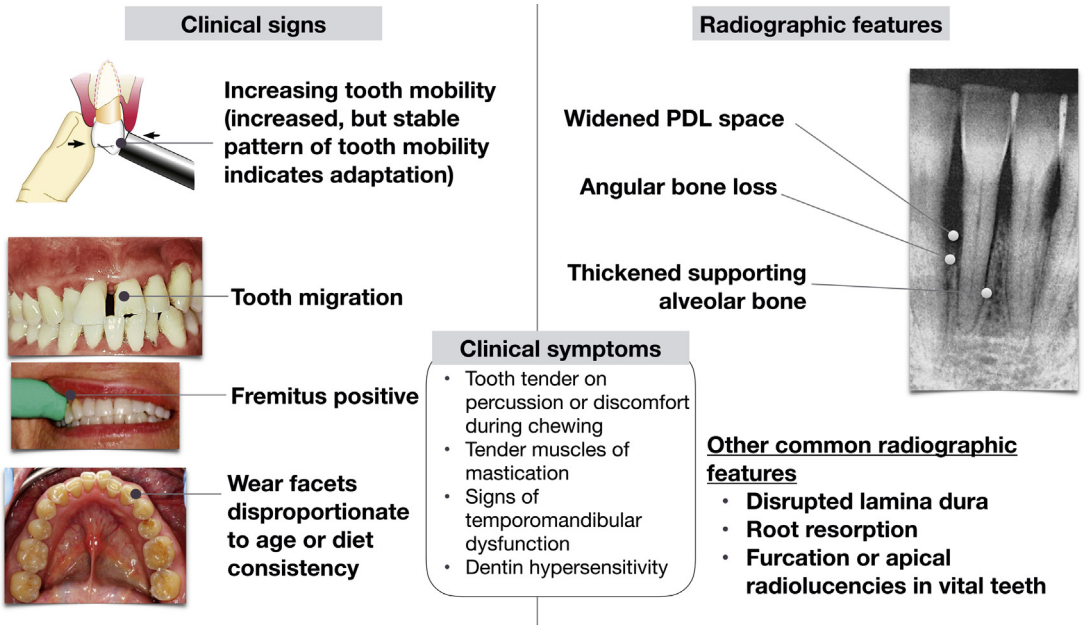
Although tooth mobility is not always due to occlusal trauma, it is one of the primary clinical signs of TFO. When significant and increasing mobility exists in teeth, the clinician should perform:[3]

- Periodontal inflammation control measures (e.g., scaling, root planing, oral hygiene reinforcement) before definitive occlusal therapy.
- Occlusal therapy before attempting any periodontal regenerative therapy.

Stages of Tissue Response in TFO

Excessive forces usually result in bone resorption in areas of pressure, and bone formation in areas of tension. The changes seen in the periodontal ligament space and bone occur in three stages: injury, repair, and adaptive remodeling of the periodontium (Table 16.1):

1. **Injury**: Injury to the periodontium produces a temporary suppression of mitotic activity, collagen formation, and bone formation. These return to normal levels after the dissipation of the forces.
2. **Repair**: The damaged tissues are removed and new connective tissue cells and fibers, bone, and cementum are formed in an attempt to restore the injured periodontium. Forces remain traumatic only as long as the damage produced exceeds the reparative capacity of the tissues.
3. **Adaptive remodeling**: If the repair process cannot keep pace with the destruction enhanced by the occlusion, the periodontium is remodeled in an effort to create a structural relationship in which the forces are no longer injurious to the tissues.



• **Fig. 16.3** Clinical and Radiographic Features of Trauma From Occlusion. The body usually attempts to repair the tissue injury caused by excessive occlusal forces. This can occur if the forces are diminished or if the tooth drifts away from them. If the offending force is chronic, however, the periodontium is remodeled to cushion its impact. The ligament is widened at the expense of the bone, which results in angular bone defects without periodontal pockets, and the tooth becomes loose.[4] *Fremitus*, a "palpable" or "visible" movement of a tooth when subjected to occlusal forces which indicates functional mobility; *PDL*, periodontal ligament. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

TABLE 16.1 Stages of Tissue Response to Increased Occlusal Forces

	Injury	Repair	Adaptive Remodeling
	• ↑ Bone resorption • ↓ Bone formation	• ↓ Bone resorption • ↑ Bone formation	Bone resorption and bone formation return to normal
Areas of slightly excessive tension: • Elongation of the PDL fibers and the apposition of alveolar bone • Blood vessels enlarged	**Areas of slightly excessive pressure:** • Resorption of the alveolar bone, with a resultant widening of the periodontal ligament space • Blood vessels reduced in size **Areas of greater pressure:** • Compression of the PDL fibers, produces areas of hyalinization and subsequent necrosis • Increased resorption of alveolar bone and resorption of the tooth surface • Blood vessels appear to be packed with erythrocytes, which start to fragment; disintegration of the blood vessel walls and release of the contents into the surrounding tissue occurs	**Changes in alveolar bone:** *Buttressing bone formation* occurs to reinforce the thinned bony trabeculae with new bone and to compensate for lost bone. This can be: • *Central buttressing* that occurs within the jaw • *Peripheral buttressing* (e.g., lipping) that occurs on the bone surface	**Changes in alveolar bone:** • Angular bone defects with no pocket formation
Areas of severe tension: • Widening of the PDL space • Thrombosis and hemorrhage within PDL • Tearing of the PDL fibers	**Areas of severe pressure:** • Root is forced against bone causing necrosis of the periodontal ligament and bone • *Undermining bone resorption* occurs when bone is resorbed from the side of marrow spaces rather than from the side of the PDL. This happens mostly due to the presence of viable cells on the bone marrow side (versus the necrosis present on the PDL side) that would be able to take part in osseous remodeling	**Changes in PDL:** • Cartilage-like material may be formed in the aftermath of trauma	**Changes in PDL:** • Funnel-shaped widening of the PDL at the alveolar crest with resultant increase in tooth mobility

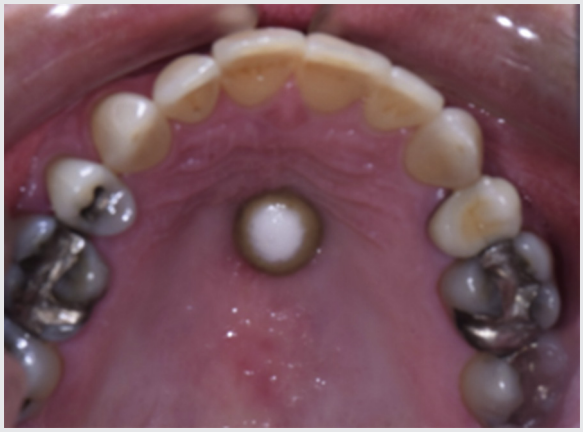
PDL, periodontal ligament. ↑, Increase in; ↓, Decrease in

Masticatory System Disorders

The masticatory system consists of the TMJs, the masticatory muscles, the teeth in occlusion, and the blood and nerve supply to all of these structures. Discomfort associated with masticatory system disorders is categorized as *orofacial pain*. Pain associated with TMJ dysfunction is most frequently muscular in origin, with possible amplification by both occlusal parafunction (e.g., bruxism) and stress. The reader is referred to Chapter 26 of *Newman and Carranza's Clinical Periodontology* (13th ed.) for a detailed discussion of masticatory system disorders.

CASE-BASED LEARNING EXERCISE

Scenario: The patient, a dental hygienist, presented to the dental office with the chief complaint: "I have burning pain around my lips and circling the mouth, as well as discomfort in the upper and lower anterior teeth. I also have facial muscle pain from the cheekbone down on both sides and I am aware that I have malocclusion." Medical history: fibromyalgia and depression. Dental history: she had previously seen multiple health care providers (general dentists, psychologist, and specialists) but had not found an effective solution.



Questions

1. In order to diagnose pain in the face and head, a _____ ganglion block is usually performed.
- **a.** Otic.
- **b.** Ciliary.
- **c.** Sphenopalatine.
- **d.** Submandibular.

2. During the initial consultation, the incidental finding on the hard palate could be a(n):
- **a.** Oral tablet.
- **b.** Pathology lesion.
- **c.** Orthodontic device.

3. The motor innervation of the TMJs is provided by branches of the _____ nerve.
- **a.** Facial.
- **b.** Vagus.
- **c.** Trochlear.
- **d.** Trigeminal.

4. If the clinician suspects a perforation of the articular disc, the recommended TMJ imaging would be:
- **a.** Cone beam computed tomography (CBCT).
- **b.** Panoramic radiograph.
- **c.** Arthrography.
- **d.** Conventional computed tomography (CT).

This chapter was developed from Chapters 25 and 26 in Newman and Carranza's Clinical Periodontology (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: c

Explanation: The sphenopalatine ganglion is a collection of parasympathetic nerves. A sphenopalatine ganglion block is done to diagnose the root cause of pain in the face and head in order to manage facial pain and headaches effectively.

2. Answer: a

Explanation: Patient uses these oral tablets to stimulate saliva secretion. It is an over-the-counter medication, and her increased health literacy led her to decide to start using it.

3. Answer: d

Explanation: The motor and sensory innervation of the TMJs and the rest of the masticatory system are provided by branches of the trigeminal nerve.

4. Answer: c

Explanation: Arthrography is used for certain diagnostic situations, such as suspected perforation of the articular disc, and nuclear medicine has developed protocols to image the TMJ to determine if active deterioration is occurring.

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17 Periodontitis

Relevant Terminology

Terminology / Abbreviation	Explanation
adjunctive systemic antibiotic therapy	The use of a metronidazole-amoxicillin combination improves clinical outcomes of nonsurgical periodontal therapy in patients with aggressive forms of periodontitis. These antibiotics have an additive effect against *Aggregatibacter actinomycetemcomitans*, often implicated in what was previously considered as aggressive periodontitis.
asynchronous, multiple-burst model	Several models have been proposed to explain the disease progression rate of periodontitis. This model is characterized by bursts of periodontal destruction during defined periods, which are asynchronously interrupted by periods of disease stagnation or remission in sites and teeth.
chronic periodontitis	Defined as a chronic and infectious disease resulting in inflammation within the periodontal tissues, progressive bone loss, and attachment loss. Since the 2017 World Workshop, periodontitis is no longer subcategorized into chronic periodontitis and aggressive periodontitis.[1]
continuous model of periodontitis progression	Several models have been proposed to explain the disease progression rate of periodontitis. This model is characterized by slow, continuous, and consistent disease progression throughout the duration of the disease.
familial aggregation	Familial aggregation is the clustering of certain traits or disorders within a family. A familial aggregation of aggressive periodontitis cases is a secondary feature of this form of periodontal disease.
generalized aggressive periodontitis	In addition to the common features of aggressive periodontitis, generalized aggressive periodontitis is characterized by relatively early age of onset (< 30 years), generalized periodontal destruction in dentition, and poor serum antibody response against pathogens. In the 2017 classification, there is no separate category for this older disease terminology.
localized aggressive periodontitis	In addition to the common features of aggressive periodontitis, localized aggressive periodontitis is characterized by early age of onset (circumpubertal), localized periodontal destruction on first molars and incisors, and robust serum antibody response against pathogens. In the 2017 disease classification, localized aggressive periodontitis is now termed as periodontitis with molar/incisor pattern (followed by the stage and grade).
random or episodic burst model	Several models have been proposed to explain the disease progression rate of periodontitis. This model is characterized by short bursts of periodontal destruction followed by periods of stagnation; these episodes may happen randomly throughout the duration of the disease.

Fast Facts

Clinical signs of periodontitis	It is common to observe one or many of the following characteristics in patients with untreated periodontitis: supragingival and subgingival calculus, gingival swelling, bleeding on probing, gingival recession, pocket formation, attachment loss, alveolar bone loss, furcation involvement, tooth mobility, and tooth loss.
Symptoms	Bleeding or suppuration upon brushing, gingival recession and associated sensitivity, tooth mobility, and/or localized dull pain.
Disease distribution / extent	Periodontitis is site-specific, and alveolar bone loss and attachment loss are not equally distributed around the teeth and dentition. If fewer than 30% of teeth are involved, it is called localized periodontitis. If 30% or more of teeth are involved, it is called generalized periodontitis. In the new disease classification, what was previously termed localized aggressive periodontitis is now included as molar/incisor pattern under disease extent.

Fast Facts—cont'd

Disease severity	According to the 2017 Workshop, the severity of periodontitis can be categorized based on bone loss (BL), attachment loss (CAL) and pocket depth (PD):[1] • Mild (Stage I): BL < 15%, CAL 1–2mm, PD 4 mm • Moderate (Stage II): BL 15%–33%, CAL 3–4 mm, PD 5 mm • Severe (Stage III/IV): BL > 33%, CAL ≥ 5 mm, PD ≥ 6 mm
Progression	Periodontitis progression is generally slow but can be increased by local (e.g., restoration overhang), systemic (e.g., uncontrolled diabetes) and environmental factors (e.g., smoking). If a tooth has ≥ 2 mm attachment loss over 5 years, the progression rate can be considered rapid.[1]
Microbiologic aspects	Increased prevalence of some bacterial species is associated with the severity of periodontitis. These periodontal pathogens (e.g., *Porphyromonas gingivalis*) interacting with host responses may result in a dysbiotic microbial environment.
Local factors	Factors like calculus, furcations, deep probing depth, and poor crown margins may facilitate plaque accumulation or prevent plaque removal, resulting in periodontal inflammation.
Systemic factors	These factors include diseases that affect the host immune response. For example, uncontrolled diabetes negatively affects the activities of immune cells and bone cells in a way that may contribute to periodontal destruction.
Environmental and behavioral factors	Smoking and psychological stress are two major contributors to periodontitis. Smoking, apart from causing vasoconstriction, stimulates the release of reactive oxygen species and hinders the immune response, leading to periodontal destruction. Patients suffering from stress may have poor oral hygiene that may cause periodontal inflammation.
Features of aggressive periodontitis	According to the 2017 World Workshop, current evidence does not support a distinction between chronic and aggressive periodontitis.[1] However, periodontitis still has distinct clinical patterns in different patients. The features of aggressive periodontitis include rapid periodontal destruction, early onset, and disproportionate destruction of the periodontal attachment for the given biofilm deposits.
Pathobiology of aggressive form of periodontitis	Longitudinal studies showed the presence of *A. actinomycetemcomitans* to be associated with localized aggressive periodontitis, and impairment in neutrophil-based defense functions was found in patients with localized aggressive periodontitis.
Therapeutic considerations in patients with aggressive form of periodontitis	Patients with aggressive form of periodontitis usually have severe bone loss, deep probing depth, and vertical bony defects, so they may receive surgical treatments more often than patients with chronic periodontitis. Adjunctive use of systemic antibiotics during scaling and root planning, and a stringent maintenance program, can usually also benefit these patients.

Core Knowledge

Introduction

Periodontitis is a microbe-associated, host-mediated inflammatory disease that results in destruction of periodontal attachment. Its pathophysiology is characterized by the microbe-associated activation of host-derived proteinases that enable:

- Loss of marginal periodontal ligament fibers and gingival connective tissue attachment
- Apical migration of the junctional epithelium on the root surface
- Consequent apical spread of the bacterial biofilm along the root surface
- Loss of the supporting alveolar bone

The 1999 Classification of Periodontitis: Rationale for Revision

The accumulated scientific evidence on periodontal disease pathogenesis and clinical presentations impacted the 1999 classification system, which emphasized different periodontitis phenotypes, leading to the recognition of four different forms of periodontitis:

1. necrotizing periodontitis
2. chronic periodontitis
3. aggressive periodontitis
4. periodontitis as a manifestation of systemic diseases

The perception that individual features characterized the different forms of periodontitis emerged from:

- **Microbiologic evidence**—such as the identification of certain bacteria or specific bacterial complexes as probable etiologic agents of periodontitis (e.g., *A. actinomycetemcomitans* is associated with aggressive periodontitis).
- **Environmental and immunologic evidence**—such as that recognize multiple modifiable risk factors exist for periodontitis (e.g., smoking affects an individual's susceptibility to chronic periodontitis and its rate of progression; impaired resolution of inflammation leads to a destructive chronic inflammatory response within the periodontium).

- **Genetic evidence**—such as those that identify genetic susceptibility and specific polymorphisms associated with disease severity as relevant in etiology / pathogenesis (e.g., an IL-1 gene polymorphism is associated with chronic periodontitis).

The clinical perspective on periodontitis, however, was somewhat different from the research perspective. The past two decades saw clinicians, epidemiologists, researchers and educators raise concerns about the practical difficulties in effectively differentiating aggressive forms of periodontitis from chronic periodontitis due to[2]:

- The substantial overlap between the diagnostic categories encountered when trying to apply the stipulated criteria for the two diseases in daily practice
- The unconfirmed validity of many of the stipulated criteria for aggressive periodontitis so far tested in adequately designed studies

CLINICAL CORRELATE

What is the current summary and interpretation of evidence regarding the different forms of periodontitis?

- Despite substantial research on aggressive periodontitis since the 1999 workshop, there is currently insufficient evidence to consider aggressive and chronic periodontitis as two pathophysiologically distinct diseases.
- There is, however, sufficient evidence to consider necrotizing periodontitis as a separate disease entity. Evidence indicates:[2]
  - a unique pathophysiology characterized by marked bacterial invasion and epithelial ulceration;
  - rapid and full-thickness marginal soft tissue destruction, resulting in distinct soft and hard tissue defects;
  - noticeable disease-specific signs and symptoms such as pain, bleeding and papillary necrosis.
  - rapid disease resolution following specific antimicrobial treatment.
- There is sufficient evidence to point out that in cases of periodontitis influenced by systemic diseases (that negatively affects the host response), the primary diagnosis should be the systemic disease and periodontits should be considered a manifestation of that systemic disease.

The 2017 Classification of Periodontitis

Based on pathophysiology, three clearly different forms of periodontitis have now been identified:[1]

1. Necrotizing periodontitis
2. Periodontitis as a direct manifestation of systemic diseases
3. Periodontitis

The reader is referred to chapters, 9, 10 and 18 for further reading on necrotizing periodontitis and systemic diseases that directly impact the periodontium.

Clinical Diagnosis of Periodontitis

Periodontitis is considered a complex inflammatory disease. The word "complex" relates to the multiple clinical symptoms and factors that lead to and influence periodontal inflammation. Clinical diagnosis of this condition involves the following considerations:

- Clinical attachment loss (CAL), a clinical parameter measured using standardized periodontal probes walked circumferentially around erupted dentition, uses the cementoenamel junction (CEJ) as a reference point to detect this disease.
- Bleeding on probing (BOP) is another important clinical parameter used to assess periodontitis treatment outcomes and disease risk following treatment. However, BOP alone (or as a secondary parameter with CAL) does not change the initial case definition determined by CAL, or the classification of periodontitis severity.

The reader is referred to Chapter 19 for a detailed review of assessing CAL, BOP, and so on during clinical diagnosis. Fig. 17.1 discusses the three components involved in clinically defining a case of periodontitis.

CLINICAL CORRELATE

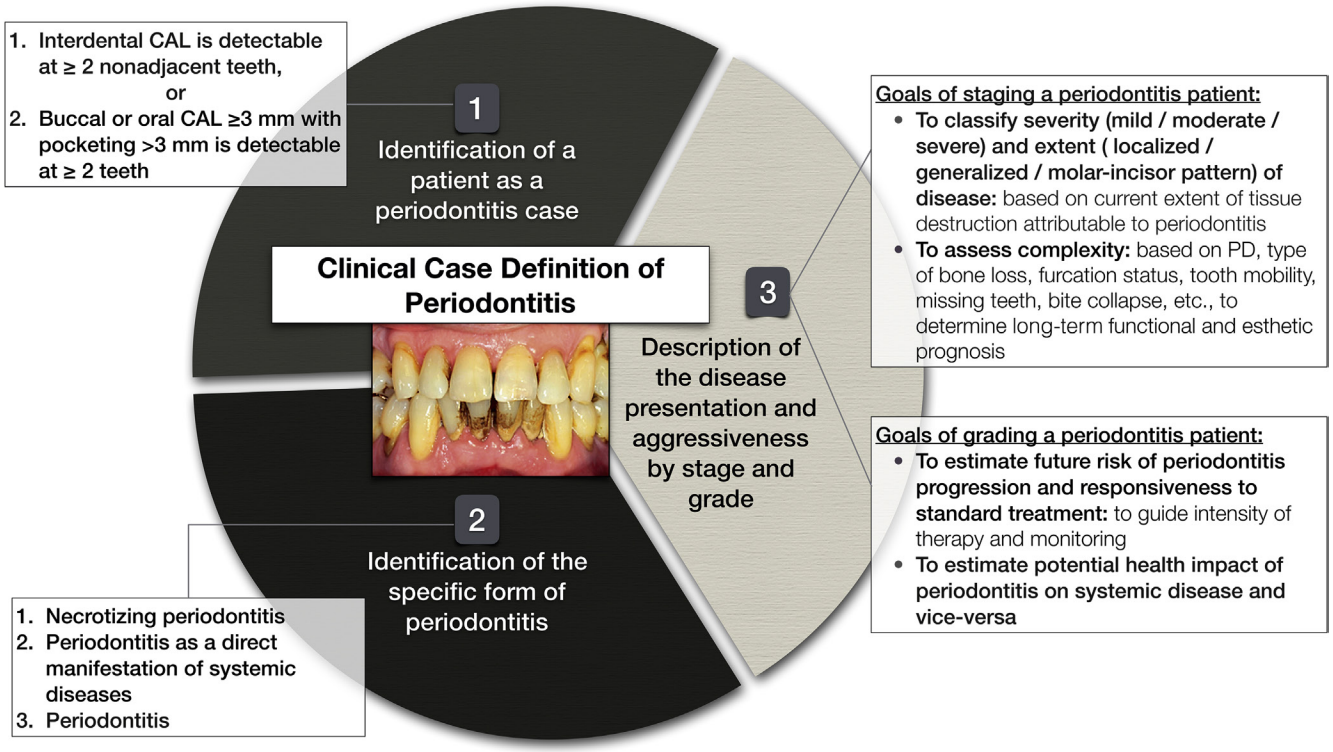
Why is it important to allow space for future integration of biomarkers in clinically defining and diagnosing a case of periodontitis?

Biomarkers may contribute to improved diagnostic accuracy in the detection of periodontitis because of:[2]

- Varying disease susceptibilities—current evidence suggests that some individuals are more susceptible to developing severe periodontitis and less responsive to standard prevention and treatment modalities. Currently employed clinical parameters are not proven to be *sufficient* in monitoring disease progression and treatment outcomes. In this context, it is believed that biomarkers (some of which are already available) may be valuable additions to the information provided by standard clinical parameters.
- Challenges in early diagnosis—because periodontal probing (the current gold standard for defining periodontitis) may be inaccurate when trying to estimate early clinical attachment loss, assessment of biomarkers may increase early detection of stage I periodontitis in a variety of settings. Biomarkers may also assist in both staging and grading of periodontitis.

Clinical and Radiographic Features of Periodontitis

Periodontitis, a multifactorial immuno-inflammatory chronic condition, is associated with dysbiotic plaque biofilms and characterized by progressive destruction of tooth-supporting structures. Its primary features include:



• **Fig. 17.1** Clinical Definition of Periodontitis. A periodontitis diagnosis for an individual patient should encompass three dimensions:[2]

1. **Identification of periodontitis**—the clinical definition for a case of periodontitis is given in the upper left box. The observed CAL here must *not* be due to nonperiodontal causes such as: 1) trauma induced gingival recession; 2) dental caries extending in the cervical area of the tooth; 3) CAL on the distal aspect of a second molar and associated extraction of a third molar or tooth malposition; 4) an endodontic lesion draining through marginal periodontium; or 5) vertical root fracture.
2. **Identification of the specific form of periodontitis**—the currently accepted forms of periodontitis are described in the lower left box. Differential diagnosis is based on history, the specific signs and symptoms of necrotizing periodontitis, and the presence or absence of systemic diseases that definitively alter the host immune response. The remaining clinical cases of periodontitis which do not fit into necrotizing periodontitis or the systemic characteristics of a rare immune disorder with periodontitis as manifestation should be diagnosed as "periodontitis" and further characterized using a staging and grading system.
3. **Description of the clinical presentation and other elements that affect clinical management and prognosis—**This includes assessing factors like disease extent, severity, complexity, risk factors, progression and presence of systemic diseases.

Staging (indicates current disease status)—this is determined using CAL; if CAL measurements are not available, radiographic bone loss (RBL) should be used.

Grading (indicates future disease status)—grade should be used as an indicator of the rate of periodontitis progression. The primary criteria are either direct or indirect evidence of progression. Whenever available, longitudinal observation-based direct evidence in the form of older radiographs is used; in its absence, this is estimated indirectly by calculating bone loss (%) at the most effect tooth and dividing it by patient's age.

CAL, clinical attachment loss; *PD*, probing depth; *RBL*, radiographic bone loss. The reader is referred to Chapter 3 (Tables 3.3 and 3.4) of this book for a review of the staging and grading system used in diagnosis of periodontitis. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

- Loss of periodontal tissue support (manifested through CAL and radiographic alveolar bone loss)
- Periodontal pockets
- Gingival bleeding

Other characteristic features include:

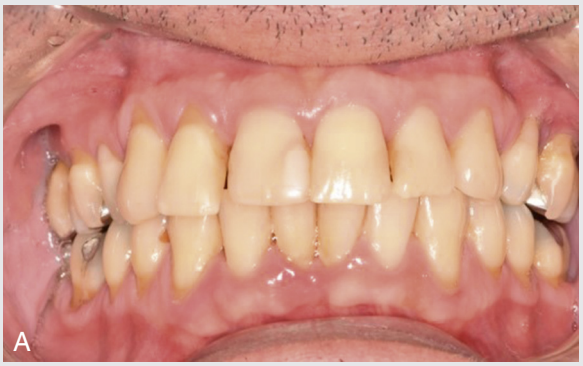
- Supragingival and subgingival plaque and calculus
- Gingival swelling, redness, and loss of gingival stippling
- Altered gingival margins (rolled, flattened, cratered papillae, recessions)
- Bone loss (angular/vertical or horizontal)
- Root furcation involvement
- Increased tooth mobility

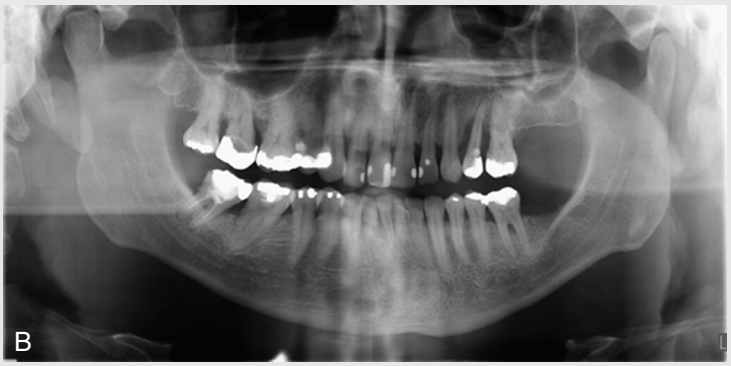
- Pathologic migration of teeth
- Tooth loss

The reader is referred to Chapter 19 for further discussion on clinical and radiographic diagnosis of periodontal disease.

CASE-BASED LEARNING EXERCISE

Scenario: A 57 year old male patient presented with the chief complaint: "My gums constantly bleed when I brush and I want a healthy mouth." His last dental exam had been 8 months ago and prior to that, he had infrequent care for 6 years. Five years previously, he had been diagnosed with hypertension and hyperlipidemia. Patient's body mass index at the time of the exam was 32. Patient's body mass index at the time of the exam was 32. His oral hygiene was poor, with a plaque index of 30% and bleeding index of 70%.





Generalized heavy plaque with moderate supragingival and subgingival calculus was noted (Fig. A). Generalized deeper probing depths in the range of 5–11 mm with generalized 1–4 mm recession, mobility, and furcation involvement were detected. Radiographic exam revealed localized areas of moderate to severe horizontal bone loss and vertical defects (Fig. B).

Questions

1. Based on the clinical presentation, what will be the recommended treatment sequence?
   a. Scaling and root planing and reevaluation
   b. Surgical therapy and re-evaluation
   c. Scaling and root planing, re-evaluation, and possible surgical therapy

2. Select the statement that is characteristic for chronic periodontitis.
   a. With optimal oral hygiene, this condition can be resolved completely (is reversible).
   b. The dental implant counterpart of periodontitis is peri-implant mucositis.
   c. The attachment loss is irreversible, even if the inflammation is controlled successfully.

3. The episodic occurrence of short progressive bursts of periodontal destruction followed by periods of stagnation is known as the _____ model.
   a. Continuous
   b. Random burst
   c. Asynchronous

4. What systemic marker is expected to have an increased value given this patient was obese?
   a. HbA1C
   b. CRP
   c. CTX

This chapter was developed from Chapters 27 and 28 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Solutions

1 Answer: c

Explanation: Scaling and root planing, followed by reevaluation and possible surgical therapy, is the recommended treatment sequence for this patient. The need for surgical therapy is dictated by several factors, which will be assessed at the time of reevaluation. If surgery is not indicated or required, the patient will usually be placed on an individualized periodontal maintenance program.

2 Answer: c

Explanation: Statement (a) is characteristic for gingivitis and statement (b) is not correct; the counterpart of periodontitis is peri-implantitis. The only statement characteristic for

chronic periodontitis is (c). Unlike gingivitis, controlling inflammation in periodontitis will not reverse the disease outcome (attachment loss).

3 Answer: b

Explanation: The random or episodic-burst model describes the episodic occurrence of short progressive bursts of periodontal destruction followed by periods of stagnation.

4 Answer: b

Explanation: It is known that systemic inflammatory markers, such as C-reactive protein (CRP), are elevated in patients who are overweight or obese, who present with low grade systemic inflammation.

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18

Necrotizing Periodontitis and Management Considerations for Patients With Human Immunodeficiency Virus

Relevant Terminology

Terminology /Abbreviation	Explanation
acquired immunodeficiency syndrome	Acquired immunodeficiency syndrome (AIDS), caused by human immunodeficiency virus (HIV) infection, is characterized by a significantly impaired immune system. The condition is potentially life-threatening but can be controlled by medications. It can be transmitted through sexual activity, body fluid contact, childbirth, or breastfeeding.
human immunodeficiency virus (HIV)	HIV is a retrovirus. Helper T lymphocytes (T4 cells) are most affected by HIV, but other immune cells can be affected too. HIV has two types (HIV-1 and HIV-2) and three subgroups (M, N, O). HIV-1 subgroup M is primarily responsible for worldwide spread.
immune reconstitution inflammatory syndrome (IRIS)	In HIV/AIDS patients receiving highly active antiretroviral therapy (HAART), complications involving an exaggerated inflammatory reaction (to existing or new antigens) sometimes occur when the immune system begins to recover. This condition is called IRIS. IRIS can be induced by antigens including infectious organisms, tumor antigens, and host autoimmune antigens.
Kaposi sarcoma	Kaposi sarcoma, caused by human herpesvirus 8 (HHV-8), is the most common oral malignancy found in patients with HIV/AIDS. It is a vascular neoplasm that affects skin or mucosa. In the oral cavity, the lesion can be seen in the palate, gingiva, and tongue.
necrotizing periodontitis	Necrotizing periodontitis (NP) is distinct from necrotizing gingivitis (NG). In addition to ulceration on the gingiva, NP is also characterized by the loss of periodontal attachment and alveolar bone.
necrotizing stomatitis	Sometimes the ulcerative lesion in NP may extend from gingiva to other soft tissue areas (e.g., palate, vestibule) with the exposure of bone and sequestration of bone fragments in patients with HIV/AIDS. This condition is called necrotizing stomatitis.
oral candidiasis	Opportunistic infection caused by the overgrowth of *Candida* species. It is the most common oral lesion in patients with HIV/AIDS and may manifest in all immunocompromised patients.
oral hairy leukoplakia	Oral hairy leukoplakia is characterized by a poorly demarcated and keratotic area (white patch) on the lateral border of the tongue. It is induced by Epstein–Barr virus, usually asymptomatic and primarily occurs in patients with HIV/AIDS.
staging of HIV/AIDS	The staging system is published by the World Health Organization. Staging is based on the clinical appearance and symptoms. The severity of symptoms increases from stage 1 to stage 4.
T4 lymphocytes (CD4+ or T helper cells)	T4 lymphocytes express CD4 receptors on their surface and become activated after binding to antigen presented by antigen-presenting cells (e.g., dendritic cells). They can assist other immune cell functions, including maturation of plasma cells and activation of cytotoxic T cells and macrophages. In the blood, the normal count is between 500–1200 cells/mm^3.

Fast Facts

Clinical features of necrotizing periodontitis	Ulceration at gingival margin and papilla, gingival inflammation and bleeding, clinical attachment loss, and bone loss. It is uncommon to find deep pockets in NP cases because the necrosis of junctional epithelium prevents pocket formation.
Histologic findings of NP	Similar to necrotizing gingivitis: biofilm layer, aggregations of polymorphonuclear leukocytes, and necrotic cells can be seen in the ulcerative lesion.
NP in patients with HIV/AIDS	It is more frequent to see NP in HIV/AIDS patients than other patients. The attachment loss and bone loss caused by NP are more severe in patients with HIV/AIDS than patients without HIV/AIDS.
Correlation between severity of NP and immunosuppression	HIV/AIDS patients with NP may have more severe immunosuppression (e.g., lower $CD4^+$ T helper cell counts) than HIV/AIDS patients without NP.
Pathogenesis of HIV/AIDS	HIV infection gradually and significantly impairs the immune system by interfering with functions of T4 lymphocytes ($CD4^+$ T helper cells) and other immune cells. Infected T4 lymphocytes can indirectly dysregulate B lymphocytes and neutrophils.
High-risk populations for HIV/AIDS	The high-risk populations for HIV/AIDS infection include homosexual and bisexual men, intravenous drug abusers, infants of HIV-infected mothers, and promiscuous heterosexuals.
Diagnosis of HIV/AIDS	According to the Centers for Disease Control and Prevention (CDC), a person with HIV infection can be diagnosed as having AIDS if they have a CD4 count less than 200 cells/mm^3 and AIDS-defining conditions (e.g., Kaposi sarcoma, candidiasis).
Combined therapeutic regimens for HIV/AIDS	The combined regimens (HAART), consisting of reverse transcriptase inhibitors, proteinase inhibitors, and fusion inhibitors, improve the health of infected patients effectively. While patients are receiving the therapy, the detectable level of virus may be low, but it is never completely eradicated.
Challenges in long-term control of HIV/AIDS	It is challenging to control HIV/AIDS in the long term because of adverse side effects of medications (e.g., gastrointestinal syndrome, bone marrow suppression) and the emergence of drug-resistant variant viral strains.
Oral and periodontal manifestations of HIV/AIDS patients	Patients with HIV/AIDS often have some oral complications, including oral candidiasis, oral hairy leukoplakia, atypical periodontal diseases (e.g., NP), oral Kaposi sarcoma, and oral non-Hodgkin lymphoma.
Periodontal treatment in patients with HIV/AIDS	In general, special precautions are not necessary while performing nonsurgical and surgical periodontal treatments if the disease is well controlled and infection control can be done properly. Antibiotics should be prescribed with caution for these patients to avoid the risk of opportunistic infections and microorganism resistance.

Core Knowledge

Rationale for Revision of the Classification of Necrotizing Periodontal Diseases

In the 1999 classification, the term Necrotizing Periodontal Diseases (NPD) included the conditions necrotizing ulcerative gingivitis(NUG) and necrotizing ulcerative periodontitis (NUP). Since ulceration was considered to be secondary to the necrosis, the term 'ulcerative' was later dropped favouring the new terminology necrotizing gingivitis (NG) and necrotizing periodontitis (NP). Research shows that NPD patients are:

- Prone to recurrent disease
- Susceptible to developing a 'chronic' form of the condition with a slower rate of destruction
- Capable of progression to other oral lesions such as necrotizing stomatitis (NS) or noma.

Though both diseases (NG and NP) were associated with a compromised host immune response, this was a rather simplistic view. Actually, there exist major differences in prevalence, progression, extent and severity of NPD among patients with different predispositions.[1] Table 18.1 reviews the proposed updated classification for necrotizing forms of periodontal diseases.

CLINICAL CORRELATE

What is the relationship between necrotizing gingivitis (NG) and necrotizing periodontitis (NP)?

The lesions of NG are confined to the gingiva without a loss of periodontal attachment or alveolar bone support; this is the feature that distinguishes NG from NP.

Studies have suggested that NG and NP may represent different stages of the same disease, due to similar etiology, clinical characteristics, and treatment, with the potential to progress to more severe forms such as necrotizing stomatitis (NS) and noma.

Until a clear distinction between NG and NP can be proved or disproved, it has been suggested that NG and NP be classified together under the broader category of necrotizing periodontal diseases (NPD), albeit representing differing levels of severity.[2]

TABLE 18.1 Classification of Necrotizing Periodontal Diseases

	NPD in Chronically, Severely Compromised Patients		NPD in Temporarily and/or Moderately Compromised Patients
Clinical Condition	May represent a severe and even life-threatening condition (e.g., HIV/AIDS patients or in malnourished children)		Normally seen as a nonthreatening condition in patients with a systemic compromise of limited duration (e.g., smokers, stressful situations in students or the military)
	Higher risk of faster and more severe progression from NG to NP, and even to NS and noma.		NG may not progress, although the lesions would be different if they affected a gingivitis or a periodontitis patient
Patients	**Adults**	**Children**	**Gingivitis/Periodontitis**
Predisposing conditions	• HIV/AIDS with CD4⁺ counts < 200 and detectable viral load • Other severe systemic conditions that cause immunosuppression	• Severe malnourishments (retinol, zinc, ascorbate) • Extreme living conditions (debilitating childhood diseases, living near livestock, poor oral hygiene, poor sanitary disposal of human and animal fecal waste) • Severe infections (measles, herpes viruses, chickenpox, malaria, febrile illness)	• Psychological stress and insufficient sleep • Malnutrition • Smoking • Previous NPD: residual craters • Local factors: root proximity, tooth malposition, inadequate oral hygiene, preexisting gingivitis

AIDS, acquired immunodeficiency syndrome; CD, cluster of differentiation; CD4⁺, helper T cells; HIV+, human immunodeficiency virus positive; NG, necrotizing gingivitis; NP, necrotizing periodontitis; NPD, necrotizing periodontal diseases; NS, necrotizing stomatitis.
Adapted from Herrera, D., Retamal-Valdes, B., Alonso, B., & Feres, M. (2018). Acute periodontal lesions (periodontal abscesses and necrotizing periodontal diseases) and endo-periodontal lesions. *Journal of Periodontology*, *89*(Suppl. 1), S85–S102.

Necrotizing Periodontitis

Impaired host resistance to infection appears to be a significant factor in the onset and progression of NP. For example, the compromised immune status in HIV-infected or AIDS patients renders them vulnerable to opportunistic periodontal infections. Such periodontal diseases merit deep consideration because they represent the most severe dental biofilm-associated disease sequelae, characterized by very rapid tissue destruction. Necrotizing periodontal diseases present three typical clinical features:[3]

- papilla necrosis
- gingival bleeding
- pain

Fig. 18.1 shows the clinical and histopathological features and the etiology of NP.

Pathology and Management of Periodontal Problems in Patients With HIV Infection

Acquired immunodeficiency syndrome (AIDS) is an infection-driven suppression of the host immune system that is caused by the human immunodeficiency virus (HIV). The oral and periodontal manifestations of this disease and their treatment protocols are reviewed in this section.

Oral Manifestations of HIV Infection

Oral lesions are common in HIV-infected patients; the dentist must be prepared to diagnose and manage these conditions in collaboration with the patient's physician. Fig. 18.2 reviews the oral conditions commonly seen in patients with HIV infection and AIDS.

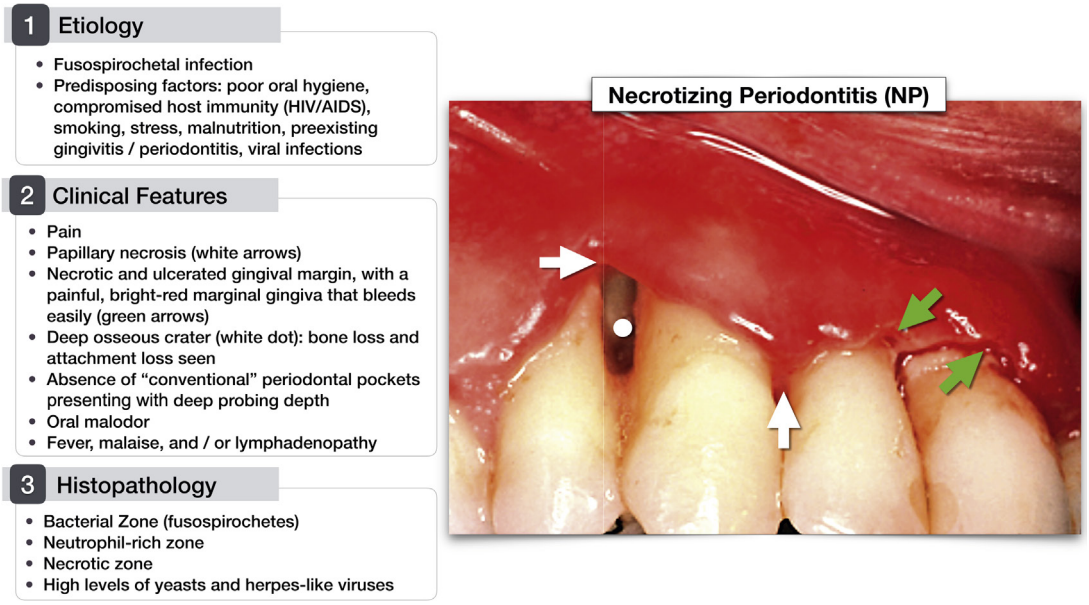
Gingival and Periodontal Diseases in HIV Infection

Periodontal diseases are more common among HIV-infected users of injected drugs, but this may also be related to poor oral hygiene and a lack of dental care rather than just decreased CD4⁺ cell counts. Fig. 18.3 reviews common periodontal diseases in HIV-infected patients.

Periodontal Treatment Protocol in HIV Infection

Delayed wound healing and an increased risk of postoperative infection are possible complicating factors in patients with HIV infection or AIDS, but neither concern should significantly alter treatment planning for an otherwise healthy, asymptomatic HIV-infected patient with a normal or near-normal CD4⁺ cell count and a low viral load. Management considerations that are important while treating patients with HIV infection are:

- Health status—history of viral loads and CD4⁺ cell counts, duration of HIV infection from time of exposure, history of drug abuse or intravenous drug usage, current medications, and adverse effects experienced must be noted.
- Infection control—strict compliance with sterilization protocols is essential.
- Goals of dental therapy:
  - Primary goal should be the restoration and maintenance of oral health, comfort, and function
  - Control of HIV-associated mucosal diseases (e.g., chronic candidiasis and recurrent oral ulcerations)
  - Management of acute periodontal and dental infections
  - Provision of detailed oral hygiene instructions



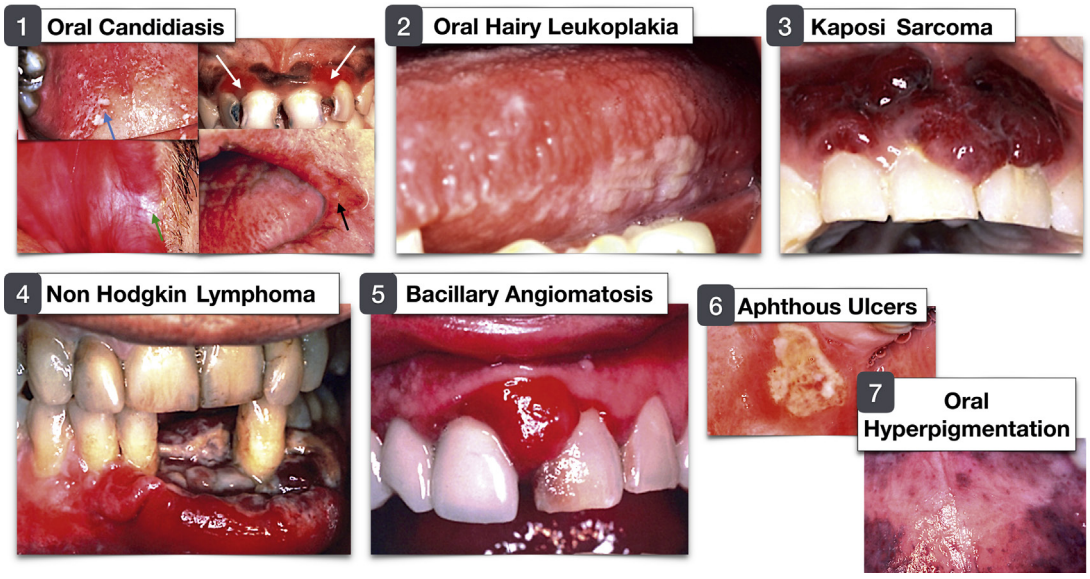
• **Fig. 18.1** Necrotizing Periodontitis: Clinical Features, Microscopic Findings, and Etiology. NP is an infectious condition; clinical improvements observed after mechanical debridement and antimicrobial treatment lends support to the bacterial etiology of this condition. However, predisposing factors, including a compromised host immune response, are critical in its pathogenesis. NP could be the result of (1) one or various episodes of NG or (2) NG occurring at a site previously affected by periodontitis (in this case, periodontal pocketing would be found). Note that pocket formation is not the *direct* result of NP, as this disease destroys JE cells. Apical migration of viable JE cells along the root surface is required for pocket formation; necrosis of the JE in NP creates an ulcer that prevents this epithelial migration, and a pocket cannot form.

Clinical features: Diagnosis of NP is mainly based on clinical findings, with microbiological assessment or biopsies being recommended for atypical or nonresponding cases. In addition to the features presented in the figure, bone sequestration may occur in cases of severe immunosuppression. Advanced NP lesions may lead to severe bone loss and tooth mobility, and ultimately to tooth loss.

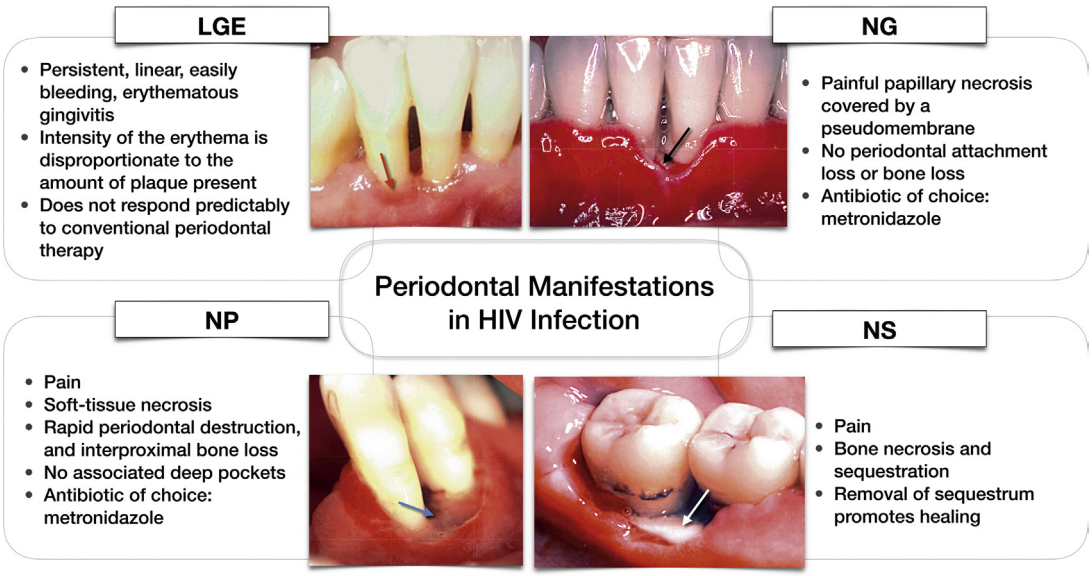
Microscopic findings: Include (1) a surface biofilm composed of a mixed microbial flora with different morphotypes, and a subsurface flora with dense aggregations of spirochetes (i.e., the bacterial zone); dense aggregations of PMNs below the bacterial layers (i.e., the neutrophil-rich zone), and necrotic cells (the necrotic zone). The finding of yeasts and herpes-like viruses is most likely due to opportunistic infections occurring in immunocompromised hosts.[1,4]

JE, junctional epithelium; NG, necrotizing gingivitis; NP, necrotizing periodontitis; PMN, polymorphonuclear leukocyte.

(From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)



• **Fig. 18.2** Oral Manifestations of HIV Infection. A strong correlation exists between HIV infection and oral candidiasis, oral hairy leukoplakia, oral Kaposi sarcoma, and oral non-Hodgkin lymphoma. Oral lesions less strongly associated with HIV infection include melanotic hyperpigmentation, recurrent aphthous stomatitis, and bacillary angiomatosis (epithelioid angiomatosis). (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)



• **Fig. 18.3** Periodontal Manifestations of HIV Infection. Nonspecific bacterial infections flare up in HIV infections, especially when $CD4^+$ cell counts are significantly depressed. Periodontal conditions seen due to immunosuppression are LGE and necrotizing periodontal diseases like NG and NP, and NS. Common treatment protocols for all these conditions include local debridement, scaling and root planing, in-office irrigation with an effective antimicrobial agent (e.g., chlorhexidine gluconate or povidone–iodine [Betadine]), and the establishment of meticulous oral hygiene, including the home use of antimicrobial rinses or irrigation. If an antibiotic is necessary, metronidazole is the drug of choice, with a prophylactic prescription of a topical or systemic antifungal agent to avoid opportunistic candidiasis. *HIV*, human immunodeficiency virus; *LGE*, linear gingival erythema; *NG*, necrotizing gingivitis; *NP*, necrotizing periodontitis; *NS*, necrotizing stomatitis. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

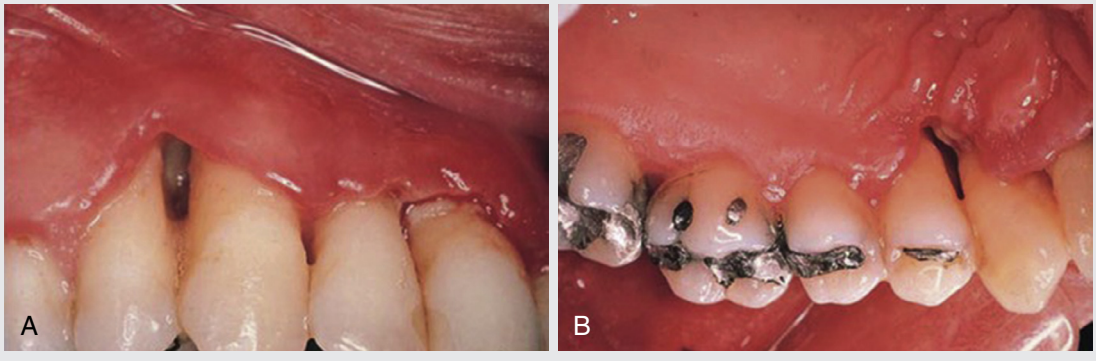
- Conservative nonsurgical periodontal therapy should be a treatment option for virtually all HIV-positive patients.
- Elective periodontal procedures should be performed after obtaining medical clearance.
- Maintenance therapy should be performed every 2–3 months.
- Addressing psychological factors:
  - Coping with a life-threatening disease may elicit depression, anxiety, and anger in patients; this anger may be directed toward the dentist and the staff. An empathetic and sensitive approach is required.
  - Patients may be greatly concerned with the maintenance of medical confidentiality, and such confidentiality must be upheld.
  - If the dentist elects to request testing for the HIV antibody, the patient must be informed. In most circumstances, written informed consent is desirable before testing.

This chapter was developed from Chapters 29 and 30 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

CASE-BASED LEARNING EXERCISE

Scenario: A 38-year-old Caucasian male presented with the chief complaint: "There's a gap between my teeth. It hurts, and my teeth are loose." Patient has a known history of HIV/AIDS. His last CD4 count was 150 cells/μL, and his viral load was undetectable. He had no known drug allergies. The patient reported multiple episodes of recurrent pneumonia over the past year. He had a history of smoking (one pack per day for 28 years). Current findings: Generalized probing depth from 2 to 4 mm. Severe, localized recession with necrosis and ulceration of the interproximal tissues between the maxillary right premolar and canine (Fig. A). Localized moderate-severe biofilm with associated moderate-severe gingival erythema.

Clinical photo from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.



Questions

1. Which of the following is NOT an etiologic/predisposing factor for NP?
   - **a.** Stress
   - **b.** Malnutrition
   - **c.** Viral infection
   - **d.** Diabetes mellitus
2. Based on the case description, what will be the correct diagnosis according to the AAP-EFP(2017) classification?
   - **a.** Necrotizing periodontal disease (NPD) in temporarily moderately compromised patients
   - **b.** NPD in chronically moderately compromised patients
   - **c.** NPD in chronically severely compromised patients
3. After periodontal therapy has been completed, how often would you recommend maintenance therapy for this patient?
   - **a.** Every 2 weeks
   - **b.** Every month
   - **c.** Every 3 months
   - **d.** Every 6 months
4. Select the most likely antibiotic of choice for this condition.
   - **a.** Amoxicillin
   - **b.** Azithromycin
   - **c.** Clindamycin
   - **d.** Metronidazole

Case-Based Learning Exercise

Solutions

1. Answer: d

Explanation: All of the options except (d) have been identified as etiologic/predisposing factors (see Fig. 18.1). Other factors include poor oral hygiene, compromised host immunity (HIV/AIDS), smoking, and preexisting gingivitis/periodontitis.

2. Answer: c

Explanation: Given that the patient has a known history of HIV/AIDS with $CD4^+$ count of 150 cells/μL, the diagnostic category is NPD in chronically, severely compromised patients.

3. Answer: c

Explanation: Considering the significantly immunocompromised medical history, maintenance therapy is most likely to be recommended every 2–3 months.

4. Answer: d

Explanation: Metronidazole is the antibiotic of choice, but because this patient is severely immunocompromised, the use of antibiotics has to be restricted due to a higher incidence of opportunistic infections (e.g., oral candidiasis) with antibiotic use (see Fig. 18.3).

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19

Clinical and Radiographic Evaluation in Periodontics

Relevant Terminology

Terminology	Explanation
automatic and electronic periodontal probing	• Classical probing presents some problems in reproducibility of the measurements of probing depth and attachment loss • New commercially available computer-assisted technology may be used to improve probing accuracy and reproducibility • This method combines the advantages of a constant probing force with precise electronic measurement and computer storage of data, thereby eliminating the potential errors associated with visual reading and the need for an assistant to record the measurements • For example, the Florida Probe System consists of a probe handpiece, a digital readout, a foot switch, a computer interface, and a computer
biologic depth	The histologic distance between the gingival margin and the base of the gingival crevice (the coronal end of the junctional epithelium)
bite-wing radiography	Bite-wing radiographs can show alveolar bone levels more accurately than other radiographs because it is easier to place the film parallel to the long axis of the targeted teeth. Vertical bite-wing is the radiograph of choice in patients with periodontitis.
clinical attachment loss	Refers to the amount of periodontal tissue loss measured in reference to a stable landmark such as the cementoenamel junction. Clinical attachment loss, measured in millimeters, represents the severity of periodontitis.
dental stains	Pigmented deposits on the teeth
full mouth series of intraoral radiographs	A full mouth series of intraoral radiographs (usually consisting of 14 periapical and 4 posterior bite-wing radiographs) is required for periodontal diagnosis and treatment planning. These radiographs should be updated on a case-by-case basis, once every 2 years for a patient with moderate to severe periodontitis.
long-cone paralleling technique	The most reliable technique to assess alveolar bone level. The film should be positioned parallel to the long axis of the targeted teeth, and the x-ray beam should be directed at a right angle to the teeth and film.
probing depth	The distance from the gingival margin to the base of the gingival crevice where the probe tip stops. The tip usually penetrates beyond the coronal end of the junctional epithelium. The penetration is deeper when the inflammation is more severe.
tooth wear	Any gradual loss of tooth substance, characterized by the formation of smooth, polished surfaces. It can be: • Abrasion—loss of tooth substance that is induced by mechanical wear other than that of mastication (e.g., aggressive tooth brushing by horizontal scrubbing motion). Abrasion results in saucer-shaped or wedge-shaped indentations with a smooth, shiny surface. • Attrition—occlusal wear that results from functional contact with opposing teeth. Occlusal or incisal surfaces worn by attrition are called *facets.* Facets generally represent functional or parafunctional wear or iatrogenic dental treatment through coronoplasty (occlusal adjustment). • Abfraction—results from occlusal loading causing tooth flexure and mechanical microfractures in the cervical area. • Erosion—Loss of tooth structure caused primarily by chemical process (acidic beverages or citrus fruits) beyond contribution from dental plaque.

Fast Facts

Health history	Health history, including information on medical problems, tobacco/alcohol use, past surgeries and hospitalizations, physical examination results, and medications, has to be obtained in the first visit by means of a questionnaire or verbally
Dental history	Dental history includes records of past dental visits, received dental treatments, oral hygiene regimen, current symptoms, and parafunctional habits like grinding/clenching habit (bruxism)
Examination of extraoral structures	Examination of extraoral structures includes assessing temporomandibular joints, masticatory muscles, lymph nodes of the head and neck, and facial symmetry
Examination of the oral cavity	Examination of the oral cavity includes examining oral hygiene status, oral malodor (halitosis), and various regions of the oral cavity like the floor of the mouth, tongue, palate, mucosa, and oropharyngeal area
Examination of the periodontium	The examiner should visually examine the degree of biofilm and calculus accumulation, gingival texture, and appearance before probing. Probing includes measuring probing depth, bleeding on probing, and other periodontal parameters.
Calculus accumulation	Supragingival calculus can be commonly identified on the lingual surfaces of the mandibular anterior teeth and buccal surfaces of the maxillary molars due to the presence of duct openings of salivary glands and ineffective brushing. Subgingival calculus usually has to be detected by probing.
Visual examination of gingiva	Gingiva has to be dry before it can be properly examined. Gingival inflammation induced by biofilm usually begins at the gingival margin; the severity of inflammation is usually consistent with biofilm/calculus accumulation.
Tactile examination of gingiva	A periodontal probe can be used to assess consistency, gingival adaptation, bleeding, and probing depth of gingiva. If the gingiva is inflamed, bleeding can easily be seen by gentle probing (using a force of 0.25 N). While probing, it is important to circumferentially walk the probe to avoid missing any deep pocket.
Timing of probing	Periodontal probing sometimes is not accurate when the patient has severe inflammation due to discomfort and/or calculus accumulation • When discomfort is present, the examiner should confirm probing when gingiva is anesthetized for treatment • In moderate and advanced cases, probing depths will change dramatically with improved biofilm control and scaling and root planing. In such cases, obtaining accurate probing depths is much more important at the reevaluation following nonsurgical therapy than at the pretreatment periodontal examination.
Bleeding on probing	Probing can induce bleeding when the gingiva is inflamed. Bleeding on probing (BOP) may occur 30–60 seconds after probing. Absence of BOP is a good predictor for periodontal stability at a single site. Overall BOP percentage is correlated with increased risk of periodontal disease progression.
Evaluation of tooth mobility	Tooth mobility should be measured by holding the tooth using flat ends of two metallic instruments, then moving the tooth to evaluate how much the tooth moves in horizontal and vertical directions. Increased tooth mobility is usually associated with bone loss, trauma from occlusion and/or periodontal/periapical inflammation.
Adequate angulation of periapical radiographs	There are four criteria to determine the adequate angulation of a periapical radiograph: • The tips of the molar cusps should be seen without showing the occlusal surface • Enamel cusp and pulp chambers should be clearly visible • Interproximal spaces should be seen clearly • Proximal contacts should not overlap
Bone level in radiographs	It is important to be aware of the potential errors caused by film/receptor angulation, defect pattern, and anatomic variants in identifying bone level in radiographs. Generally, the distance between the cementoenamel junction and the alveolar bone crest is approximately 2 mm in healthy periodontium.
Pattern of bone destruction	When the alveolar bone height is reduced with crestal bone parallel to the imaginary line joining the cementoenamel junction levels of adjacent teeth bone loss is horizontal. When the alveolar bone height is reduced with crestal bone not parallel (oblique destruction pattern) to the cementoenamel junction levels of adjacent teeth, bone loss is vertical.
Radiographic appearance of periodontitis	For a periodontitis patient, reduced bone height, disruption of lamina dura, and/or furcation involvement can be seen in radiographs. Actual bone loss is usually greater than radiographic appearance; therefore it is important to use both clinical and radiographic findings to make a diagnosis.
Cone beam computed tomography in diagnosing periodontitis	Cone beam computed tomography (CBCT) provides three-dimensional images showing defects that cannot be seen in two-dimensional images. However, the radiation exposure and costs of CBCT are higher than for two-dimensional radiography; CBCT should not be routinely used for periodontal diagnosis.

Core Knowledge

Clinical Diagnosis

The process of clinical diagnosis includes an overall appraisal of the patient, recording chief complaint, history taking, a clinical examination (which involves evaluation of the periodontium and the teeth), and a radiographic examination, supplemented by microbiologic and histopathologic tests and assessment of host immunity. Once a provisional diagnosis is established, risk factor assessment is also done in order to arrive at a working prognosis and draft a treatment plan (Fig. 19.1).

CLINICAL CORRELATE

What are the factors that affect probing depth?

- **Clinical factors:** Probe penetration can vary depending on the probing force, the size of the probe tip, the direction of penetration, tissue resistance, the convexity of the crown, and the degree of tissue inflammation.[1] Probing depth is generally ≤3mm in gingival health and >3mm in the presence of gingival inflammation.
- **Histologic factors:** The probe is "walked" circumferentially around the surface of each tooth to detect the areas of deepest depth. Attachment of the junctional epithelium to the tooth surface stops probe penetration in healthy tissue. If the gingival tissue is severely inflamed, the probe may not encounter any resistance at all and penetrate beyond the junctional epithelium into connective tissue/ bone crest.[2]

Examination of the periodontium consists of two parts:
1. Visual examination (Fig. 19.2)
2. Tactile examination (Fig. 19.3).

Radiographic Aids in the Diagnosis of Periodontal Disease

Clinical diagnosis is greatly enhanced by the use of radiographs, which play an integral role in the assessment of periodontal disease.

CLINICAL CORRELATE

What is the role of the x-ray beam in the appearance of the radiographic image?

A dental radiographic image is produced when an x-ray beam passes through oral structures and is captured on a digital sensor or analogue film which has to be processed to see an image. The *gray level* (range of black to white) of the exposed radiographic film at any particular spot is a reflection of the amount of bone or other calcified structure (and, to a lesser extent, soft tissue) in the path of the x-ray beam.

In order to interpret a radiograph properly, one must understand the way in which x-rays are attenuated by the mineralized and non-mineralized structures of the periodontium.[3]

The radiograph reveals alterations in calcified tissue; it does *not* reveal current disease activity, but shows the effects of past cellular experience on the bone and roots.

Normal Radiographic Anatomy of the Periodontium

Radiographic assessment of the diseased periodontium is efficient when one has a clear understanding of the normal radiographic anatomy of the alveolar bone, periodontal ligament space, the lamina dura, and interdental septum (see Fig. 19.4).

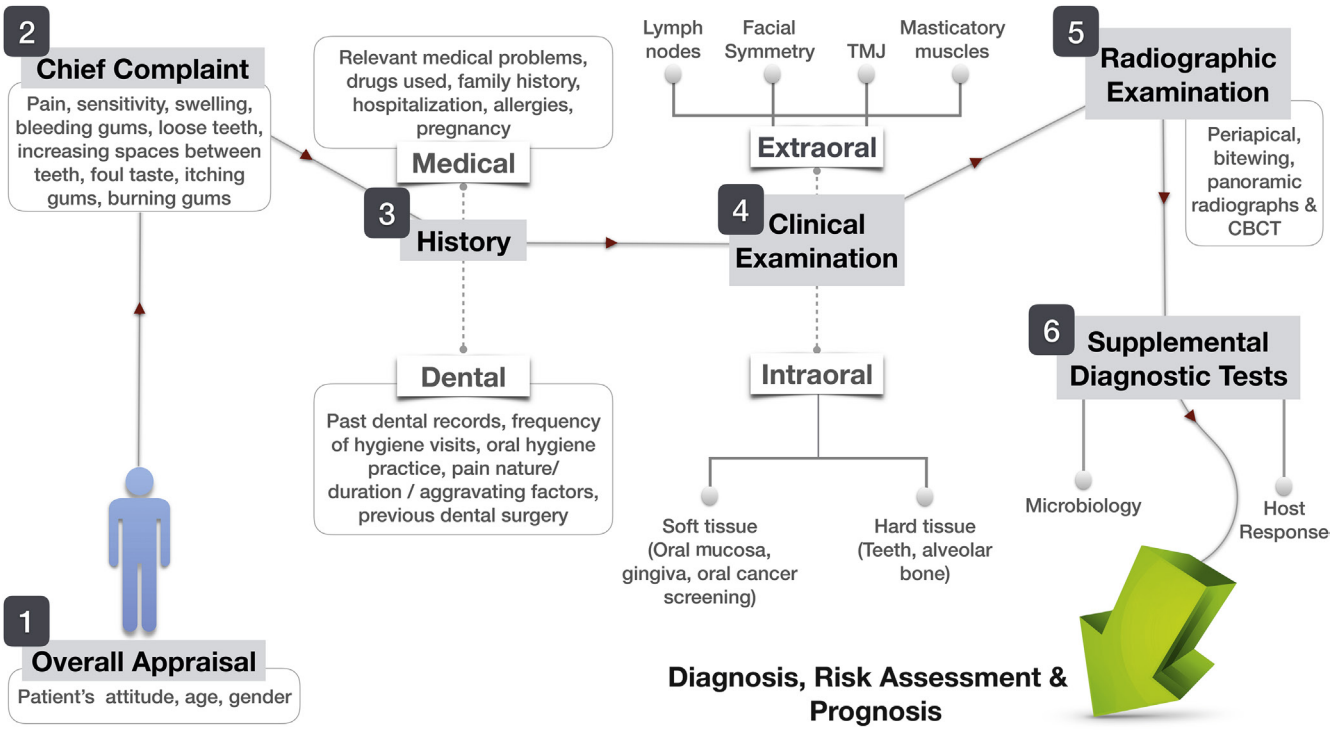
Use of Radiographs in Periodontal Conditions

Radiographs are useful for the following information[5]:
- To get an idea of the existing bone volume
- To assess the alveolar crests and diagnose bone loss patterns that have implications for treatment planning and prognosis
- To assess bone loss in the furcation areas
- To assess the width of the periodontal ligament space
- To identify local (plaque-retentive) factors that cause or intensify periodontal disease, such as:
  - calculus
  - poorly contoured or overextended restorations
  - biologic width violations;
- To assess root length, morphology, root proximity and crown-to-root ratio (abutment evaluation of periodontally affected teeth for support)
- To evaluate anatomic considerations such as:
  - proximity of maxillary sinus
  - missing, supernumerary, or impacted teeth that may cause periodontal problems
- To assess pathologic conditions such as:
  - decay (especially root surface caries)
  - periapical pathology
  - root resorptions
- To assess bone and related anatomic structures as part of presurgical assessment for patients who are candidates for dental implant therapy

Fig. 19.5 reviews commonly seen radiographic appearances of periodontal disease and trauma from occlusion.

Limitations of Radiographs

Although radiographs play a vital role in treatment planning, careful clinical examination must always supplement its use for the following reasons[5]:
1. They provide only a two-dimensional view of a three-dimensional situation. This prevents bony defects superimposed by higher bone walls (e.g. interdental craters) from being seen clearly. Besides, due to tooth roots overlapping buccal/lingual cortical plates, only interdental bone can be clearly seen.
2. Radiographs underestimate bone destruction especially in mild cases.
3. Radiographs cannot help diagnose soft tissue /pseudo pockets or clinical attachment loss.
4. Radiographs cannot distinguish between treated and untreated cases.

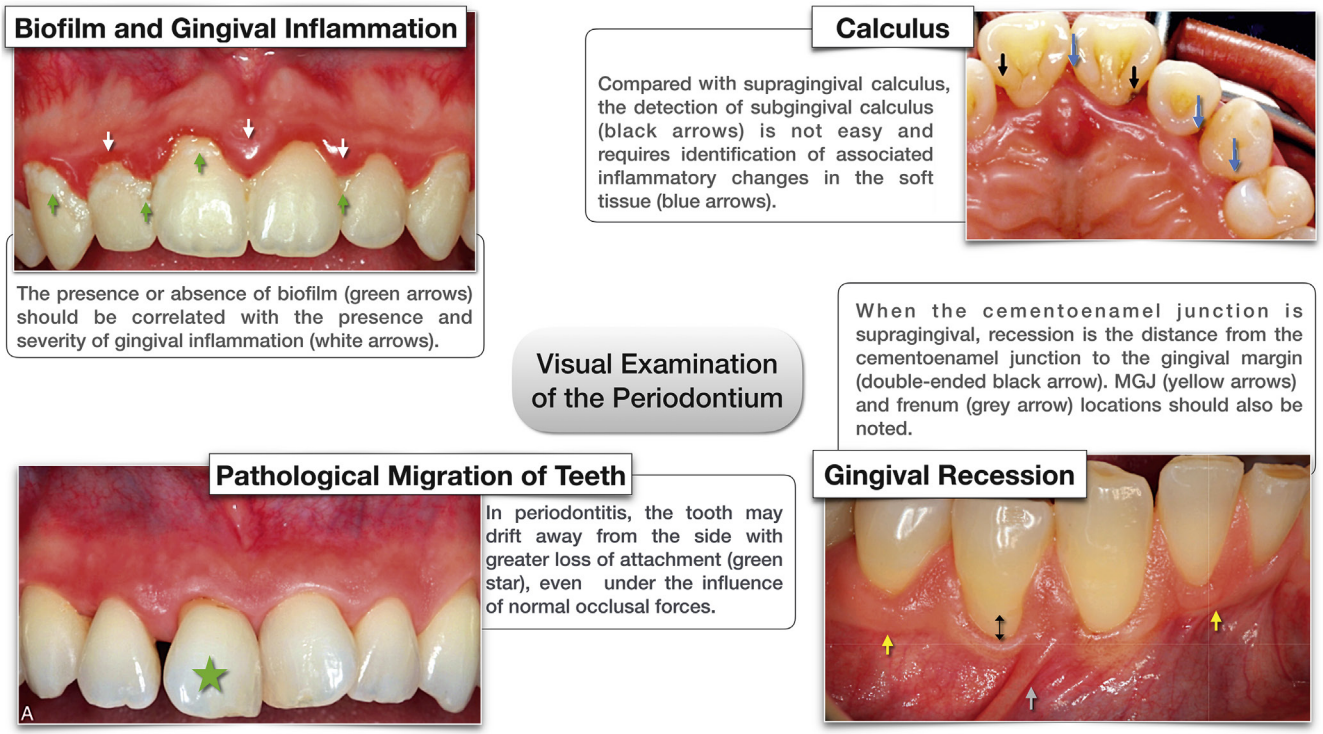


• **Fig. 19.1** Clinical Diagnosis: Workflow.

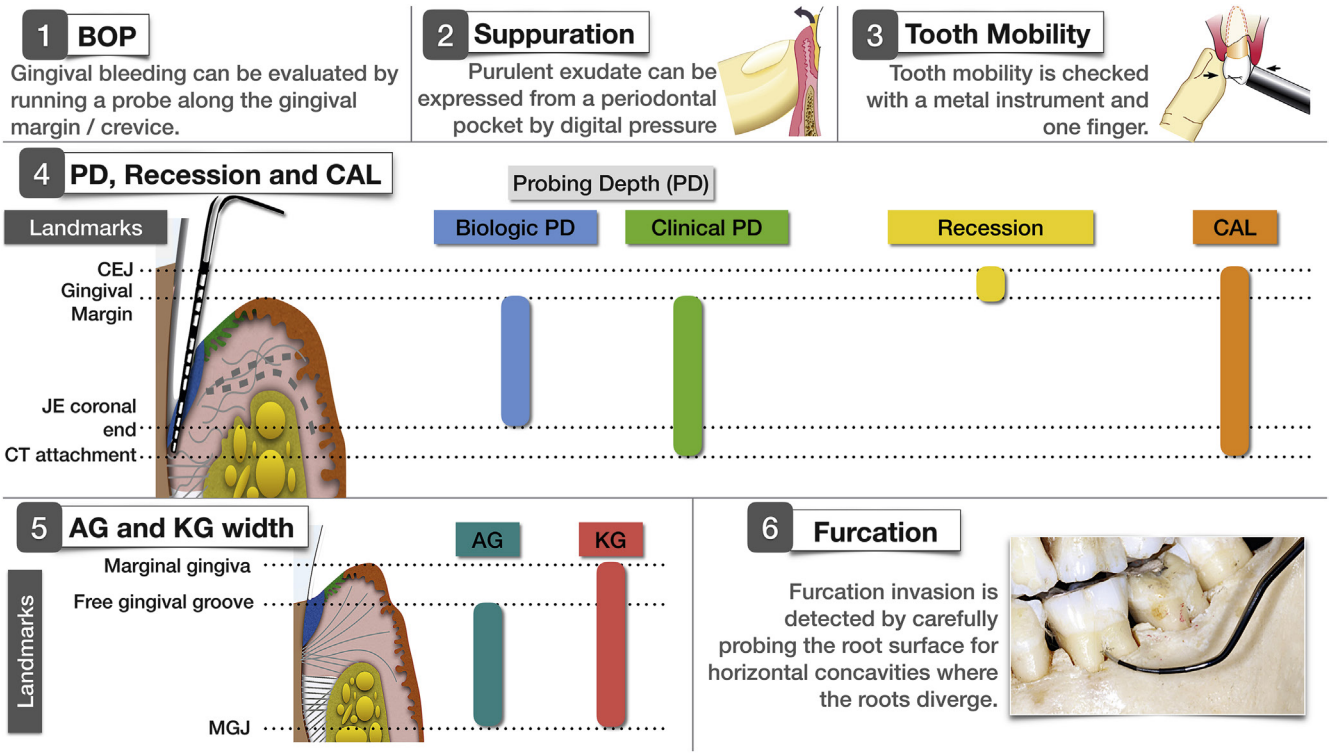
Diagnostic procedures must be systematic and organized, with a proper flow to the process. In addition to assembling facts, the clinician must piece together findings to provide a meaningful explanation of the patient's periodontal problem. A recommended sequence of procedures for the diagnosis of periodontal diseases is:

1. **Overall appraisal of the patient**—the interest should be in the patient who has the disease, not just in the disease itself. Diagnosis must therefore include a general evaluation of the patient.
2. **Chief complaint.**
3. **Relevant history**—in order to avoid unwitting omission of relevant information by the patient (due to lack of awareness regarding the impact of certain systemic diseases and drugs on dental/oral health), the patient should be made aware of the following:
   (i) the possible impact of certain systemic conditions and medications on periodontal disease, its treatment, and treatment outcomes
   (ii) the requirement for special precautions or modifications of treatment procedures in certain systemic conditions
   (iii) the possibility that oral infections may have a significant influence on the occurrence and severity of a variety of systemic diseases/conditions
4. **Clinical examination:**
   Extraoral—after assessing for facial asymmetry (e.g., any swelling due to abscess formation), the TMJ should be assessed for pain, crepitus, clicking, and range of motion. The muscles of mastication should be palpated for pain and tenderness. Periodontal, periapical, and other oral diseases may result in lymph node changes, and routine evaluation of the lymph nodes of the head and neck must be performed.
   Intraoral—the lips, floor of the mouth, tongue, palate, vestibule, and the oropharyngeal region should be evaluated for abnormalities and pathologies, including screening for oral cancer. The cleanliness of the oral cavity should be appraised in terms of the extent of accumulated food debris, biofilm, calculus, and tooth surface stains and dental caries. Periodontal examination is discussed in Figs. 19.2 and 19.3.
5. **Radiographic examination**—panoramic radiographs provide an informative overall radiographic picture of the distribution and severity of bone destruction with periodontal disease and surrounding oral structures, but a complete intraoral series (14 periapical and 4 bite-wing radiographs) may be required for detailed periodontal diagnosis and treatment planning. A CBCT helps with planning complex cases requiring dental implants and implant site development.
6. **Supplemental diagnostic tests**—when unusual gingival or periodontal problems are detected that cannot be explained by local causes, the possibility of microbiological and host factors contributing to disease must be explored. Bacterial culture of plaque samples obtained from pockets, testing antibiotic susceptibility, dark-field microscopy, DNA tests, bacterial enzyme testing (BANA test), and so on are supplemental aids in testing microbial etiology. Tests of host response include genetic susceptibility tests (e.g., IL-1 gene polymorphism).
7. **Risk Assessment**—the reader is referred to Chapter 20 for more details.

CBCT, cone-beam computed tomography; *TMJ*, temporomandibular joint.



• **Fig. 19.2** Visual Examination of the Periodontium.
The systematic process of periodontal examination should not immediately begin with insertion of the periodontal probe into the gingival crevice. Not only can this be uncomfortable and traumatic for a patient, but it may induce bleeding and make visualization of inflammatory changes in the soft tissue challenging. Periodontal disease occurs as a result of response of host tissue to the biofilm accumulation: therefore a thorough visual examination assessing the gingival margin for biofilm / calculus accumulation and inflammatory changes is vital. Gingival recession and frenal pulls on marginal gingiva should be noted, and assessment of the width of keratinized gingiva should also be undertaken (discussed in Chapter 36). Alterations in tooth position should be carefully noted; they may be caused by, for example, periodontitis, lack of posterior stops due to missing posterior teeth that cause a bite collapse resulting in anterior teeth flaring out, or abnormal forces from a tongue thrusting habit. Only after visual examination is finished should probing of the gingival crevice and periodontal charting begin. *MGJ*, mucogingival junction.
From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.



• **Fig. 19.3** Tactile Examination of the Periodontium.

Tactile periodontal examination begins with the probing of the gingival crevice and the tooth surface to check for aberrations, concavities, and subgingival calculus. The response of the gingival tissue to probing is evaluated in terms of resistance to probe penetration, depth of probe penetration, and pain on probing. This is followed by a series of periodontal assessments:

1. **Assessment of bleeding on probing**—under pressure, healthy gingival tissue will blanch and not bleed, but in the presence of gingival inflammation, marginal bleeding may be observed. The ease and severity of marginal bleeding are correlated with the severity of gingival inflammation. Sometimes, bleeding appears immediately after probe removal, but it may be delayed for a few seconds. After 30 to 60 seconds of probing, the clinician should recheck for bleeding. The absence of bleeding on probing is an excellent predictor of periodontal stability.
2. **Assessment of pocket suppuration**—palpation of the marginal gingiva with a probe, or digitally by placing the ball of the index finger on the gingiva apical to the margin and pushing coronally toward the gingival margin, may squeeze a whitish-yellow exudate from the gingival crevice. Note that absence of suppuration does not indicate absence of disease.
3. **Assessment of tooth mobility**—physiologic mobility is movement up to 0.2 mm horizontally and 0.02 mm axially. Mobility beyond the physiologic range is termed abnormal or pathologic. The three main etiologic factors of tooth mobility are periodontal inflammation, attachment loss, and occlusal trauma. Mobility is scored according to the ease and extent of tooth movement using the Miller Index:
   - Grade 1: first clear sign of movement greater than physiological limit.
   - Grade 2: crown movement up to 1 mm (any direction)
   - Grade 3: crown movement more than 1 mm in any direction, or vertical depression or rotation of the crown in its socket.
4. **Assessment of probing depth, gingival recession, and clinical attachment loss:**
   - **Probing depth (PD)—**when probing, the probe tip should be in contact with the tooth surface as it slides down to the bottom of the gingival sulcus, allowing detection of tooth surface irregularities, subgingival calculus and furcation involvements. There are two different pocket depths:
     - The biologic or histologic depth: the distance between the gingival margin and the base of the gingival crevice (i.e., the coronal end of the JE). This can be measured only in histologic sections.
     - The clinical or probing depth: the distance from the gingival margin to where the probe tip stops. In inflamed gingiva, usually this point is where the probe encounters the most coronal intact fibers of the CT attachment.
   - **Gingival recession—**recession is the distance from the CEJ to the gingival margin. The presence of recession indicates that attachment loss has occurred, but not necessarily that inflammation is present.
   - **Clinical attachment loss (CAL)—**CAL measures the amount of attachment loss that has occurred, with the CEJ as the reference point. It is measured as the distance from the CEJ to the bottom of the probeable crevice. In inflamed gingiva, usually this point is where the probe encounters the most coronal intact fibers of the CT attachment or sometimes even bone. CAL is the sum of gingival recession and clinical PD.

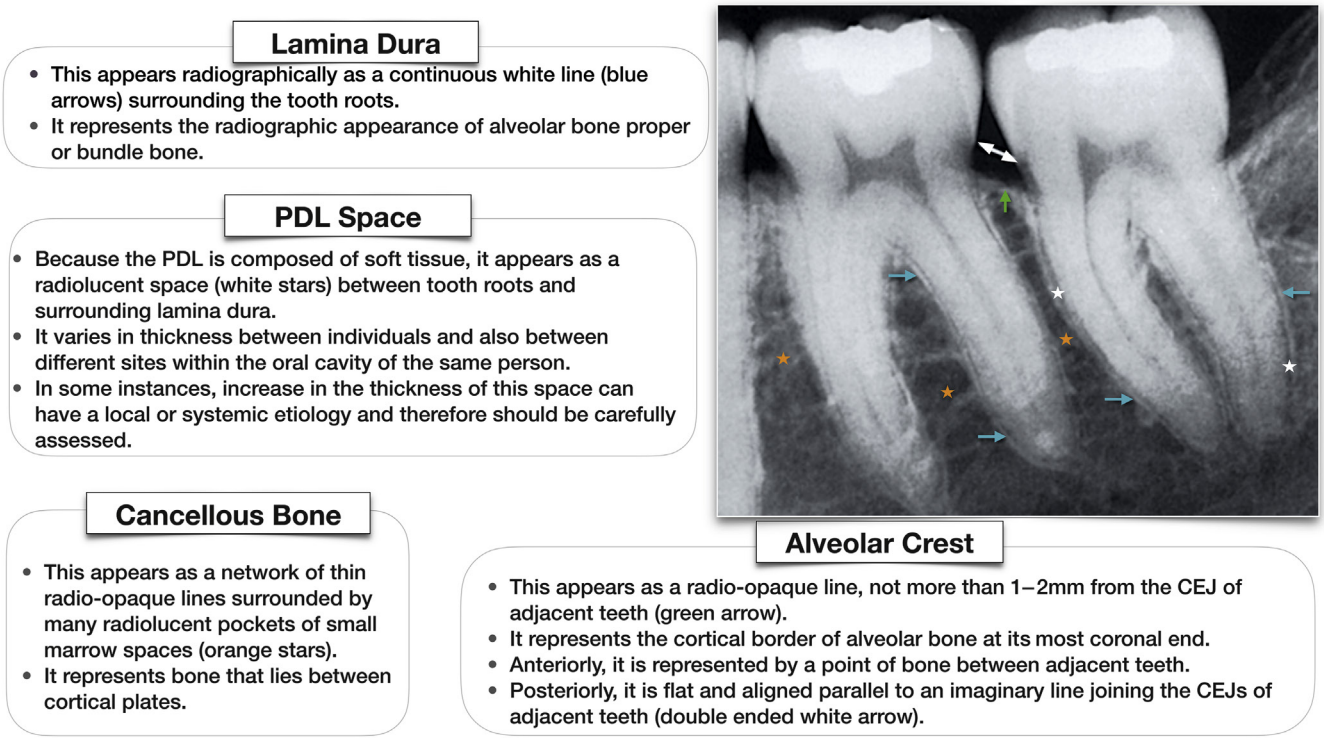
• **Fig. 19.3, cont'd**

5. **Assessment of width of attached gingiva (AG) and keratinized gingiva (KG)**—the width of the attached gingiva is the distance between the MGJ and the projection on the external surface of the bottom of the gingival sulcus or the periodontal pocket (when present, this landmark is called the free gingival groove). It should not be confused with the width of the KG, because the latter also includes the marginal gingiva.
6. **Assessment of furcation involvement**—furcation invasion is the pathologic resorption of interradicular bone within a furcation of a multirooted tooth due to periodontal disease. It is detected using the Nabers probe. (See Chapter 35 for further reading on furcation diagnosis and management).

Disclaimer: The graphics in this figure are diagrammatic representations to aid in concept learning and are not representative of actual dimensions of tissues or periodontal probe.

AG, attached gingiva; *BOP*, bleeding on probing; *CAL*, clinical attachment loss; *CEJ*, cementoenamel junction; *CT*, connective tissue; *JE*, junctional epithelium; *KG*, keratinized gingiva; *MGJ*, mucogingival junction; *PD*, probing depth.

From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.



• **Fig. 19.4** Normal Radiographic Anatomy of Interdental Alveolar Bone.[5]

Radiographic evaluation of bone changes in periodontal disease is primarily based on how the interdental bone looks in the radiographs, since the facial and lingual bone levels are masked by the radio opaque root structure. This figure shows the normal radiographic appearance of various structures that constitute the interdental bone, namely: the lamina dura, the PDL space, the cancellous bone between the cortical plates, and the alveolar crest.

- In health, interdental bone between posterior teeth remains intact; this healthy appearance is seen as a sharp angle formed between the alveolar bone crest (usually cortical bone) and lamina dura of adjacent roots bordering the interdental bone.
- In posterior dentition, the angulation of the crest of the interdental septum is generally parallel to a line between the CEJs of the approximating teeth. When there is a difference in the level of the adjacent CEJs, the crest of the interdental bone appears angulated rather than horizontal. In this circumstance, such an appearance is considered "normal" and should not be mistaken for angular or vertical bone loss.

CEJ, cementoenamel junction; *PDL*, periodontal ligament. *Radio-opaque* describes the appearance of dense structures that are opaque to the passage of x-rays and appear white on a radiograph; *radiolucent* refers to less dense tissues which appear more black than radio-opaque structures.

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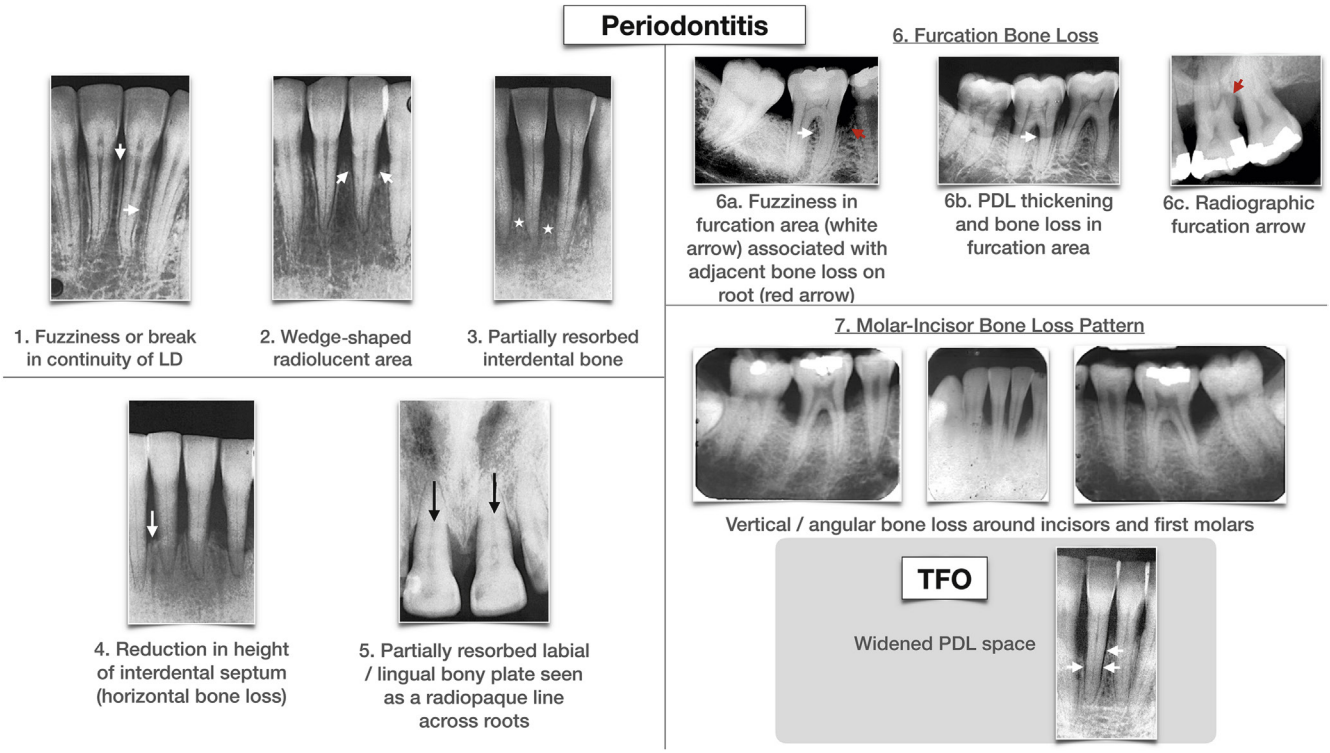


• **Fig. 19.5** Radiographic Appearance of Periodontitis and Trauma From Occlusion (TFO). This figure shows the various ways in which periodontitis can present itself on radiographs, and also the radiographic appearance of TFO.

Radiographic appearance of periodontitis—the earliest changes of periodontal disease often do not manifest on radiographs, or radiographs may underestimate the extent of bone loss. Thus the assessment of periodontal bone level should be based on both the results of clinical and radiographic evaluation. Features are:

1. *Fuzziness and break in the continuity of the lamina dura* at the crest of the interdental septum—considered as the earliest radiographic change in periodontitis. It results from bone resorption activated by extension of gingival inflammation into the alveolar bone. The presence of an intact crestal lamina dura could indicate periodontal health.
2. *A wedge-shaped radiolucent area* at the mesial or distal aspect of crest of interdental septum with the apex pointed in the direction of the root (caused by lateral bone resorption of the interdental septa on either side of root along with PDL space widening).
3. *Interdental bone can appear partially eroded*—due to increased osteoclastic activity that results in increased bone resorption along the endosteal margins of the medullary spaces.
4. *Reduction in the height of the interdental septum*—due to deeper extension of inflammation into the bone.
5. *Radiopaque horizontal line across the roots*—demarcates the portion of the root where the labial or lingual bony plate has been partially or completely destroyed. In the radiograph the radicular bone structure is superimposed on the root structure.
6. *Furcation bone loss*—to assist in the radiographic detection of furcation involvement, the following diagnostic criteria are suggested:
   a. The slightest radiographic change in the furcation area should be investigated clinically, especially if there is bone loss on adjacent roots.
   b. Diminished radiodensity in the furcation area in which outlines of bony trabeculae are visible suggests furcation involvement. Thickened PDL space may also be present.
   c. Whenever there is marked bone loss in relation to a single molar root, it may be assumed that the furcation is also involved. Mesial or distal furcation involvement of the maxillary molars is not obviously visible on periapical radiographs, because the furcations defects may be superimposed with the images of buccal/ palatal cortical plates, and the palatal root as well. Lesions involving mesial and distal furcations have also been described radiographically as furcation "arrows."[4]
7. *Molar-incisor bone loss pattern*—previously called localized aggressive periodontitis, this condition is typically characterized by bone loss in the maxillary and mandibular incisor and/or first molar areas, usually bilaterally, resulting in vertical, arc-like destructive patterns.

Radiographic appearance of TFO—The radiographic changes listed below are not pathognomonic of TFO and must be interpreted in combination with clinical findings, particularly tooth mobility, presence of wear facets, pocket depth, and analysis of occlusal contacts and habits.

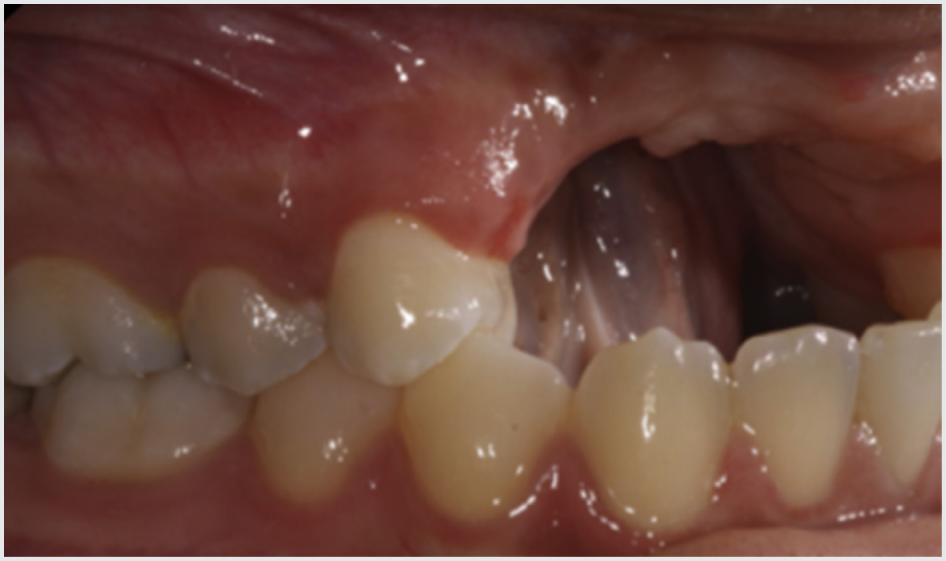
- Widening of the periodontal ligament space
- Widening of the lamina dura
- Bone loss—in terminal stages, deep angular bone loss extends around the root apex, producing a wide, radiolucent periapical image (cavernous lesions)
- Increase in number and size of trabeculae
- Hypercementosis
- Root fractures
- Root resorption

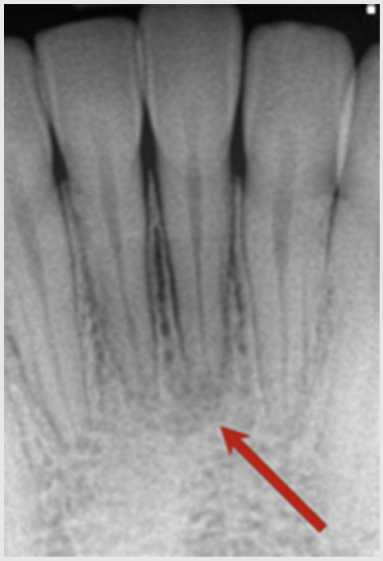
LD, lamina dura; PDL, periodontal ligament; TFO, trauma from occlusion.

From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

CASE-BASED LEARNING EXERCISE

Scenario: A 21-year-old female patient was involved in a traumatic car accident that led to loss of her maxillary anterior teeth; the chief complaint of the patient was to reconstruct this sextant. An interdisciplinary team was convened in order to evaluate the clinical and radiographic findings and to arrive at different treatment options, in order to successfully restore the edentulous anterior maxilla.





Questions

1. What will be the recommended type of radiograph to plan for the maxillary reconstruction?
   - **a.** Bite-wing
   - **b.** Periapical
   - **c.** Panoramic
   - **d.** Cone beam computed tomography (CBCT)
2. In the present case, the widening of the periodontal ligament highlighted by the red arrow is most probably caused by:
   - **a.** Occlusal wear.
   - **b.** Past traumatic injury.
   - **c.** Genetics.
   - **d.** Periodontal etiology.
3. In a patient who is periodontally stable and has no past conditions, how often should the radiographic information be updated?
   - **a.** Every 6 months
   - **b.** Every 2 years
   - **c.** Only when covered by insurance
4. When assessing the furcation involvement of a maxillary and/or mandibular tooth, the ideal radiograph is:
   - **a.** Bite-wing.
   - **b.** Periapical.
   - **c.** Panoramic.

This chapter was developed from Chapters 32 and 33 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Solutions

1. Answer: d

Explanation: The only radiograph that will provide a 3D assessment of the bone morphology is CBCT. This will facilitate the treatment planning process for the interdisiplinary team.

2. Answer: b

Explanation: Considering the medical history of the patient and the current clinical presentation, traumatic injury is the most probable cause. The patient does not have any occlusal wear and there are no signs of active periodontal conditions.

3. Answer: b

Explanation: Complete mouth series or full mouth radiographs should be part of every patient's periodontal evaluation, coupled with a detailed recording of probing depths, gingival margin location, and bleeding on probing. Radiographic evaluation should be updated every 2 years in the form of posterior bite-wings. In patients with active periodontal disease or a history of the disease, vertical bite-wings are the radiographs of choice.

4. Answer: a

Explanation: Bite-wings are more favorable to identifying the morphology of furcation defects than the other options.

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20 Periodontal Risk and Prognosis

Relevant Terminology

Terminology	Explanation
individual prognosis	Determined at the individual tooth level after determining the overall prognosis
overall prognosis	• Prognosis of the dentition as a whole • Consider patient age, current severity of disease, systemic factors, smoking, presence of biofilm, calculus, other local factors, patient compliance, prosthetic possibilities
prognosis	• A prediction of the probable course, duration, and outcome of disease based on a general knowledge of its pathogenesis and the presence or absence of known risk factors
risk assessment	An important component of patient management that allows clinicians to customize treatment for a specific patient
risk determinant	Risk factors that cannot be modified (e.g., genetic factors, age, gender, socioeconomic status, stress)
risk factor	• Environmental, behavioral, or biologic factor that increases the likelihood of developing disease • Identified through longitudinal studies • The exposure must occur before disease onset • Established periodontal risk factors include tobacco smoking, diabetes, pathogenic bacteria, and microbial tooth deposits
risk indicators	Probable or putative risk factors that have been identified in cross-sectional studies but not confirmed through longitudinal studies (e.g., HIV/AIDS, osteoporosis, infrequent dental visits)
risk markers/ predictors	• Factors that are associated with increased risk of disease but do not cause the disease • Identified through cross-sectional or longitudinal studies • Previous history of periodontal disease and bleeding on probing are examples

Fast Facts

Bleeding on probing (BOP)	• Bleeding is the best clinical indicator of gingival inflammation[3] • Lack of BOP serves as an indicator of periodontal health, but the presence of BOP alone is not a good predictor of future attachment loss[4] • Lack of BOP is an indicator of periodontal health (high negative predictive value)
Plaque accumulation and periodontitis	• The quantity of plaque is not of major importance in the disease process • The composition or quality of the complex plaque biofilm is important • Keystone pathogens contribute to a shift from a symbiotic microbial community to a more dysbiotic community
Specific bacteria in the pathogenesis of periodontitis	• Specific bacteria matching the criteria for disease causation: *Aggregatibacter actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia* • Their elimination or suppression impacts the success of therapy • There is a host response to these pathogens • Virulence factors are associated with these pathogens • Inoculation of these bacteria into animal models induces periodontal disease

Fast Facts—cont'd

Anatomic factors	These factors increase the likelihood of periodontal disease by harboring bacterial plaque and making oral hygiene and professional instrumentation difficult (e.g., root concavities, developmental grooves, enamel projections, enamel pearls, bifurcation ridges)
Restorative factors	May result in increased plaque accumulation, increased inflammation, and increased bone loss (e.g., subgingival or overhanging margins of restorations)
Presence of calculus	• Reservoir of bacterial plaque • The presence of calculus in healthy individuals with routine dental care does not result in attachment loss, while lack of maintenance or a systemic condition with the presence of calculus may negatively impact attachment loss
Genetic factors as risk determinants	• Polymorphisms in IL-1α and IL-1β are associated with severe chronic periodontitis in nonsmoking subjects • Immunogenic alterations that are genetically controlled: neutrophil abnormalities, monocytic hyperresponsiveness to lipopolysaccharides, and alterations in the monocyte/macrophage receptors for the Fc portion of antibody
Age as risk determinant	• The prevalence and severity of periodontal disease increase with age • Attachment loss and bone loss in older individuals are the result of prolonged exposure to other risk factors over the life span, creating a cumulative effect • The younger the patient, the longer the time for exposure to causative factors • Aging alone does not increase disease susceptibility
Gender as risk determinant	• Men have more loss of attachment than women • Men have poorer oral hygiene than women • Gender difference in prevalence and severity is related to differences in preventive practices
Socioeconomic status as risk determinant	Decreased dental awareness and decreased frequency of dental visits may contribute to gingivitis and poor oral hygiene
Stress as risk determinant	• The incidence of necrotizing gingivitis increases during periods of emotional and physical stress • Emotional stress may alter normal immune function, which can affect the periodontium. Stressful events may increase psychosocial factors and risk behaviors (e.g., smoking, poor oral hygiene) • Individuals with financial strain, distress, depression, or inadequate coping mechanisms have more severe loss of attachment
HIV/AIDS as risk indicator	• HIV infection or AIDS may increase susceptibility (although the association is inconclusive) • AIDS-affected individuals with good preventive oral health measures can maintain periodontal health
Osteoporosis as risk indicator	The reduced bone mass may aggravate periodontal disease progression, though the association is inconclusive
Infrequent dental visits as risk indicator	• Failure to visit the dentist regularly may increase the risk for severe periodontitis • Individual's age, susceptibility to disease, and other factors, together with infrequent dental visits, may be involved in disease presentation
Previous history of periodontal disease as risk marker	Individuals with a history of previous periodontal disease are at higher risk of future loss of attachment
Factors in determination of prognosis	• Patient age • Disease severity • Biofilm control • Patient compliance and cooperation • Systemic and environmental factors (e.g., smoking, systemic disease, genetic factors, stress) • Local factors (e.g., biofilm and calculus, subgingival restorations, anatomic factors, tooth mobility, caries, tooth vitality, root resorption) • Prosthetic and restorative factors
Relevance of patient age in prognosis	For two patients with comparable levels of attachment and bone loss, the prognosis is worse for the younger patient because the destruction in a relatively short period may indicate an aggressive type of periodontitis
Anatomic factors in prognosis	Anatomic factors that present accessibility problems are: • Furcation—in 58% of maxillary and mandibular first molars, the furcation entrance diameter is narrower than the width of conventional periodontal curettes[5] • Developmental grooves • Root proximity • Root concavity

Continued

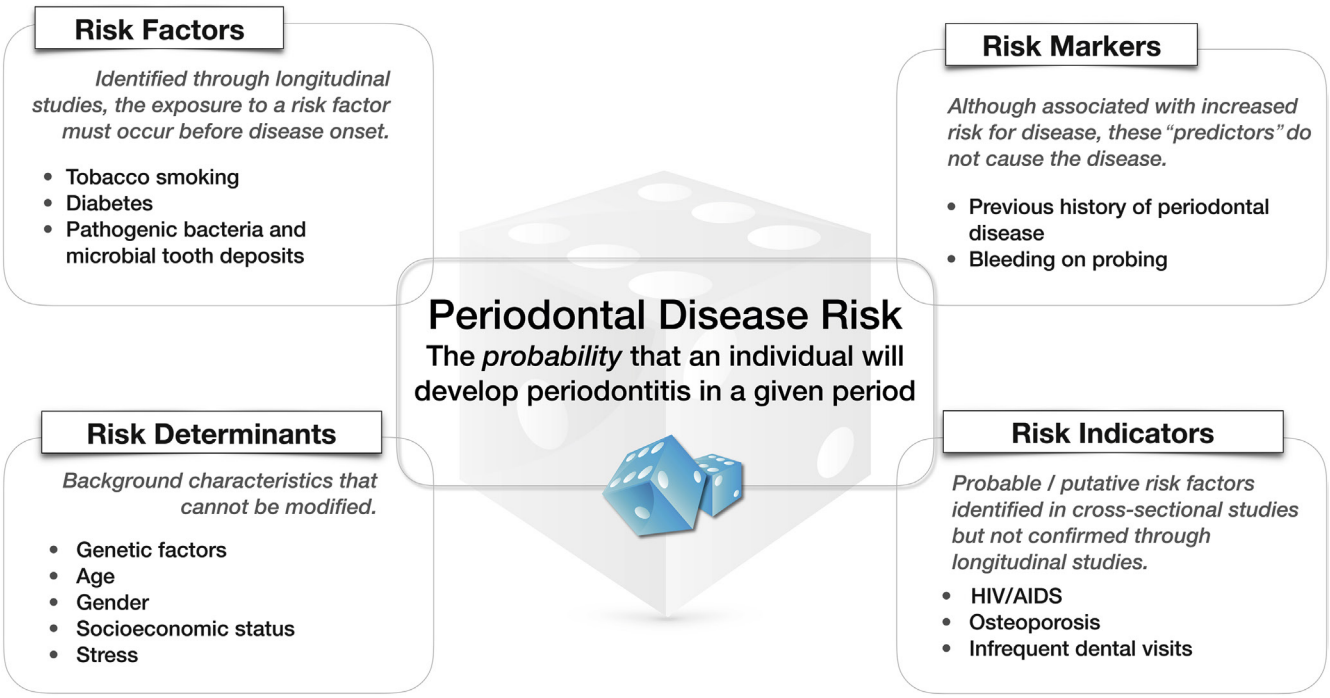
Fast Facts—cont'd

Tooth mobility in prognosis	Tooth mobility resulting from loss of alveolar bone is not likely to be corrected, while mobility from inflammation or trauma from occlusion can be corrected
Tooth vitality in prognosis	The periodontal prognosis of treated nonvital teeth does not differ from that of vital teeth
Type of bony defect in prognosis	• The prognosis for horizontal bone loss depends on the height of the existing bone, because it is challenging to gain bone height through regeneration • The prognosis for angular (vertical) intrabony defects is excellent if the contour of the existing bone and the number of osseous walls are favorable for regeneration
Effect of strategic extraction on prognosis	• May improve the prognosis of adjacent teeth, enhance the prosthetic treatment, and increase the success rate of implants replacing the extracted teeth • "Watch and wait" approach may allow deterioration of the site and limit the possibility of future implant treatment
Prognosis for biofilm-induced gingival diseases	• Gingivitis associated with only dental plaque has a good prognosis; elimination of local factors (bacterial biofilm and its retentive factors) will likely resolve disease • In biofilm-induced gingival diseases modified by systemic factors (hormonal changes, diabetes or blood dyscrasias), the prognosis depends not only on control of the bacterial biofilm but also on control or correction of the systemic factors • In biofilm-induced gingival diseases modified by medications, e.g. drug influenced gingival enlargements, the prognosis depends not only on whether the cause of the inflammation can be completely eliminated, but also on the patient's systemic problem being amenable to treatment with an alternative medication that does not cause gingival enlargement
Prognosis of non-biofilm-induced gingival lesions	• Disease is not attributed to biofilm accumulation • Prognosis depends on elimination of the etiologic factor(s) • Prognosis of gingival manifestation of dermatologic disorders (lichen planus, pemphigoid, pemphigus vulgaris, erythema multiforme, and lupus erythematosus) depends on the management of the associated dermatologic disorder
Kwok and Caton prognostication system[1]	• **Favorable**: comprehensive periodontal treatment and maintenance will stabilize the status of the tooth. Future loss of periodontal support is unlikely • **Questionable**: local or systemic factors influencing the periodontal status may or may not be controllable. If controlled, the periodontal status can be stabilized with comprehensive periodontal treatment. If not, future periodontal breakdown may occur • **Unfavorable**: local or systemic factors influencing the periodontal status cannot be controlled Comprehensive periodontal treatment and maintenance are unlikely to prevent future periodontal breakdown • **Hopeless**: the tooth must be extracted
McGuire prognostication system[2]	• **Good prognosis**: Control of etiologic factors and adequate periodontal support ensure the tooth will be easy to maintain by the patient and clinician • **Fair prognosis**: Approximately 25% attachment loss or grade I furcation invasion; location and depth allow proper maintenance with good patient compliance • **Poor prognosis**: 50% attachment loss, grade II furcation invasion; location and depth make maintenance possible but difficult • **Questionable prognosis**: >50% attachment loss, poor crown-to-root ratio, poor root form, grade II furcation invasion (location and depth make access difficult) or grade III furcation invasion; mobility 2 or 3; root proximity • **Hopeless prognosis**: Inadequate attachment to maintain health, comfort, and function

Core Knowledge

Introduction

Periodontitis is a destructive inflammatory disease that manifests with varying severity in people with differing susceptibilities. Periodontal risk assessment and prognosis are valuable adjuncts for the diagnosis and management of patients with periodontal disease; their inclusion among the diagnostic and treatment considerations helps to customize patient management and to increase the overall well-being of the patient.



• **Fig. 20.1** Periodontal Risk Assessment. The eventual goal of doing periodontal risk assessment is to develop a treatment plan that is personalized for a given patient, taking into account their periodontal risk profile. Based on the risk profile and diagnosis, the treatment plan may be modified accordingly. This figure highlights various categories of risk for periodontal disease. The reader is referred to eFig. 34.1 in Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.) to understand the steps in clinical risk assessment before initiation of therapy, and the need for reassessment after a negative response to therapy.

Periodontal Risk Assessment

The American Academy of Periodontology (AAP) Guidelines say that risk assessment is "increasingly important in periodontal treatment planning and should be part of every comprehensive dental and periodontal evaluation."[6]

- **Goal of periodontal risk assessment**—the goal is "long-term retention of teeth by combining prevention, early intervention, and directed therapy in the management of a patient with periodontitis"[7]
- **Disease risk**—the probability that an individual will develop a specific disease or experience a change in health status in a specified interval of time
- **Risk factor**—any characteristic, behavior, or exposure that has an association to a particular disease. Fig. 20.1 reviews various risk factors associated with periodontal disease.

CLINICAL CORRELATE

What is the need to incorporate risk assessment as a component of periodontal evaluation in the process of diagnosis and treatment planning?

The term 'diagnosis' refers to current disease status. Also, traditional clinical parameters of periodontal disease (probing depth, attachment loss, radiographic assessment of alveolar bone level, etc.) are cumulative measures of past disease; they do not accurately reflect current disease activity, or predict future disease activity. Contrary to diagnosis which denotes existing disease status, risk assessment predicts the *future* disease status as well as the likelihood and rate of disease progression. Incorporation of risk assessment in clinical evaluations has the potential to alter the *traditional model of care* (where a lesion/condition is diagnosed and repaired, regardless of the patient's risk for future disease). Instead, a *wellness model of care* can be followed, which emphasizes prevention and targeted reduction of risk factors *in addition to* reparative treatment. This addresses the goals of improving the overall well-being of the patient, decreasing morbidity, and reducing the overall costs of health care.[7]

Periodontal Prognosis

Prognosis is integral to therapy as treatment decisions are made based on prognosis, with the goal to improve prognosis.

- A prognosis is a prediction of the probable course, duration, and outcome of a disease based on a general knowledge of its pathogenesis and the presence of associated risk factors.
- The determination of prognosis is a dynamic process. The initial or *provisional prognosis*, derived from the initial clinical examination and preliminary diagnosis, must be revised at various points during treatment and maintenance phases.

Fig. 20.2 describes the different types of prognosis and lists the factors in determination of prognosis.

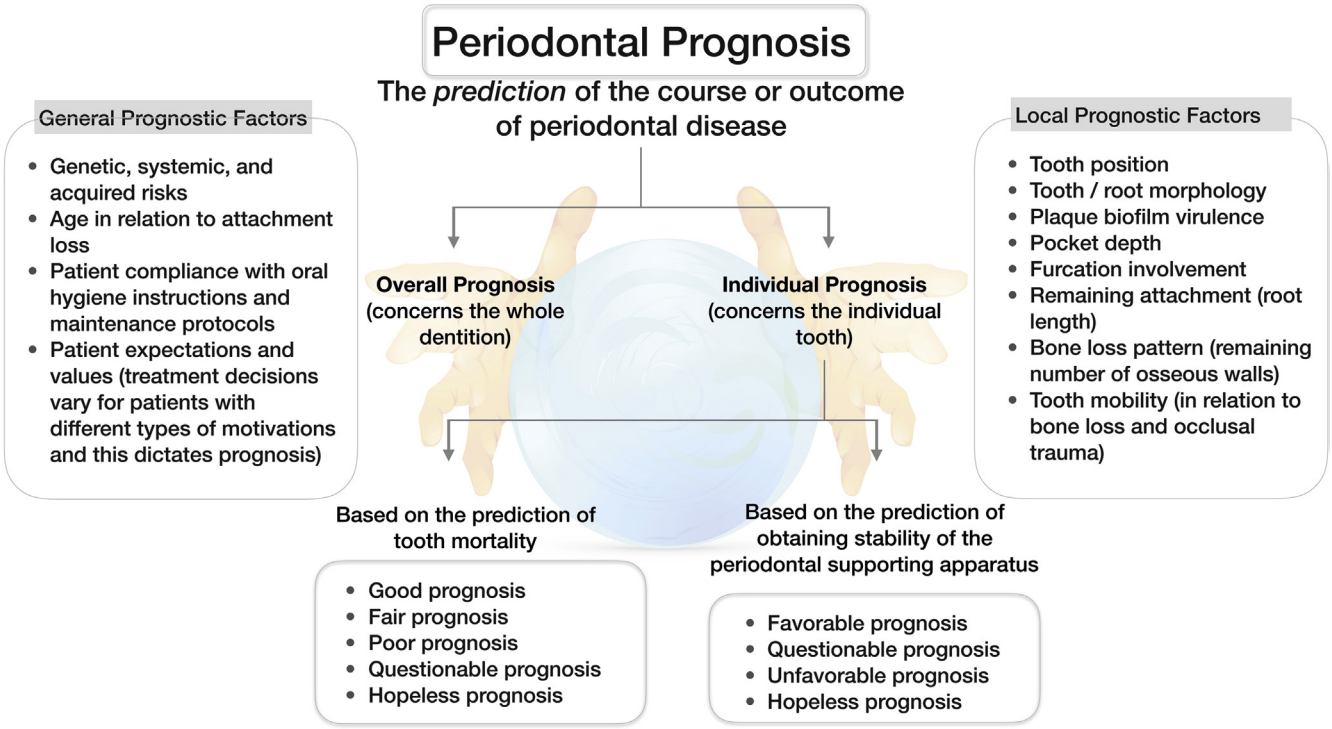
CLINICAL CORRELATE

What is the relationship between examination, diagnosis, prognosis, and treatment?

Examination → Diagnosis → Prognosis ↔ Treatment

- Diagnosis requires thorough and careful examination.
- Prognosis is based on accurate diagnosis.
- Treatment decisions are based on prognosis.
- Treatment decisions are made to improve prognosis.
- Diagnosis and prognosis will change with treatment.

Adapted from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.



• **Fig. 20.2** Periodontal Prognosis: Types and Prognostic Factors.

Prognosis can be divided into overall prognosis and individual tooth prognosis. The overall prognosis deals with the dentition as a whole and considers:

- Should treatment be undertaken?
- Is treatment likely to succeed?
- When prosthetic replacements are needed, are the remaining teeth able to support the added burden of the prosthesis?

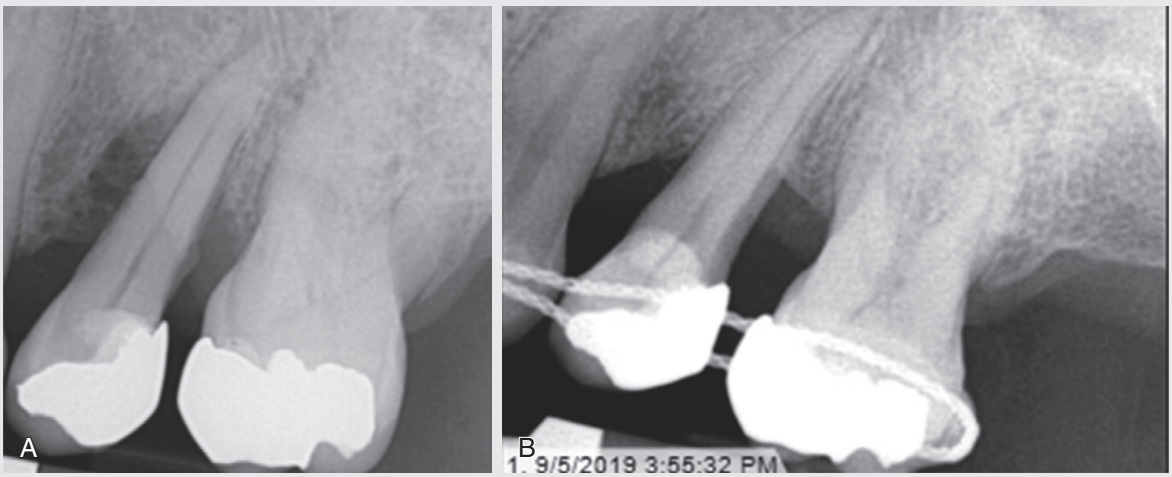
The individual tooth prognosis is determined after the overall prognosis and is affected by it. Historically, prognostic classification schemes have been developed based on studies evaluating tooth mortality as the end outcome. One such scheme has the following prognosis categories: good, fair, poor, questionable, or hopeless.[2] In contrast to schemes based on tooth mortality, Kwok and Caton[1] proposed a scheme based on "the probability of obtaining stability of the periodontal supporting apparatus." This scheme grades the prognosis as:

- Favorable—comprehensive periodontal treatment and maintenance will stabilize the status of the tooth
- Questionable—local or systemic factors influencing the periodontal status of the tooth *may or may not* be controllable
- Unfavorable—local or systemic factors influencing the periodontal status *cannot* be controlled
- Hopeless—the tooth must be extracted

Local and general factors listed in this figure are taken in to account before overall and individual tooth prognosis are made.

CASE-BASED LEARNING EXERCISE

Scenario: A 53-year-old male patient presented with the chief complaint: "My gums constantly bleed when I brush and I want a healthy mouth." The initial diagnosis was localized periodontitis Stage III Grade B (Fig. A). He was seen for periodontal comprehensive care. Nonsurgical therapy included oral hygiene instruction, splinting, scaling and root planing, and antimicrobial therapy. Surgical therapy included pocket reduction surgery using guided tissue regeneration and growth factors. Twelve months after the treatment, a periapical radiograph was exposed (Fig. B).



Questions

1. The wellness model emphasizes:
- **a.** Regenerative treatment.
- **b.** Reduction of risk factors.
- **c.** Diagnosis and reparative treatment.

2. Identify the correct sequence in clinical care:
- **a.** Examination-diagnosis-treatment-prognosis.
- **b.** Examination-treatment-diagnosis-prognosis.
- **c.** Examination-diagnosis-prognosis-treatment.
- **d.** Examination-prognosis-diagnosis-treatment.

3. Based on the Kwok and Caton scheme, identify the correct periodontal prognosis for #13 after treatment:
- **a.** Hopeless.
- **b.** Unfavorable.
- **c.** Questionable.
- **d.** Favorable.

4. In this case, the previous history of periodontal diseases is considered a risk:
- **a.** Factor.
- **b.** Marker.
- **c.** Indicator.
- **d.** Determinant.

5. If the same diagnosis of 'localized periodontitis, stage III, grade B', is made in a 23 year old patient, the prognosis is considered better than in a 53-year old.
- **a.** True
- **b.** False

This chapter was developed from Chapters 34 and 35 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Solutions

1. Answer: b

Explanation: The traditional model of care involves diagnosing and treating for a lesion or condition, regardless of the patient's risk for future disease. Instead, a *wellness model of care* can be followed which emphasizes prevention and targeted reduction of risk factors *in addition to* reparative treatment (Clinical Correlate Box 1).

2. Answer: c

Explanation: The correct sequence in clinical care is examination-diagnosis-prognosis-treatment. As highlighted in the second Clinical Correlate box, diagnosis requires thorough and careful examination. Prognosis is based on accurate diagnosis. Treatment decisions are based on prognosis and are made with the aim of improving it. Diagnosis and prognosis will change with treatment.

3. Answer: d

Explanation: Kwok and Caton proposed a scheme based on "the probability of obtaining stability of the periodontal supporting apparatus." Considering that the radiograph was exposed 12 months after the treatment was rendered,

radiographic positive changes are noted (periodontal ligament formation, stable lamina dura). These signs indicate stability of the tooth after treatment and prognosis is therefore favorable.

4. Answer: b

Explanation: Risk markers are associated with increased risk for diseases, but do not cause the disease itself. Apart from previous history of periodontal disease, bleeding on probing is another risk marker (see Fig. 20.1).

5. Answer: b

Explanation: A younger patient when compared with an older patient is considered as having a worse prognosis for the same level of periodontal destruction.

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21

Periodontal Treatment Planning and Rationale for Treatment

Relevant Terminology

Terminology / Abbreviation	Explanation
emergency phase	Sometimes emergency treatment is necessary to address the patient's symptoms (e.g., pain, acute infection) before starting periodontal treatments; this is one of the immediate goals.
nonsurgical periodontal therapy	Nonsurgical periodontal therapy includes scaling and root planing, reduction of risk factors (e.g., removal of overhanging restorations), and oral hygiene instruction.
periodontal evaluation	Periodontal evaluation includes periodontal examination, oral hygiene instruction, educating patients to reduce risk factors, and producing a periodontal treatment plan.
periodontal reevaluation	Periodontal reevaluation includes review of medical and dental history, periodontal examination, oral hygiene instruction, assessment of outcomes of nonsurgical therapy, and determining future treatments (e.g., periodontal surgery, adjunctive therapy, or maintenance).
periodontal treatment plan	A detailed plan to treat periodontal disease, tailored for the individual's need. The plan should be based on the diagnosis and prognosis and should have immediate, intermediate, and long-term goals.
supportive periodontal therapy	Supportive periodontal therapy (SPT) is also called *periodontal maintenance* or *periodontal recall*. SPT includes updating medical and dental history, extraoral and oral examinations, periodontal charting, review of radiographs, oral hygiene instruction, and nonsurgical periodontal treatment at regular intervals based on the patient's needs.
surgical periodontal therapy	Surgical therapy usually should be done after the patient has been through nonsurgical periodontal therapy and demonstrates acceptable oral hygiene. Surgical therapy includes resective surgery, regenerative surgery, mucogingival surgery, and other treatments depending on the patient's needs.

Fast Facts

Immediate goals of periodontal treatment plan	The immediate goal is to control the disease by treating acute or severe periodontal infection that is causing symptoms or affecting the patient's general health.
Intermediate goals of periodontal treatment plan	The intermediate goals are to reconstruct a healthy and functional dentition by treating periodontal disease and restoring decays and missing teeth, to improve aesthetics, and to provide other dental treatments.
Long-term goal of periodontal treatment plan	The long-term goal is to maintain periodontal and oral health in the compliant patient. After receiving treatments, patients must be educated about the importance of maintaining good oral hygiene to prevent disease recurrence.
Considering systemic conditions	It is always important to review the patient's medical history to evaluate the impact of systemic diseases on tissue healing and treatment response. If there is any concern, it is always necessary to consult the patient's physician prior to initiating therapy.

Continued

Fast Facts—cont'd

Extracting or preserving a tooth	Sometimes it is not easy to make a decision regarding retention, temporary retention, or removal of a problematic tooth. A tooth probably should be extracted if: • It is very mobile and causes pain • It has acute infection that cannot be controlled • It will not be functional
Temporary retention of a tooth	Sometimes temporarily retaining a hopeless tooth may be better for the patient and future treatment. Some of the scenarios include: • The tooth serves as a posterior stop • An anterior tooth is retained for aesthetic reasons until the prosthesis is ready • Hopeless teeth can be extracted in conjunction with periodontal surgery of adjacent teeth to reduce the number of clinical visits; or • Forced orthodontic extrusion of a tooth destined to be extracted in order to develop the future implant site
Periodontal treatment sequence	The sequence of periodontal treatment generally should be periodontal evaluation, nonsurgical therapy, periodontal reevaluation, surgical therapy, and supportive (maintenance) therapy, but this sequence could change based on patient needs.
Timing to start periodontal supportive therapy	After the completion of nonsurgical therapy, the patient should be placed in the supportive phase. In this phase, the patient can have surgical therapy or restorative treatment depending on their need.
Explaining the treatment plan	To help the patient understand the treatment plan, the clinician should give specific statements, begin the discussion on a positive note, and present the entire treatment plan as a unit.
Discussing tooth extraction	Although a dentist usually tries to save a tooth, it is important to let the patient know that extracting hopeless teeth is sometimes necessary. Maintaining hopeless teeth with severe periodontal destruction may cause severe infection, affect systemic health, compromise adjacent teeth, and delay definitive restorative treatments.
Patient compliance	For successful treatment outcomes, it is important to have a compliant patient with the motivation to save existing teeth, reduce risk factors, and have good oral hygiene.
Examination, diagnosis, and prognosis	Diagnosis is made based on a comprehensive and accurate periodontal examination (clinical and radiographic). Prognosis is assigned based on accurate diagnosis.
Treatment, diagnosis, and prognosis	The treatment plan is devised based on diagnosis and prognosis, and successful treatment can improve prognosis. Both diagnosis and prognosis are dynamic and can change depending on disease control and progression. The treatment plan has to be modified accordingly.

Core Knowledge

Introduction

After diagnosis and prognosis are established in a patient with periodontal disease, the next step is to formulate a treatment plan. Unforeseen developments during treatment may require modification of the initial treatment plan; however, unless emergency treatment is needed, no therapy should be initiated until a treatment plan has been established. Keeping both *short-term goals* (elimination of infection and reduction of inflammation) and *long-term goals* (reconstruction and maintenance of a healthy dentition that fulfills all functional and esthetic requirements) in mind, a blueprint for case management is formed; this treatment plan is usually carried out in phases. The preferred sequence of treatment phases and the rationale behind each one are reviewed in Fig. 21.1.

Therapeutic Goals and Wound Healing Possibilities

The primary goal of periodontal therapy is to eliminate infection and inflammation within the periodontal structures. The next goal is to restore structure, function, and esthetics of affected periodontal tissue. The therapeutic goals and various possibilities for wound healing following periodontal therapy are reviewed in Fig. 21.2.

CLINICAL CORRELATE

What should be kept in mind when deciding to extract a tooth of questionable prognosis?

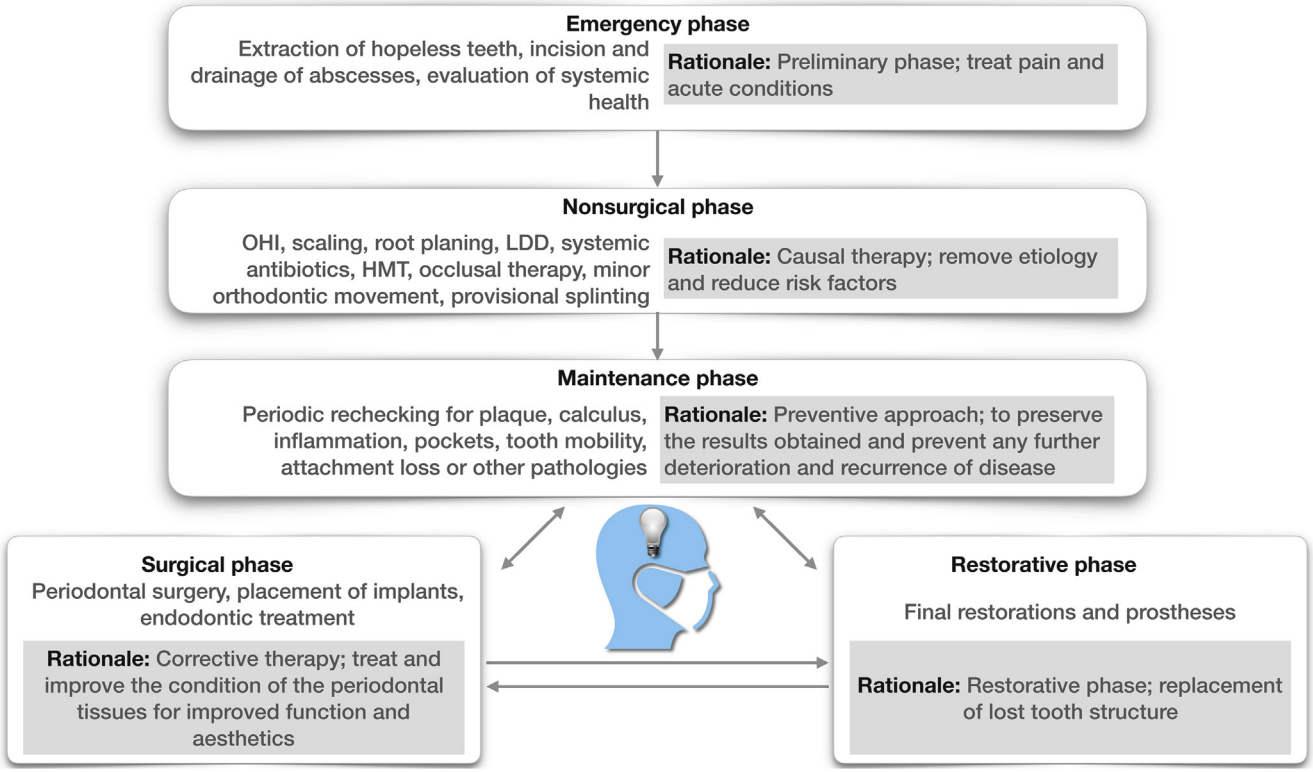
In cases of questionable prognosis, treatment is focused on restoring and maintaining the long-term health of the periodontium throughout the mouth, rather than attempting spectacular efforts to "tighten loose teeth." Keeping this in mind, a tooth with questionable prognosis may be extracted under certain conditions:

- The tooth exhibits mobility-induced pain or discomfort during function.
- It can cause acute abscesses during therapy.
- There is no use for it in the overall treatment plan (e.g., it is not a strategic abutment or is not in function).

Therapeutic Outcomes

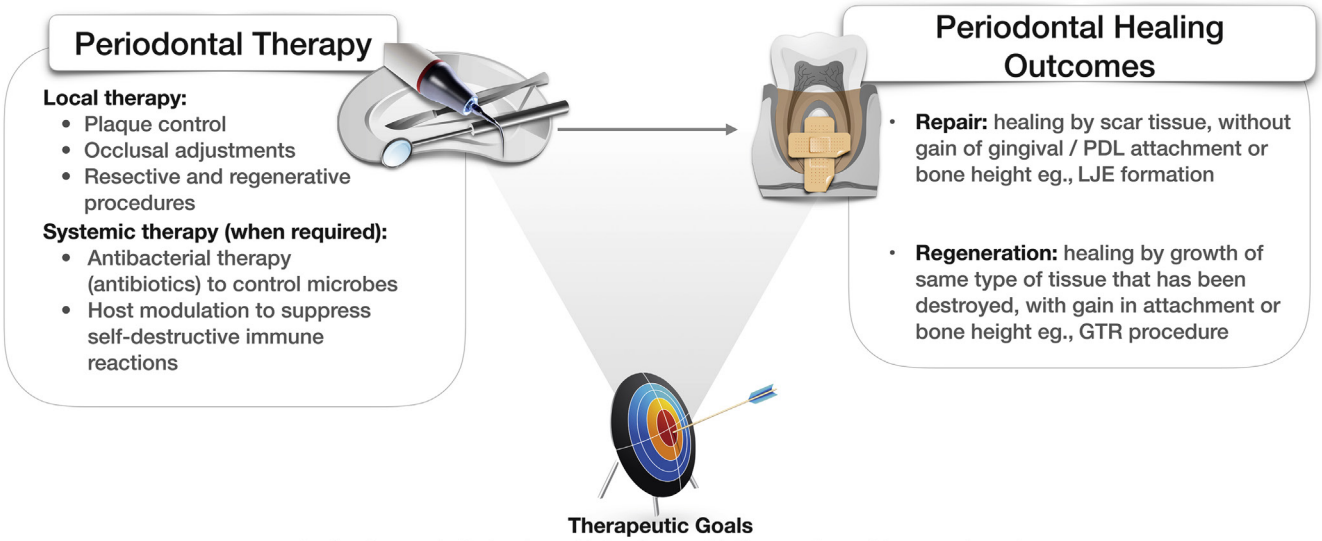
When properly performed, periodontal treatment can achieve several results:

- elimination of pain



• **Fig. 21.1** Periodontal Treatment Plan: Rationale and Treatment Sequence. *HMT*, host modulatory therapy; *LDD*, local drug delivery; *OHI*, oral hygiene instructions. (Adapted from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

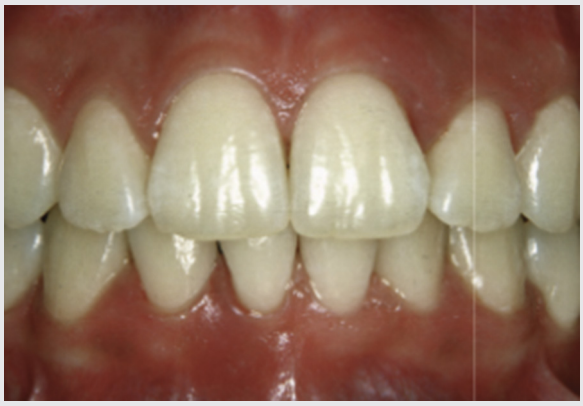
- elimination of exudate
- elimination of infection
- control of gingival inflammation and bleeding
- arrest of destruction of soft tissue and bone
- reduction of periodontal pockets and gain in clinical attachment
- reduction in abnormal tooth mobility
- restoration of optimal occlusal function
- restoration of tissue destroyed by disease
- reestablishment of physiologic gingival contour



• **Fig. 21.2** Periodontal Therapy: Goals and Wound Healing Possibilities. The figure reviews the main therapeutic goals behind periodontal therapy. Healing after periodontal therapy is similar to healing in other areas of the body and follows stages of inflammation, proliferation of precursor cells, matrix synthesis, wound organization, wound contraction, and finally tissue maturation by remodeling. However, because many different tissues (epithelium, gingival connective tissue, PDL connective tissue, bone, and cementum) are involved in trying to create a new connective tissue attachment to an avascular root surface cementum, the process of periodontal healing is considered complex and may have different possible outcomes: **Repair:** This process basically restores the continuity of the diseased gingiva and its attachment to the tooth surface and reestablishes a normal gingival sulcus at the base of the *preexisting* periodontal pocket. This type of *healing by scar tissue* arrests bone destruction but does not result in gain of gingival attachment or bone height. Restoration of gingival epithelial continuity by formation of LJE is an example of a periodontal repair process; here, epithelial attachment takes over areas previously occupied by connective tissue attachment in an attempt to restore continuity with the root surface. This is the type of healing commonly observed following scaling and root planing. **Regeneration:** This process involves the renewal of a structure by growth of the same type of tissue that was destroyed, or its precursor. Reconstruction of lost periodontium by GTR is an example of periodontal regeneration. *GTR*, guided tissue regeneration; *LJE*, long junctional epithelium; *PDL*, periodontal ligament.

CASE-BASED LEARNING EXERCISE

Scenario: A 23-year-old African American female presented to the dental clinic for periodontal examination. Clinical examination revealed deeper probing depths (6 mm or greater) in all her first molars and in her mandibular central incisors. Other teeth were periodontally not affected. Radiographs revealed vertical bony defects on the mesial aspect of her first molars, where the deep pockets were located. Her oral hygiene was good and she was otherwise healthy with no known drug allergies. The patient reported that her elder brother also had similar gum problems.



Adapted from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

Questions

1. Based on the 1999 Periodontal Disease Classification, which of the following diagnoses will fit the case presentation?
   a. Chronic periodontitis
   b. Localized aggressive periodontitis
   c. Generalized aggressive periodontitis
   d. Refractory periodontitis
2. Aggressive periodontitis is not a separate disease category in the 2017 Periodontal Disease classification.
   a. True.
   b. False.
3. Based on the clinical presentation, will this patient benefit from systemic antibiotic administration during nonsurgical periodontal therapy (scaling and root planing)?
   a. Yes
   b. No
4. Put the following periodontal therapy phases in order: 1. Periodontal maintenance; 2. Scaling and root planing; 3. Periodontal reevaluation; 4. Deriving a diagnosis.
   a. 3, 2, 1, 4
   b. 4, 2, 3, 1
   c. 2, 3, 4, 1
   d. 4, 3, 2, 1

This chapter was developed from Chapter 36 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: **b**

Explanation: Because only first molars and incisors were involved, it would have been diagnosed as localized aggressive periodontitis under the 1999 classification.

2. Answer: **a**

Explanation: Based on the accumulated evidence, in spite of distinct clinical presentations, it was unclear whether aggressive periodontitis was completely distinct from chronic periodontitis in terms of its etiology and pathogenesis. Therefore a decision was made not to list it as a separate disease entity in the 2017 classification.

3. Answer: **a**

Explanation: Studies have confirmed a statistically significant clinical benefit (in terms of pocket depth reduction and attachment gain) when systemic antibiotics are used in combination with scaling and root planing.

4. Answer: **b**

Explanation: The treatment plan should be based on the diagnosis. Treatment typically starts with nonsurgical periodontal therapy. When periodontal health is established and has been assessed during periodontal reevaluation, the patient is put on an individualized periodontal maintenance program.

22

Periodontal Treatment in Medically Compromised Patients

Relevant Terminology

Terminology/Abbreviation	Explanation
adrenal insufficiency	Patients with adrenal insufficiency do not produce enough hormones, including cortisol and aldosterone. Addison disease is a primary cause of adrenal insufficiency. Intake of exogenous glucocorticosteroids may also cause adrenal insufficiency.
antibiotic prophylaxis	Administration of antibiotics 0.5–1 hour before dental procedures to prevent infection (bacteremia); this is only required for some patients with high risk of infection
bisphosphonates	Commonly used to treat osteoporosis or control bone remodeling in cancer patients. The mechanism is inhibiting the activity of osteoclasts, resulting in less bone resorption and remodeling. The use of bisphosphonates may cause alveolar bone necrosis in some patients who experience surgical trauma to oral tissues.
diagnostic criteria for diabetes mellitus	Diabetes can be diagnosed by any of the following laboratory methods (American Diabetes Association, 2017): 1. Fasting plasma glucose level ≥126 mg/dL (≥7.0 mmol/L) 2. Two-hour postprandial glucose level ≥200 mg/dL (≥11.1 mmol/L) during an oral glucose tolerance test 3. Glycated hemoglobin (HbA1c) value ≥6.5% (≥48 mmol/L) 4. Random plasma glucose level ≥200 mg/dL (≥7.0 mmol/L) for a patient with classic symptoms of hyperglycemia (polydipsia, polyuria, polyphagia)
medication-related osteonecrosis of the jaw (MRONJ)	MRONJ is characterized by alveolar bone necrosis and unhealed exposed bone over 8 weeks. It occurs in patients using bisphosphonates, especially high-potency, nitrogen-containing bisphosphonates given intravenously. MRONJ is also associated with other risk factors, including poor oral hygiene, smoking, extraction, chemotherapy, and radiotherapy.
partial thromboplastin time (PTT) test	Blood test measuring coagulating function. It measures intrinsic and common coagulation pathways: coagulation factors III, IX, and XI, and low levels of coagulation factors I, II, V, X, and XII. Normal range is 25–40 seconds.
prothrombin time (PT) test	Blood test measuring coagulating function. It measures extrinsic and common coagulation pathways: coagulation factors I, II, V, VII, and X. The results are reported as INR (international normalized ratio). Normal INR is about 1.
renal failure	Medical condition in which the kidney loses its normal function. Renal failure can cause severe electrolyte imbalance, cardiac arrhythmia, pulmonary congestion, congestive heart failure, and prolonged bleeding.

Fast Facts

Management of patients with stroke history	• Unnecessary dental therapy should not be performed for the first 6 months following stroke • Because patients with stroke history usually take anticoagulants, it is necessary to consult the physician before any surgical treatment • Blood pressure should be carefully monitored

Fast Facts—cont'd

Management of patients with diabetes	• Generally, dental surgery should not be performed for patients with uncontrolled diabetes (especially those with HbA1c ≥10%) • In these patients, the blood glucose should be checked before any long procedure and checked immediately if the patient has hypoglycemia symptoms (e.g., shakiness/tremor, confusion, agitation/anxiety, sweating) • It is important to ask diabetic patients taking medications (e.g., insulin) to eat before dental surgery to avoid hypoglycemia
Management of patients with adrenal insufficiency	Most patients with adrenal insufficiency can receive dental surgery without supplemental glucocorticosteroids if they take their usual dose of corticosteroids within 2 hours of the planned procedure. However, medical consultation is still recommended before performing any surgical procedure.
Management of patients receiving hemodialysis	A patient with renal failure may receive hemodialysis. For these patients, the dentist should check for a history of hepatitis, consider prophylactic antibiotics before procedures to prevent endarteritis of the arteriovenous shunt, and plan periodontal/surgical treatment on the day after dialysis (waiting for the effects of heparin to subside).
Management of patients with liver disease	Patients with liver diseases (e.g., cirrhosis, hepatitis B/C) may have coagulation disorders because most coagulation factors are produced by the liver. It is important to consult their physician and check the results of lab tests of coagulating function (e.g., prothrombin time).
Management of patients with cancer	Some medications used by cancer patients may lead to complications affecting patient management: • Bisphosphonates—may cause MRONJ after extraction or other surgical trauma • Anticoagulants—may increase the risk of bleeding complications • Steroids—can induce secondary adrenal insufficiency • Chemotherapy—can cause thrombocytopenia, anemia, and leukopenia • Radiotherapy—can cause mucositis, xerostomia, dysphagia, radiation caries, and trismus

Core Knowledge

Introduction

Patients with periodontal treatment needs can present with a wide range of medical conditions; some of these comorbidities will have a direct impact on how these patients are managed and treated for periodontal disease. Some of the systemic medical conditions will have oral manifestations, but the presence of periodontitis is also associated with some distant chronic systemic conditions. In this chapter, the focus is primarily on management considerations for patients with common medical conditions. Readers are referred to Chapter 39 in *Newman and Carranza's Clinical Periodontology* (13th ed.) for in-depth information on clinical management and periodontal treatment considerations in patients who are medically compromised.

Cardiovascular Diseases

The management considerations for patients with common cardiovascular conditions are listed in Table 22.1. For other cardiac conditions, please refer to Chapter 39 in *Newman and Carranza's Clinical Periodontology* (13th ed.).

CLINICAL CORRELATE

What are the dental care implications when treating patients with cardiac pacemakers?

In spite of conflicting evidence, the American Dental Association (ADA) recommends that consideration be given to potential interference from ultrasonic devices on cardiac pacemaker function. Newer devices are better shielded and some manufacturers recommend a safe distance of approximately 15" between the ultrasonic instrument and the pacemaker to avoid interference. When in doubt, it is prudent to contact the patient's cardiologist. For more information on this topic, please refer to the ADA webpage on Cardiac Implanted Devices and Electronic Dental Instruments: https://www.ada.org/en/member-center/oral-health-topics/cardiac-implanted-devices-and-electronic-dental-instruments.

Diabetes

Management considerations in patients with diabetes are listed in Table 22.2.

BASIC SCIENCE CORRELATE

What is HbA1c and what does it measure?

Glycosylation is a process that involves enzyme-mediated protein modification. Glucose binds to hemoglobin and forms glycosylated hemoglobin, or hemoglobin A1C (HbA1c). Because the life cycle of red blood cells is 120 days, HbA1c is a good objective measure of average glycemic (diabetic) control over the past 3 months. Periodic HbA1c and regular home measurements are used as yardsticks for treatment recommendations and modifications in patients with diabetes.

Conditions Requiring Antibiotic Prophylaxis

Conditions that require antibiotic prophylaxis are listed in Table 22.3.

TABLE 22.1 Treatment Considerations in Patients With Common Cardiovascular Conditions

Condition and Reference Values (if Applicable)	Patient Management Considerations	Periodontal Relevance
Hypertension Categories:[1] Normal: < 120/80 mm Hg Elevated: 120–129 (systolic) and < 80 (diastolic) Stage 1: 130–139 (systolic) or 80–89 (diastolic) Stage 2: ≥ 140 (systolic) or ≥ 90 (diastolic) Hypertensive Crisis: > 180/120	• Use of local anesthetics with epinephrine should be minimized (up to two carpules of 1:100,000 epinephrine) or completely avoided. At the same time, pain management is critical to reduce stress and anxiety • Hypertension can increase the chances of bleeding complications intraoperatively and postoperatively. These patients are also at a higher risk for experiencing other cardiac related events • Stress reduction is essential in hypertensive patients. Sedation options should be considered in these patients to minimize anxiety • Except for emergency treatment, elective procedures should be postponed in patients presenting with very high blood pressure (> 180/100) and medical consult is warranted prior to treating these patients • Certain antibiotics and nonsteroidal antiinflammatory agents are known to interfere with the efficacy of antihypertensive medications via drug interactions • It is prudent to avoid rapid changes in chair positioning, which may cause postural hypotension • Monitor vitals	Calcium channel blockers are implicated in drug-induced gingival overgrowth. Some of these medications can cause dry mouth, which may affect patient's oral hygiene
Ischemic Heart Diseases (Angina and Myocardial Infarction)	• Consultation with patient's physician (as needed) • Use of local anesthetics with epinephrine should be minimized (up to two carpules of 1:100,000 epinephrine) or completely avoided. At the same time, pain management is critical to reduce stress and anxiety • If patient is on anticoagulant or antiplatelet medication, intraoperative and postoperative bleeding should be anticipated and prepared for • Stress reduction is essential in hypertensive patients. Sedation options should be considered in these patients to minimize anxiety • Unexpired medications such as nitroglycerine and oxygen should be readily available • Prudent to avoid rapid changes in chair positioning; position should be comfortable for the patient • Clinician should be aware of potential adverse effects and drug interactions of the medications these patients consume • Monitor vitals	Some of the medications these patients are on can cause dry mouth, which may affect patient's oral hygiene
Congestive Heart Failure	• Consultation with patient's physician is required to establish the level of control (as needed) required to treat these patients without the risk of cardiovascular complications • If patient has prosthetic heart valve, they will require antibiotic premedication • Use of local anesthetics with epinephrine should be minimized (up to two carpules of 1:100,000 epinephrine) or completely avoided. At the same time, pain management is critical to reduce stress and anxiety • If patient is on anticoagulant or antiplatelet medication, intraoperative and postoperative bleeding should be anticipated and prepared for • Clinician should be aware of the potential adverse effects and drug interactions of the medications these patients consume. • Monitor vitals	Some of the medications these patients are on can cause dry mouth, which may affect patient's oral hygiene

TABLE 22.1 Treatment Considerations in Patients With Common Cardiovascular Conditions—cont'd

Condition and Reference Values (if Applicable)	Patient Management Considerations	Periodontal Relevance
Bleeding Disorders Common Tests: • Platelet count: *Normal:* 150,000–300,000/mm^3. *Thrombocytopenia:* <100,000/mm^3. • Bleeding time: Normal: 1–6 min Abnormal: > 6 min • Prothrombin time—PT (assess extrinsic pathway): *Normal:* 11–14 sec PT reported as International Normalized Ratio (INR) *Normal INR:* 1 *Abnormal INR:* >1.5 • Partial Thromboplastin Time—PTT (assess intrinsic pathway) *Normal: 25–40 sec* *Abnormal: >1.5× normal*	• Can be coagulation disorders or from use of medications (anticoagulant and antiplatelet medications) • Coagulation disorders include hemophilia A, hemophilia B, and von Willebrand disease • Anticoagulant medications are vitamin K antagonists that work by inhibiting the production of vitamin K–dependent coagulation factors (II, VII, IX and X) (e.g., warfarin), while antiplatelet medications inhibit platelet aggregation (e.g., aspirin) • Physician consultation is required in these patients to assess bleeding tendencies and manage the care accordingly • Intraoperative and postoperative bleeding are the primary concerns • Many clinicians no longer recommend patients to stop medications prior to surgical procedures because the risk for medical complications is high • Intraoperative use of hemostatic agents such as oxidized cellulose and microfibrillar collagen, and postoperative use of tranexamic acid, should be considered	Nonsurgical periodontal therapy can be done under local anesthesia when the INR is below 3. Simple surgical procedures can be done when the INR is 2–2.5, while complex surgical procedures will require an INR of less than 1.5–2.0.

These are general guidelines and patient management will depend on the individual needs of the patient and on the specific clinical scenario. For in-depth information and for treatment considerations in patients with other cardiac conditions, please refer to Chapter 39 in Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

TABLE 22.2 Management Considerations in Patients With Diabetes

Condition and Reference Values (if Applicable)	Patient Management Considerations	Periodontal Relevance
Diabetes (Type 1):[2] Absolute insulin deficiency caused by autoimmune destruction of β cells in the pancreas. • Hemoglobin A1c (HbA1c): *Normal (healthy): <5.7%* *Prediabetic: 5.7%–6.4%* *Controlled diabetic:≤7%* *Uncontrolled diabetic:>8%* • Fasting blood glucose: *Normal (Healthy): <100 mg/dL* *Prediabetic: 100–125 mg/dL* *Diabetes: ≥126 mg/dL (on two separate tests)*	• Certain pain medications such as aspirin and NSAIDs can interfere with diabetic medications, leading to hypoglycemia • Physician consult is required if diabetes is poorly controlled • Poorly controlled diabetes confers a higher risk for infection postoperatively; patients may benefit from antibiotic therapy • Patients with well-controlled diabetes are at a higher risk of hypoglycemia during dental procedures. Check the blood glucose level prior to dental procedure; if it is low, patient should consume some carbohydrates to minimize the risk of a hypoglycemic episode • Monitor vitals (diabetes is associated with hypertension)	Diabetes (types 1 and 2) has a direct effect on the periodontium and can aggravate the periodontal disease process (established risk factor). It also negatively affects treatment and healing outcomes. Patients with well-controlled diabetes respond well to therapy (i.e., as well as healthy patients without diabetes) Patients with uncontrolled diabetes are at a high risk for developing single or multiple periodontal abscesses. Other common oral lesions include candidiasis and lichen planus
Diabetes (Type 2): Relative insulin deficiency mainly from insulin resistance. Reference values reported in type 1 diabetes also apply to type 2 diabetes	• Most common type of diabetes • All the management considerations for Type 1 also apply to this type	Periodontal relevance is the same as for type 1 disease
Gestational Diabetes: Abnormal glucose tolerance during pregnancy	• Associated with increased risk for infant mortality and pregnancy complications • Women with GD are at higher risk for developing type 2 diabetes later in life • Refer to Chapter 24 for more information on management of periodontal disease in pregnant women	Early evidence suggests that periodontitis is associated with an increased risk for gestational diabetes.

These are general guidelines and patient management will depend on the individual needs of the patient and on the specific clinical scenario. For in-depth information and for other treatment considerations in diabetic patients, please refer to Chapter 39 in Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

TABLE 22.3 Prophylactic Antibiotic Indications

Condition	Indications for Antibiotic Prophylaxis	Preferred Antibiotics in Adults (Oral Route)	Procedures That Require Premedication

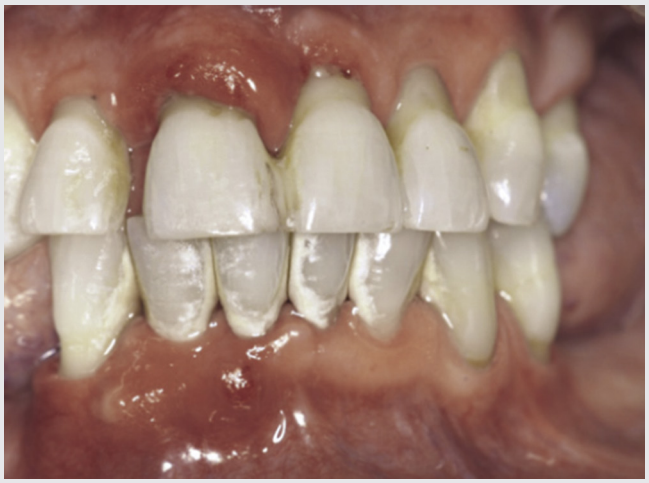
Prevention of Infective Endocarditis	• Prosthetic cardiac valves and prosthetic material used for cardiac valve repair
	• History of infective endocarditis
	• Cardiac transplant with valve regurgitation
	• Congenital heart diseases such as:
	  • Unrepaired cyanotic congenital heart disease
	  • Repaired congenital heart defect with residual shunts or valvular regurgitation
		In penicillin nonallergic patients: amoxicillin (2 g) 1 hour prior to procedure
		Allergic to penicillin: clindamycin 600 mg 1 hour prior to procedure
			Any dental procedure that involves manipulation of gingival tissue or the periapical region of teeth, or perforation of the oral mucosa, will require premedication (e.g., tooth extraction, scaling and root planing, periodontal flap surgery)

Orthopedic (Joint Replacement)	• Based on the current guidelines, prophylactic antibiotics are not generally recommended for patients with prosthetic joint replacements prior to dental procedures to prevent prosthetic joint infection • If needed, it is most appropriate for the orthopedic surgeon to recommend and prescribe antibiotics • Certain factors should be considered when deciding on the need for antibiotic prophylaxis: immune status and risk for infection, diabetic status, and history of previous joint infection.		

American Dental Association webpage on Antibiotic Prophylaxis Prior to Dental Procedures for in-depth information in this topic: https://www.ada.org/en/member-center/oral-health-topics/antibiotic-prophylaxis.

CASE-BASED LEARNING EXERCISE

Scenario: A 45-year-old Caucasian male presented with the chief complaint "I have swollen gums and they hurt." The patient, a type II diabetic, reported his last HbA1c to be 8.3%. Generalized probing depth was 2–4 mm with localized areas of 6–8 mm probing depths in the maxillary right central incisor and mandibular anterior teeth with suppuration. Gingiva looked erythematous and edematous in these areas. Palpation of the swollen areas led to pus discharge from the periodontal pockets. All the existing teeth tested vital. Oral hygiene was poor and significant supragingival and subgingival calculus noted. No signs of systemic involvement (fever, chills, malaise) were present.



Questions

1. Based on the history and clinical presentation, which of the following conditions is likely manifesting in this patient?
   a. Pericoronal abscess
   b. Periodontal abscess
   c. Endodontic abscess
2. HbA1c level reported for this patient was 8.3%, this is considered:
   a. Normal.
   b. Uncontrolled.
   c. Pre-diabetic.
   d. Controlled.
3. All of the following are associated conditions with uncontrolled diabetes, EXCEPT one. Which is the EXCEPTION?
   a. Single or multiple periodontal abscesses
   b. Candidasis
   c. Lichen planus
   d. Pyogenic granuloma
4. Does this patient require systemic antibiotics along with oral intervention?
   a. Yes
   b. No

Clinical photo from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

This chapter was developed from Chapter 39 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: b

Explanation: Attachment loss, deeper probing depths, and suppuration via periodontal pocket around endodontically sound teeth all point to periodontal abscesses.

2. Answer: b

Explanation: Because the patient's HbA1c level is greater than 8, it is considered to be uncontrolled.

3. Answer: d

Explanation: All of the listed choices can be associated with uncontrolled diabetes (type 2), except pyogenic granuloma. Pyogenic granuloma is commonly associated with pregnancy.

4. Answer: b

Explanation: Because this patient has no signs of systemic involvement of infection, systemic antibiotics need not be prescribed. Local debridement and drainage of the abscesses would suffice.

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23 Periodontal Treatment of Older Adults

Relevant Terminology

Terminology/Abbreviation	Explanation
activities of daily oral hygiene (ADOH) index	• ADOH is a dental assessment instrument used to quantify the functional ability of older adults to perform oral self-care tasks • Strategies can be developed to help older adults perform oral care based on the results[1]
aging of cells	• As individuals age, the rate of cell renewal is slower and the number of cells is reduced • The exhaustion of stem cells affects regenerative processes
chronic atrophic candidiasis	• Caused by the overgrowth of *Candida albicans* • Characterized by erythematous lesion under the denture • In older adults, lack of oral care or xerostomia can induce this condition
functional categories for older adults	• Functionally independent—lives in the community and receives little or no assistance • Frail—lives in the community and needs some degree of assistance • Functionally dependent—cannot maintain any level of independence and is totally dependent on assistance
geriatric dentistry	Geriatric dentistry is focused on providing diagnosis, prevention, and treatment of oral conditions in older adults. Dentists should work with physicians, nurses, and family members to provide the best care to older adults.
geriatrician	Physician specialized in providing care to older adults (usually those ≥65 years of age). In addition to primary care, provides specialized care that focuses on the prevention and management of medical conditions common in elderly individuals.
iatrogenic effect	Unexpected problem in a patient as a result of treatment rendered. In older adults, treatment plan should be more conservative because these patients usually have medical conditions and diseases.
root caries	• Decay on the root surface • Not common in general populations • In older patients, the prevalence of root caries is higher due to gingival recession, ineffective oral care, or xerostomia induced by medications
saliva substitutes	• Saliva substitutes contain salt ions, a flavoring agent, paraben (a preservative), cellulose-derived or animal mucins, and fluoride to simulate chemical and physical properties of saliva • Can be dispensed in a rinse bottle, spray bottle, or via an oral swab stick
xerostomia screening	• Older adults often present with xerostomia induced by medications used to control systemic diseases • Clinicians can screen for xerostomia using a specific instrument (sialometry) or by oral examination • A quick examination can be done by using a tongue blade touching the floor of the mouth or the buccal vestibule. If only the tip of the tongue blade is wet rather than a greater portion of the end of the blade, the result can be recorded as abnormal.

Fast Facts

Aging population	• According to the US Department of Health and Human Services, the number of adults aged ≥65 years worldwide will increase from 560 million in 2010 to 1.5 billion in 2050, or from 8% to 16% of the global population • In 2014 there were 46.2 million (14.5%) people over 65 years of age in the United States • Dentists should expect to treat more older adults in the future
Effects of aging on periodontium	• Thinning of epithelium • Reduction in vascularization, number of collagen fibers, elasticity of periodontal ligament and bone density • Increase in bone resorption and thickening of cementum
Interdisciplinary care team for older adults	• Geriatricians, nurses, dentists, and dieticians comprise the care team for older adults • Coordination between team members is important to allow older adults access to various treatment modalities
Functional status of older adults	Functional impairment affects the ability to perform oral self-care in older adults. Dentists can use ADOH to develop strategies to help older adults perform oral care. They should also consult with physicians or nurses to monitor the patient's functional status.
Oral diseases related to aging	Older adults are prone to some oral diseases, including caries (especially root caries), periodontal disease, oral cancer, xerostomia, and lesions/disorders related to removable prostheses
Aging and periodontitis	The prevalence of periodontal disease increases with age. However, this is caused by cumulative disease progression rather than increased disease susceptibility.
Periodontitis and systemic disease	For older adults, it is important to control both periodontal disease and systemic disease (e.g., diabetes and cardiovascular conditions) which are associated with each other, because the risks of having these diseases are higher in this age group
Periodontal treatment planning in older adults	It is important to consider medical history, functional status, and the ability to tolerate treatment while formulating the treatment plan. Patient values and preferences should also be considered.
Periodontal surgery for older adults	• Age is not a contraindication for periodontal surgery. However, clinicians should consider the compliance, medical history, and functional status of the older adult before performing surgery • Sometimes, supportive periodontal therapy may be more suitable for these patients than surgical therapy.
Periodontal health maintenance in older adults	• It is important to evaluate patients' access to supportive periodontal therapy and their ability to maintain oral hygiene • For older adults with disability, a lightweight electric-powered toothbrush may be more beneficial than a manual toothbrush.

Core Knowledge

Introduction

In the United States the proportion of older adults (≥ 65 years) is increasing; it is projected that treatment of older adults will be a large part of dental practice in the near future.[2] It is also clear that many of these older patients will retain their natural teeth.[3] Clinicians should have the knowledge and training to treat older adults effectively. Apart from the effect of aging on the different systems, including its effect on the oral cavity, treating older adults comes with its own set of challenges. The specific changes that occur in the periodontium with age are listed in Table 23.1.

Periodontal Disease in Older Adults

The current disease model emphasizes that gingivitis precedes periodontitis, but relatively few sites with gingivitis actually develop periodontitis. Based on this model, several observations are noted with regard to older adults:

- The prevalence of periodontal disease is low and potentially declining
- Progression of periodontal disease is infrequent and episodic in nature
- Active and inactive disease sites can coexist
- Periodontal disease occurs in a small, high-risk population of older adults
- Moderate levels of attachment loss are seen in a higher proportion of older adults, but severe attachment loss is noted only in a small proportion of this population
- With increasing age, there is a significant increase in the proportion of patients with gingival recession, increasing their risk for root caries

TABLE 23.1 Changes in the Periodontium With Age

Stochastic (randomly determined) changes	Structures become less soluble and more thermally stable
Physiological changes	Reduction or loss of tissue elasticity and decreased vascularity
Functional changes	Less mitotic activity and metabolic rate, reducing the healing capacity and rate
Clinical changes	• Thinning of oral epithelium and reduced keratinization • Reduction or loss of periodontal ligament tissue elasticity • Gingival recession • Attachment and bone loss • Thickening of cementum

Factors Affecting the Clinical Management of Older Adults

A multitude of factors can play a role in the delivery of care for the elderly population. Some of these factors are potential barriers to care, while others directly affect treatment outcomes; all should be carefully considered. Some of these key factors are summarized in Table 23.2.

Readers are encouraged to refer to Chapter 42 in *Newman and Carranza's Clinical Periodontology* (13th ed.) for more information on the effect of aging on the periodontium and on treatment considerations in elderly patients.

CLINICAL CORRELATE

Is age a risk factor for implant failure?

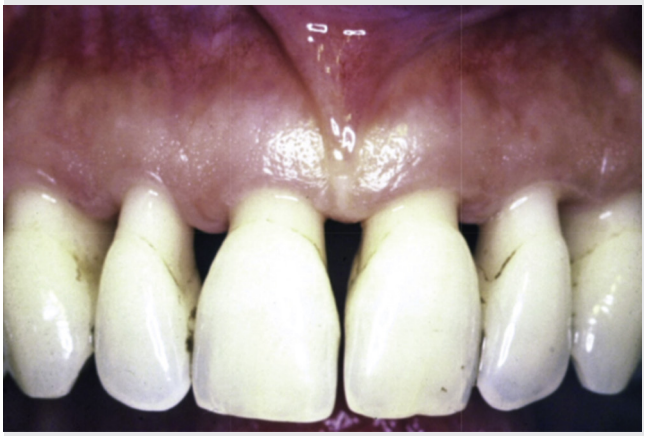
Dental implants can be successfully placed in the elderly population and can be maintained on a long-term basis, provided the systemic risk factors associated with this patient population (e.g., osteoporosis) are carefully considered and managed to minimize their effects on the treatment outcome.[4]

TABLE 23.2 Key Considerations in the Treatment of Older Adults

Systemic and health-related factors	• Elderly patients often present with multiple medical or mental health conditions and polypharmacy (intake of several medications) • These factors could significantly limit the delivery of care and may also influence the initiation and progression of periodontal disease • Alteration in dexterity may affect patient's oral self-hygiene measures • Risks and benefits of nonsurgical and surgical periodontal therapy should be carefully weighed prior to treatment execution
Psychosocial factors	• With changing priorities in life, older patients may not be compliant in maintaining adequate daily self-care • Other psychological conditions associated with age, such as geriatric depression, can exacerbate periodontal disease
Functional status	• Older adults are classified as functionally independent, frail, or functionally dependent • Functional impairments seen in frail (at risk of hospitalization) and functionally dependent individuals can have a significant impact on oral health and oral self-care
Barriers to care	• Common barriers to care include lack of insurance coverage, transportation issues, lack of finances, and medical complexity
Common oral conditions in elderly	• Clinicians should assess the oral cavity and manage conditions commonly observed in the elderly, such as root caries, disorders associated with xerostomia and wearing dentures (e.g., candidiasis) and oral manifestations of systemic diseases • Clinicians should also devise strategies to prevent such conditions

CASE-BASED LEARNING EXERCISE

Scenario: A 78-year-old female presented to one of her 6-monthly periodontal maintenance appointments. Pointing to the maxillary anterior sextant, she mentioned that from time to time, teeth in this area become very sensitive to cold. She was a controlled hypertensive, on lisinopril and also taking atorvastatin for hypercholesterolemia. The patient had a history of periodontal pocket reduction surgery in the maxillary anterior sextant. Clinical exam revealed generalized gingival recession with black triangles in the interproximal areas. Probing depths were in the range of 2–4 mm. The patient had premolar-to-premolar dentition and wore maxillary and mandibular removable partial dentures to restore her posterior teeth. Oral hygiene was good but signs of xerostomia were visible.



Questions

1. Which of the following is a common periodontal finding in elderly patients?
   - **a.** Gingival recession
   - **b.** Thinning of cementum
   - **c.** Thickening of oral epithelium
2. Root coverage can be predictably achieved in this clinical situation.
   - **a.** True
   - **b.** False
3. Which of the following is a common sequela of gingival recession in the elderly population?
   - **a.** Periodontal abscess
   - **b.** Gingival abscess
   - **c.** Root caries
   - **d.** Root resorption
4. All of the following medications can cause xerostomia (dry mouth) except:
   - **a.** Selective serotonin reuptake inhibitors (SSRIs).
   - **b.** Hydrochlorothiazide.
   - **c.** Chlorpheniramine maleate.
   - **d.** Pilocarpine.

From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

This chapter was developed from Chapter 42 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Solutions

1. Answer: a

Explanation: In the elderly population, it is very common to observe generalized gingival recession. In this patient, the past history of periodontal surgery certainly had an effect on the severity of recession defects.

2. Answer: b

Explanation: One of the important local (anatomic) determinants of predictable complete root coverage is the height of interdental bone. Based on the clinical presentation, it is safe to assume that the interdental bone levels in

the maxillary anterior sextant are at more apical locations than they should be, so achieving complete root coverage in this scenario is not feasible. The distance from the bone level to the contact point is an important parameter that dictates the presence or absence of gingival papilla.

3. Answer: c

Explanation: Of the conditions listed, root caries is very common in older adults. Exposed roots, in combination with xerostomia (in this patient), make this an ideal scenario for the initiation of caries.

4. Answer: d

Explanation: Pilocarpine stimulates salivary flow. Consumption of the other listed medications is associated with xerostomia.

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24

Periodontal Treatment of Female Patients

Relevant Terminology

Terminology/Abbreviation	Explanation
hormone replacement therapy (estrogen replacement therapy)	• A treatment with estrogen alone (or estrogen with progestin) that relieves symptoms and long-term biologic changes of menopause, such as loss of bone mass • May affect clotting times, prolong the effects of other medications, or interfere with the absorption or effectiveness of prescription medications
menopause	• A point in time 12 months after the last menstrual period, that occurs between ages 44 and 55 • Usually lasts about 7 years • Associated with symptoms of estrogen deficiency
ovarian cycle	• Consists of follicular phase and luteal phase • During the follicular phase, levels of follicle-stimulating hormone (FSH) are elevated. Estradiol (E2) is synthesized by the developing follicle, and peaks at 2 days before ovulation • During the luteal phase, the developing corpus luteum synthesizes estradiol and progesterone to complete the rebuilding of the endometrium for implantation of a fertilized egg • If the egg is not fertilized, the corpus luteum involutes, ovarian hormone levels drop, and menstruation ensues
perimolysis	Smooth erosion of enamel and dentin, typically on the lingual surfaces of maxillary anterior teeth in patients with eating disorders
pre-eclampsia	A life-threatening condition in late pregnancy, characterized by high blood pressure and excess protein in the urine
premenstrual syndrome (PMS)	• A set of physical and psychological symptoms that start up to 2 weeks before menstruation • Hormonal fluctuation is considered as a cause • Genetic and environmental factors affect the likelihood of PMS • Frequently treated with antidepressants such as serotonin reuptake inhibitors (SSRIs)
prevotella intermedia	Gram-negative pathogenic bacterium associated with puberty gingivitis; uses ovarian hormones as a substitute for vitamin K as a growth factor
puberty	• Occurs between the ages of 11 and 14 on average • Increase in the production of sex hormones (estrogen and progesterone)
supine hypotensive syndrome	• Commonly seen in pregnant patients in the supine position from the second trimester onward • Caused by decreased cardiac output (decreased systolic pressure of 15–30 mm Hg) due to the compression of the inferior vena cava and aorta by the gravid uterus • Symptoms include tachycardia, diaphoresis, nausea, vomiting, pallor, weakness, lightheadedness, and dizziness • Symptoms are resolved with change in positioning

Fast Facts

Signs of eating disorders	• Perimolysis and enlargement of the parotid glands associated with binging and purging • Diminished salivary flow rate • Mucous membrane sensitivity, gingival erythema, and caries susceptibility
Periodontal disease and preterm and low birth weight infants	• Current opinion suggests a possible association between maternal periodontal disease and preterm, low birth weight infants • Adverse pregnancy events can result from direct infection by oral microorganisms, or indirectly by the translocation of bacterial by-products (e.g., lipopolysaccharides) and the action of maternally produced inflammatory mediators
Effects of elevated estrogen and progesterone on maternal immune response during pregnancy	• Suppressed immune response during pregnancy suggests an increased susceptibility to developing gingival inflammation • Suppression of cell-mediated immunity • Reduction in neutrophil chemotaxis • Suppression of antibody and T-cell responses • Reduction in $CD4^+$:$CD8^+$ ratio of T cells • Cytotoxicity directed against macrophages and B cells • Decrease in absolute numbers of $CD3^-$, $CD4^-$, and $CD19^+$ cells in peripheral blood • Stimulation of prostaglandin production
Effects of elevated estrogen and progesterone during pregnancy on subgingival plaque composition	• Increase in anaerobic:aerobic microbial ratio • Higher proportions of *Prevotella intermedia*, *Bacteroides melaninogenicus*, *Porphyromonas gingivalis*
Effects of elevated estrogen on the gingiva	• Increased cellular proliferation in blood vessels • Decreased keratinization while increasing epithelial glycogen
Effects of elevated progesterone on the gingiva	• Progesterone increases vascular dilation and permeability of vessels, resulting in edema and accumulation of inflammatory cells • Increased angiogenesis • Increased metabolic breakdown of folate • Altered rate and pattern of collagen production • Decreased plasminogen activator inhibitor type 2 (increased tissue proteolysis)
Prenatal fluoride use	The American Dental Association does not recommend the use of prenatal fluoride because its efficacy has not been demonstrated
US FDA drug classification system based on potential to cause birth defects	• A—Controlled studies show no risk, and the possibility of fetal harm appears remote • B—No evidence of risk in humans, based on animal studies alone or on both animal and human studies • C—Risk cannot be ruled out, either concerning animal data or no data for humans • D—Positive evidence of human fetal risk, but use may be justified in some circumstances • X—Contraindicated in pregnancy because of evidence of fetal risk based on animal or human studies or human experience; the risk of using the drug in pregnant women outweighs any possible benefit
Local anesthetics during pregnancy	Category B includes lidocaine, prilocaine, etidocaine, and articaine. Others are category C
Analgesics during pregnancy	Category B includes acetaminophen, hydrocodone, and oxycodone. Aspirin and ibuprofen should not be used in the third trimester (category D)
Antibiotics during pregnancy	• Tetracycline should not be used due to adverse effects on tooth development and discoloration of teeth (category D) • Clarithromycin should not be used due to adverse effects on pregnancy outcomes and embryonic and fetal development in animals (category D)
Sedative-hypnotic drugs during pregnancy	• Benzodiazepines and barbiturates should not be used (category D) • Nitrous oxide should be avoided during the first trimester; otherwise, use with caution (no category assignment)
Medications to be avoided during breastfeeding	The amount of drug excreted in the breast milk is usually not more than 1%–2% of the maternal dose. The mother should take prescribed drugs just after breastfeeding and then avoid nursing for at least 4 hours (longer if possible). The following drugs should be avoided: • aspirin • tetracycline, ciprofloxacin, metronidazole, gentamycin, vancomycin • benzodiazepine, barbiturates

Fast Facts—cont'd

Periodontal manifestations of oral contraceptives (OCs) and implications	• An exaggerated response to local irritants occurs in gingival tissues in OC users • Caused by altered microvasculature, increased gingival permeability, and increased synthesis of prostaglandin • Increased level of *Bacteroides* species • The patient should be informed of the side effects of OCs and the need for meticulous home care and compliance with periodontal maintenance care • Due to potential antibiotic interference with OCs, OC users should be informed of the possible reduced efficacy of OCs during antibiotic therapy and the need for an additional method of contraception
Oral changes caused by menopause	• Menopausal symptoms are associated with estrogen deficiency. Estrogen affects cell proliferation and differentiation, and keratinization of the gingival epithelium • Thinning of the oral mucosa, oral discomfort (i.e., burning mouth), gingival recession, xerostomia, altered taste sensation, alveolar bone loss, and alveolar ridge resorption are manifestations of menopause • Possible alveolar bone loss and tooth loss due to osteopenia or osteoporosis are associated with menopausal or postmenopausal changes • Improved periodontal status has been reported in women on hormone replacement therapies
Clinical management of patients at menopause or postmenopause	• Thorough review of medical history • Brushing with an extra soft toothbrush to prevent trauma to the thinning gingiva • Avoid abrasive dentifrices • Medications for osteoporosis include calcium plus vitamin D supplementation, sodium fluoride, bisphosphonates, selective estrogen receptor modulators, and parathyroid hormone
Recommendations for optimal calcium intake	• Premenopausal women (25–50 years): 1000 mg/day • Postmenopausal women (estrogen therapy): 1000 mg/day • Postmenopausal women (no estrogen therapy): 1500 mg/day • Men (25–65 years): 1000 mg/day • Women and men > 65 years: 1500 mg/day

Core Knowledge

Introduction

Hormonal fluctuations that occur throughout the lives of females have a profound effect on the periodontium. Clinicians should be well aware of the hormonal influences and management considerations of a female patient depending on her stage of life, from puberty all the way to menopause. Readers are encouraged to refer to Chapter 41 in *Newman and Carranza's Clinical Periodontology* (13th ed.) for detailed information on periodontal treatment considerations in female patients.

CLINICAL CORRELATE

What is the current opinion on the association between periodontitis during pregnancy and adverse pregnancy outcomes?

Available evidence suggests a possible association between periodontal disease and preterm, low-birth-weight infants. Adverse pregnancy events can result from an infection that is mediated by one of two major pathways: directly by oral microorganisms, or indirectly by the translocation of bacterial products such as endotoxin (lipopolysaccharide) and the action of maternally produced inflammatory mediators. At this point, however, it is unclear whether periodontal treatment during pregnancy has a beneficial effect on preventing preterm birth.[1,2]

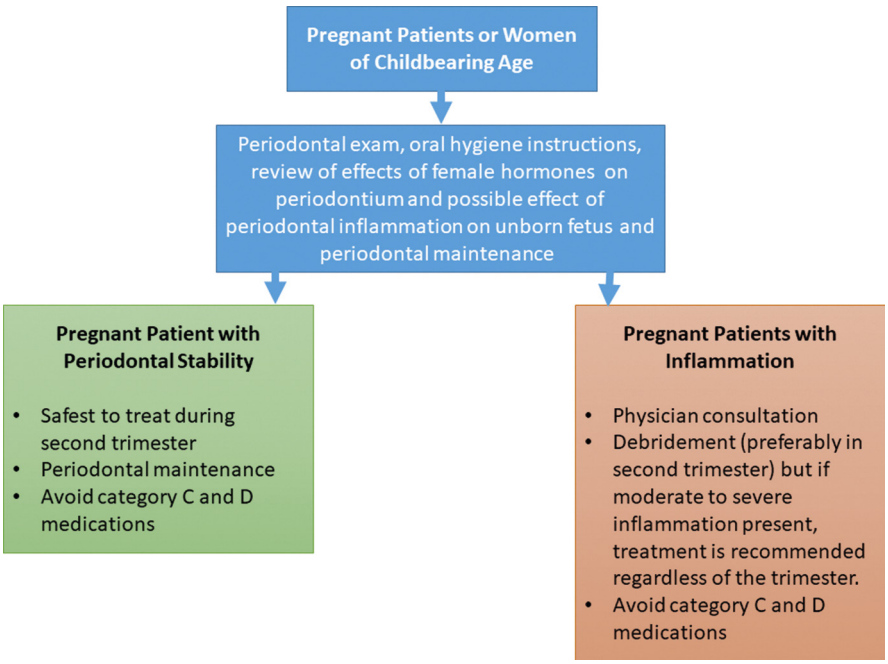
The periodontal manifestations, systemic effects, and management considerations in female patients are summarized in Table 24.1.

Current Recommendations for Treating Pregnant Patients

A simple treatment algorithm for pregnant patients presenting for periodontal therapy is presented in Fig. 24.1.

TABLE 24.1 Periodontal Manifestations and Treatment Considerations in Female Patients

Stage of Life	Periodontal Manifestations	Treatment Considerations
Puberty	• Periodontal tissues can have an exaggerated response to local factors • *Puberty gingivitis:* a hyperplastic reaction of the gingiva can occur. Inflamed tissues become erythematous, lobulated, retractable, and bleed easily • Histologically, the appearance is consistent with inflammatory hyperplasia	• Milder gingivitis: responds well to scaling and root planing, with frequent oral hygiene reinforcement • Severe gingivitis: may require microbial culturing, antimicrobial mouthwashes, and antibiotic therapy • More frequent periodontal maintenance appointments when periodontal instability is identified
Menstruation	• Interaction between estrogen and cells of the immune system contributes to increased gingival inflammation • Gingival tissues have been reported to be more edematous during menstruation and erythematous before the onset of menses in some women • An increase in gingival exudate has been noted during the menstrual period and is associated sometimes with a small increase in tooth mobility	• Increased gingival bleeding and tenderness associated with the menstrual cycle require close periodontal monitoring • Periodontal maintenance should be adjusted to the individual patient's needs. If problematic, 3- to 4-month recall intervals should be recommended • Patients in premenstrual syndrome phase are more sensitive and less tolerant to procedures and require gentle care
Pregnancy	• *Pregnancy gingivitis:* very common condition characterized by erythema, edema, hyperplasia, and increased bleeding. Presentation can range from slight inflammation to severe hyperplasia, pain, and bleeding • *Pregnancy tumor:* less common than pregnancy gingivitis, this condition is indistinguishable from the pyogenic granuloma of nonpregnant individuals. Tumors can be sessile or pedunculated and ulcerated, ranging in color from purplish red to deep blue	• Scaling, polishing, and root planing can be performed safely during pregnancy • Early in the second trimester is the safest period for providing routine dental care. Major elective oral or periodontal surgery may be postponed until after delivery • Painful pregnancy tumors that interfere with mastication, or bleed or suppurate after mechanical therapy, may require excisional biopsy before delivery • Careful selection of medications is necessary in this patient population. In most cases, amoxicillin and acetaminophen are safe to use • Tetracycline and ibuprofen are contraindicated, especially in the third trimester.
Menopause	• Changes in the oral cavity include oral mucosal thinning, burning mouth, altered taste sensation, gingival recession, and xerostomia • Alveolar bone loss and alveolar ridge resorption due to osteopenia or osteoporosis	• Closely monitoring patient's periodontal stability • Performing titrated periodontal maintenance and informing patient about potential risks of hormone depletion to the oral tissues, and physician consultation (if needed)

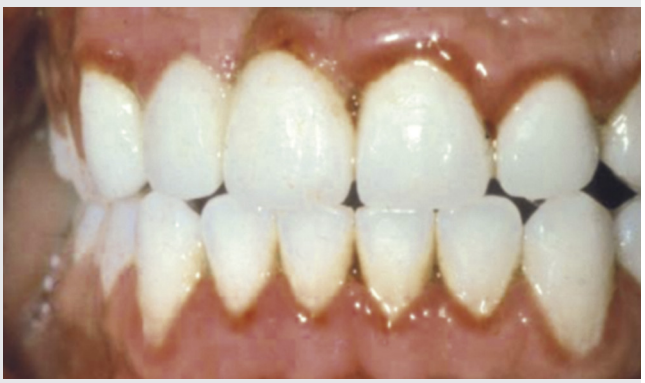


• **Fig. 24.1** Treatment Algorithm for Clinical Management of Pregnant Patients.

Adapted from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

CASE-BASED LEARNING EXERCISE

Scenario: A 29-year-old female patient presented to the dental clinic with a chief complaint of "severe pain and discomfort in the gums, which bleed when brushing." Clinical exam revealed generalized severe inflammation (erythema and edema) of the marginal gingiva. Probing depths ranged from 2 to 5 mm. Radiographic exam revealed normal bone levels even at sites with 4- and 5-mm probing depths. The patient was in her second trimester of pregnancy. She had no known medical conditions and was not taking any medications.



Questions

1. Which of the following conditions did this patient present with?
   a. Periodontal abscess.
   b. Gingival abscess.
   c. Pregnancy gingivitis.
   d. Necrotizing ulcerative gingivitis.
2. What is the ideal trimester to perform dental prophylaxis?
   a. Second.
   b. First
   c. Third
3. The fact that sites with probing depths of 4 and 5 mm are associated with normal bone levels indicate these to be _____
   a. Periodontal (true) pocket.
   b. Gingival (pseudo) pocket.
   c. normal sulcus.

Clinical photo attribution: Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

This chapter was developed from Chapter 41 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Solutions

1. Answer: c

Explanation: Based on the history and clinical presentation, the gingival condition is most likely pregnancy gingivitis.

2. Answer: a

Explanation: Of the three trimesters of pregnancy, the second is the safest in which to get dental treatment done. Major elective procedures can be postponed until after delivery.

3. Answer: b

Explanation: Based on the clinical and radiographic findings, the pockets in this patient are mainly gingival (pseudo) pockets. Resolution of inflammation following treatment will result in the reduction of these inflammation-induced pocket depths.

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25

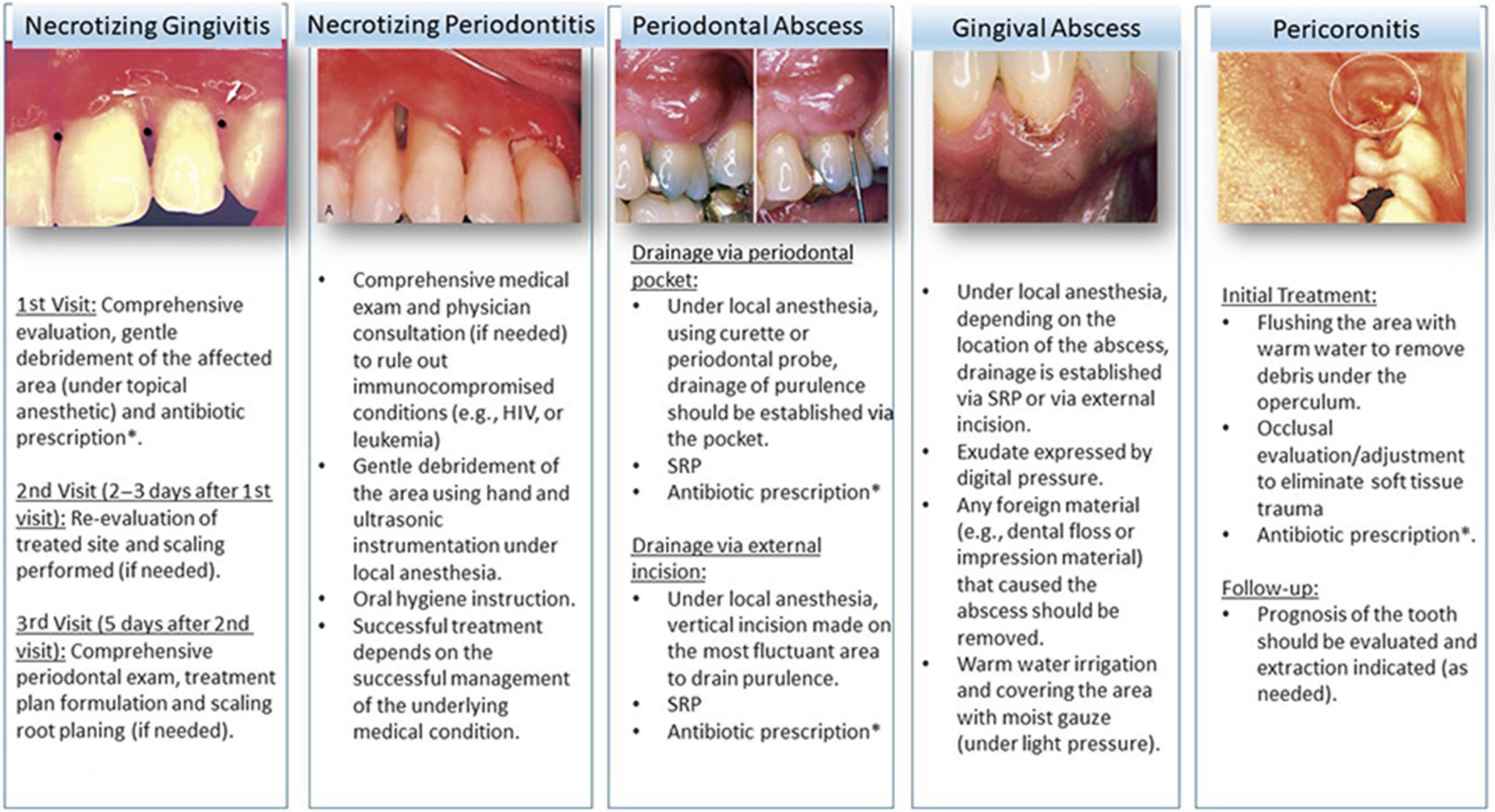
Treatment of Acute Gingival and Periodontal Conditions

Relevant Terminology

Terminology / Abbreviation	Explanation
acute abscess	Painful, red, edematous, smooth, and ovoid swelling of the gingival tissues, containing exudate.
chronic abscess	After the spreading of infection has been contained, the abscess manifests with dull pain associated with a periodontal pocket and often present with a fistulous tract.
contouring of gingiva	Surgical reshaping of the gingiva, used to correct shelf-like gingival margins resulting from acute conditions.
Incision and Drainage	Minor surgical procedure done to relieve pressure and release purulent exudate from within a fluctuant abscess cavity.
herpetic vesicles	Small, raised, fluid-containing lesions of the oral mucosa that are very high in viral titer; represent a contagious phase.
Operculum	Soft tissue that overlies the crown of an incompletely erupted tooth.

Fast Facts

Necrotizing gingivitis (NG)	Management of NG centers around alleviation of pain, reduction of the microbial load, and removal of necrotic tissue to the degree that repair and regeneration of normal tissue barriers can be reestablished. Subgingival scaling and curettage are contraindicated during the first appointment, because these procedures may spread the infection into the deeper tissues and/or cause bacteremia.
Treatment expectations for NG lesions	Even in cases of severe gingival necrosis, healing often leads to restoration of the normal gingival contour, although the normal gingival architecture may be achieved only after several weeks or months.
Primary herpetic gingivostomatitis	Treatment consists of early diagnosis and immediate initiation of antiviral therapy against the herpes simplex virus.
Treatment expectations for primary herpetic gingivostomatitis	Antiviral therapies (e.g., acyclovir) can shorten the duration of the lesions if administered within the first 36 hours of symptom presentation.
Periodontal abscess	Following anesthesia, the pocket wall is gently retracted with a periodontal probe or curette in an attempt to initiate drainage through the pocket entrance; gentle digital pressure and irrigation may be used to express the exudate and drain the pocket. If unsuccessful, external drainage via incision is utilized. Extensive scaling and root planing is deferred until initial healing.
Gingival abscess	Removal of any foreign material (e.g., dental floss, impression material), irrigation with warm water, and drainage with moist gauze under light pressure usually suffices for resolution.
Pericoronal abscess	The acute pericoronal abscess is properly anesthetized for comfort, and drainage is established by gently lifting the soft tissue operculum with a curette. If the underlying debris is easily accessible, it may be removed, followed by gentle irrigation with sterile saline. Repeated abscesses usually necessitate extraction of the associated semi-impacted tooth.



• **Fig. 25.1** Management of Acute Gingival and Periodontal Conditions. Patients with acute gingival and periodontal conditions commonly experience pain and therefore pain management during initial therapy is critical. Thorough medical history-taking is vital in elucidating the underlying etiology for the clinical presentation. For all of the listed conditions, on a case-by-case basis, rinsing with warm saline or chlorhexidine rinse can be recommended during the immediate posttreatment period. In the 2017 classification, *combined* EPL lesions are also mentioned with acute conditions like abscesses and NPD (although EPL lesions have chronic presentations too). They can be painful, cause discomfort and rapid tissue destruction. Chapter 26 in this book deals separately with the diagnosis and management of these lesions. *The use of systemic antibiotics should be reserved only for patients with signs and symptoms of systemic spread of infection. Specifically, antibiotics should be used with caution in patients with known immunosuppressive conditions due to their potential to develop opportunistic infections. *HIV*, human immunodeficiency virus; *SRP*, scaling and root planing. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

Core Knowledge

Periodontal diseases usually manifest as chronic conditions and their presence is not immediately perceived by dental patients. But a group of diseases and conditions—including necrotizing ulcerative oral diseases, periodontal abscesses, and primary herpetic gingivostomatitis—typically manifest with pain that leads patients to seek immediate treatment.

Broad guidelines for the management of common acute gingival and periodontal conditions are presented in Fig. 25.1. For additional information, readers are referred to Chapter 13 of this book and Chapters 44 and 45 in *Newman and Carranza's Clinical Periodontology* (13th ed.).

CLINICAL CORRELATE

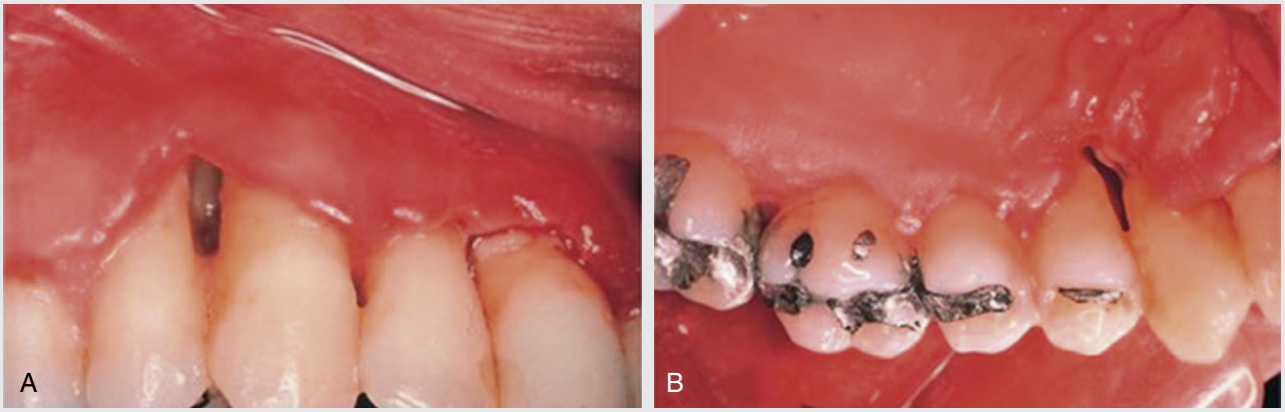
What are the expectations in treating a tooth with periodontal abscess?

Teeth with periodontal abscesses often appear to have very limited radiographic bone support, and clinical findings (e.g., mobility and furcation involvement) that deem their prognosis poor. However, with proper treatment followed by consistent preventive periodontal maintenance, abscessed teeth with significant bone loss may be retained for many years.

Clinical photo attribution: Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

CASE-BASED LEARNING EXERCISE

Scenario: A 38-year-old Caucasian male presented with a chief complaint: "There's a gap between my teeth and that area hurts badly." He was HIV positive with a CD4+ count of 150 cells/μL; his viral load was undetectable. He had no known drug allergies but reported having multiple episodes of recurrent pneumonia over the previous 12 months. He had a history of smoking (one pack per day for 28 years). Generalized probing depth ranged from 2 to 4 mm. There was severe, localized recession with necrosis and ulceration of the interproximal tissues between the maxillary right premolar and canine. The patient also complained of fever over the past 2 days, and a clinical exam revealed cervical lymphadenopathy. Radiograph indicated bone loss at the site of interest. There was localized moderate-severe biofilm with moderate-severe gingival erythema.



Questions

1 Based on the patient's CD4+ counts, he will be classified by Centers for Disease Control and Prevention as group:
   a. A.
   b. B.
   c. C.

2 Based on the clinical and radiographic information presented, what is the likely diagnosis?
   a. Necrotizing gingivitis
   b. Periodontal abscess
   c. Necrotizing periodontitis
   d. Gingival abscess

3 What is the most appropriate instrument to assess the horizontal involvement of the possible furcation lesion #5?
   a. Explorer 17
   b. Explorer 23
   c. Periodontal probe UNC15
   d. Nabers probe

4 Based on the information presented, systemic antibiotics may be indicated to treat this form of periodontitis.
   a. True
   b. False

This chapter was developed from Chapters 43, 44, and 45 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1 Answer: c

Explanation: According to the CDC, HIV patients are categorized into group A (≥500 cells/μL; asymptomatic), B (200–499 cells/μL; symptomatic), or C (<200 cells/μL: AIDS indicator condition) based on $CD4^+$ counts and clinical presentation.

2 Answer: c

Explanation: Clinical presentation of necrosis within periodontal attachment apparatus with associated bone loss is indicative of necrotizing periodontitis. This condition is prevalent in patients who are HIV positive and immunosuppressed.

3 Answer: d

Explanation: Nabers probe is the most appropriate instrument to assess the horizontal involvement of furcation lesions.

4 Answer: a

Explanation: Considering the clinical and periodontal findings with potential systemic involvement, a systemic antibiotic regimen might be indicated to reduce the gingival inflammation.

26

Diagnosis and Management of Endodontic-Periodontic Lesions

Relevant Terminology

Terminology / Abbreviation	Explanation
accessory canals	Routes of periodontal and pulpal communication found along the length of the root canals; these are additonal to the main root canal and add to the complexity of the root canal anatomy
true combined lesion	Primary pulpal and primary periodontal infections can progress extensively until the apical and cervical bone destruction zones overlap
dentinal tubules	Permeable microtubular formations along the dentin body, ranging from 0.9 µm to 2.5 µm in diameter; also allow for communications between pulpal and periodontal spaces in areas lacking cementum
primary periodontal lesion	Extensive breakdown of alveolar crestal bone due to inflammatory periodontitis that progresses from the cervical area to the root apex causing secondary pulpal involvement
primary pulpal lesion	Chronic periradicular periodontitis caused by pulpal infection in which a periapical infection can develop and progress coronally
retrograde peri-implantitis	Radiographically evident periapical lesion at the apex of an osseointegrated implant, caused by persistence of endodontic bacteria and inflammatory cells, leading to peri-implant infection
retrograde periodontitis	Pulpal infection leading to periodontal tissue destruction that progresses cervically from apical / furcation area towards gingival margin
anatomic communication	Physical channels connecting pulp and periodontium created naturally during development of tooth; e.g., apical foramina, lateral/accessory canals, dentinal tubules
non-anatomic communication	Physical channels connecting pulp and periodontium created by iatrogenic defects; e.g., root fractures, root perforations

Fast Facts

Endodontic-periodontic lesions	Periodontium and pulpal spaces are separated by a hard shell of dentin and cementum but communicate with each other via various portals, through which bacteria may trigger inflammatory responses in communicating tissues
Pathways of pulpal infection to the periodontium	The apical foramina are the main route of communication of pulpal infection leading to secondary infection and breakdown of tissues in the periodontium; accessory canals, lateral canals and dentinal tubules are additional potential pathways of communication between both entities.
Pathways of periodontal infection to the pulp	Dentinal tubules and lateral canals along the pulpo-dentinal complex provide avenues for bacterial invasion from the periodontal pocket
Apical pathoses of endodontic origin	These are inflammatory processes occurring in periradicular tissues surrounding root apices; they are caused by polymicrobial intracanal biofilms that trigger subsequent immunologic responses
Pulpal disease classification	Dental examination including type and localization of pain, percussion, and thermal testing and radiographic imaging of the periapical tissues is necessary to diagnose pulpal diseases
Differential diagnosis	Primary pulpal lesions tend to affect teeth with compromised coronal integrity and no pulp vitality, while primary periodontal lesions are more likely to affect intact, vital teeth with crestal bone loss that is generalized to the remaining dentition

Continued

Fast Facts—cont'd

Retrograde peri-implantitis management	Surgical debridement of the lesion is associated with favorable outcomes unless implant stability has been compromised
Pathogenesis of sinus tract of pulpal origin	Pulpal inflammation can lead to reduction in pulpal blood flow caused by an increase in intrapulpal pressure, causing pulpal necrosis. Pulpal necrosis, if left untreated, can cause chronic inflammation of periradicular tissues and abscess formation, leading to a draining sinus tract

Core Knowledge

Introduction

Bacterial infection in pulpal and periodontal tissues usually occurs independently in each space; however, there are some circumstances in which infections spread from one entity to another via anatomic communication channels such as apical foramen, lateral and accessory canals, dentinal tubules, and crack lines. Migration of microorganisms and inflammatory mediators between the root canal and the periodontium may lead to the development of endodontic-periodontal lesions (EPL).

Etiology

EPL are usually a result of pathologic communications between the pulpal and periodontal tissues of a given tooth that may be triggered by:[1]

1. **Endodontic and/or periodontal infections:**
   - Primary pulpal involvement due to a carious lesion, with secondary periodontal involvement;
   - Primary periodontal destruction that secondarily affects the root canal
   - By concomitant presence of both pathologies
2. **Trauma and/or iatrogenic factors:**
   - Root/pulp chamber/furcation perforation (e.g., because of faulty root canal instrumentation or tooth preparation for post-retained restorations)
   - Root fracture (e.g., due to trauma or preparation for post-retained restorations)
   - External root resorption or pulp necrosis (e.g., because of trauma) draining through the periodontium

EPLs caused by trauma or iatrogenic factors usually have a hopeless prognosis, whereas EPLs associated with dental infections may have a prognosis ranging from favorable to hopeless depending on many factors.

Fig. 26.1 provides a review of the pathogenesis and differential diagnosis of EPL.

Classification of Endodontic-Periodontal Lesions

The main drawback of the 1999 classification was that the various categories of EPL (primary endodontic, primary periodontic, combined lesion, etc.) were based on the primary source of infection, i.e., whether the infection was of pulpal or periodontal origin. It was, however, pointed out that determining the primary source of infection is not relevant for the treatment of EPL because in most cases both the pulpal and periodontal tissues require treatment. Hence the diagnosis and classification of EPL should be based on both the disease status and on the prognosis of the involved tooth. This would determine the first step of the treatment planning—to make a decision on whether to maintain or extract the tooth.

Based on this rationale, EPL lesions were then classified (Table 26.1) according to the signs and symptoms that have a direct impact on their prognosis and treatment (e.g., presence/absence of fractures/perforations, presence/absence of periodontitis, extent of periodontal destruction).[1]

CLINICAL CORRELATE

What are other possible reasons for extraradicular infections that affect the periodontium and are not responsive to routine endodontic therapy?

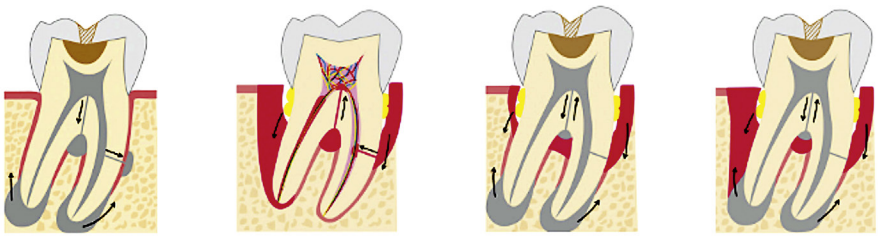
An extraradicular infection occurs periapically along the external surface of the root apex and can lead to, or exacerbate, acute periapical abscess development. It can occur independent of intraradicular infection and may be due to:

- **Periapical actinomycosis**—this may develop when infected intraradicular tissue or debris is moved past the apex during instrumentation. It does not respond to routine endodontic therapy and requires apical surgery.
- **Accumulation of calculus on the external surface of the root apex**—the origin of calculus around the root apex is thought to be the accumulation and calcification of plaque from the external oral environment by means of the open persistent sinus tract present in these cases. This is associated with failed periapical lesion healing, persistent sinus tract formation, and refractory apical periodontitis.

Management of Endodontic-Periodontal Lesions

Based on many factors, management includes one or more treatment modalities:

- establish drainage (e.g., incision and drainage of abscess)
- nonsurgical pocket-reduction therapy
- administration of antimicrobials
- endodontic treatment
- surgical periodontal therapy
- periapical surgery
- splinting of mobile tooth and minor occlusal adjustments
- extraction of the affected tooth



Pathogenesis	Lesion Origin	Primary endodontic	Primary periodontic	Independent lesions of endodontic and periodontal origin present simultaneously	True combined endo-perio lesions
	Pathways of Infection Spread (black arrows)	From pulp to periodontium via • Lateral and accessory canals • Apical foramen	From periodontium to pulp via • Lateral and accessory canals • Apical foramen	Spreads both ways without confluence of primary lesions	Spreads both ways with confluence of primary lesions
Diagnosis	Patient Symptom	Varies*	Mild discomfort	Varies*	Varies*
	Coronal Integrity	Compromised	Intact	Compromised	Compromised
	Vitality	Nonvital	Vital	Nonvital	Nonvital
	Radiographic Lesions	Periapical radiolucency	Crestal bone loss	Separate periapical radiolucency & crestal lesions	Continuous bone lesions from alveolar crest to root apex
	Probing	Narrow,deep probing depth upto apex	True pocket	True pocket	True pocket with narrow probing to apex

*Varies: chronic lesions are usually asymptomatic; acute lesions display pain with no radiographic evidence of pathology.

• **Fig. 26.1** Endodontic-Periodontal Lesions: Pathogenesis and Differential Diagnosis.

Biologic effects of pulpal infection on periodontal tissues:

- Early inflammatory changes in the pulp exert very little effect on the periodontium.
- When the pulp becomes necrotic, however, a significant inflammatory response traversing the apical foramen, the furcation accessory canals, lateral canals, and dentinal tubules can occur. This can lead to secondary involvement of the periodontium.
- Extension of the infection through the periodontal tissues can result in a localized or diffuse swelling or cellulitis that invades the various fascial spaces. However, a more common sequela is eruption of infection through the labial, buccal, or lingual mucosa, resulting in a draining sinus tract. If the path of least resistance for the infectious process and inflammatory fluid is along the attached gingiva, the infection may dissect the periodontal ligament space and open into the gingival sulcus, resulting in the formation of a deep but narrow periodontal pocket.

Biologic effects of periodontal infection on the dental pulp:

- Periodontal disease seems to have a smaller influence on the pulpal tissues compared with the influence of pulpal disease on the periodontium.
- Advanced periodontal disease may infect the pulpal tissue as a retrograde infection proliferating through large accessory canals on the lateral surfaces of the tooth and the area where the main canal exits the tooth apex.

Differential diagnosis of EPL—the table provides a review of factors that help with differential diagnosis. EPL may present in acute or chronic form with the following general characteristics, irrespective of the origin of the infection:[1]

- Symptoms: Spontaneous pain, tooth mobility, foul taste/smell
- Primary signs: narrow and deep probing pocket depths that may reach the apical portion of the root with / without altered pulpal response to vitality tests;
- Secondary signs/symptoms: radiographic evidence of bone loss (apical/furcation region); pain on palpation/percussion; purulent exudate/suppuration; sinus tract/fistula

(Note: This table is a broad guideline only; every situation requires careful and thorough assessment by appropriate endodontic tests and periodontal examination.)

Modified from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

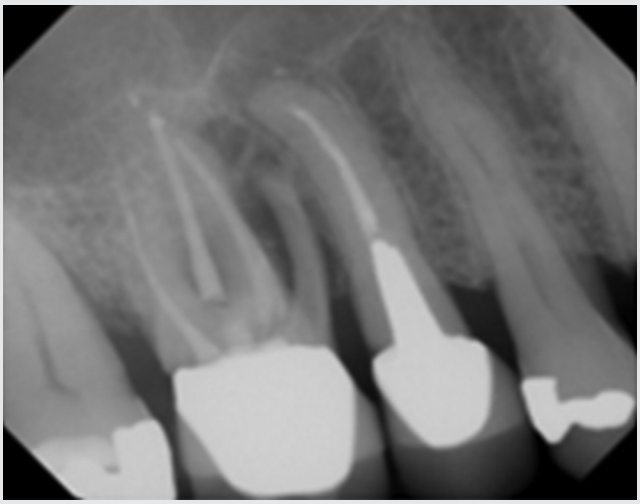
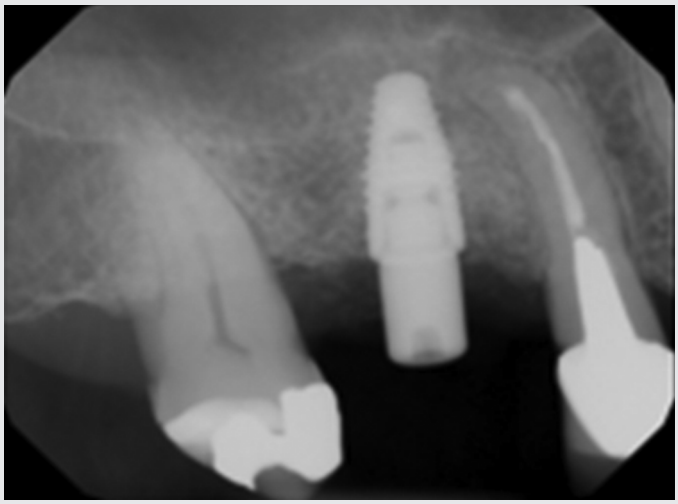
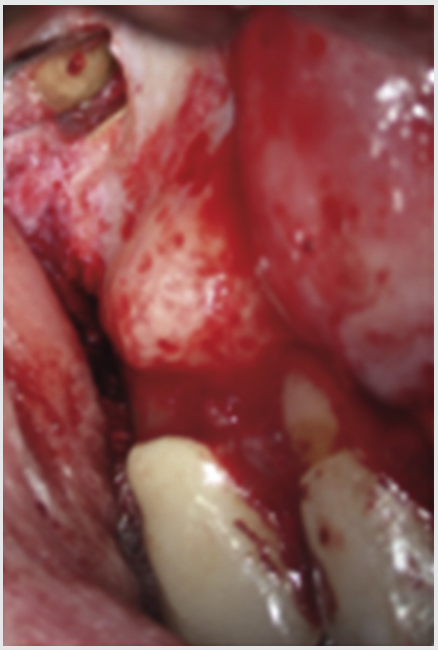
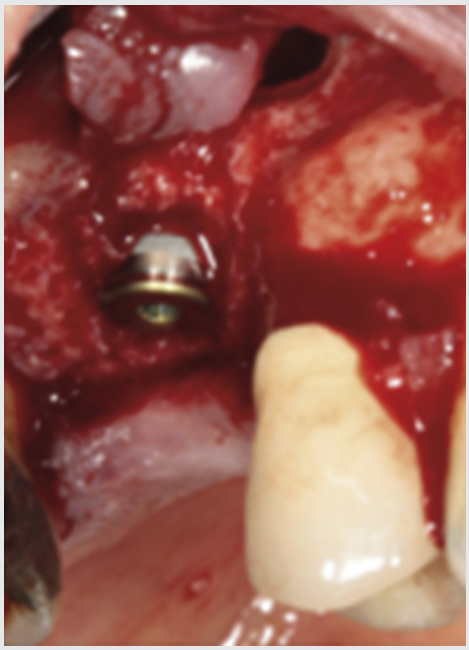
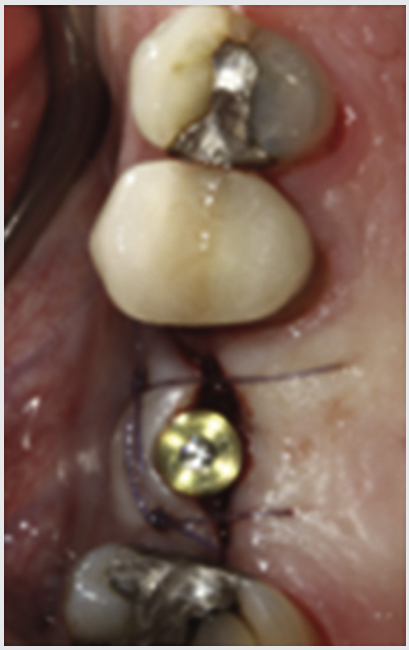
TABLE 26.1 2017 Classification of Endodontic-Periodontal Lesions[1]

Endo-Periodontal Lesion With Root Damage	Root fracture or cracking Root canal or pulp chamber perforation External root resorption	
Endo-Periodontal Lesion Without Root Damage	Endo-periodontal lesion in periodontitis patient	**Grade 1:** narrow deep periodontal pocket in 1 tooth surface **Grade 2:** wide deep periodontal pocket in 1 tooth surface **Grade 3:** deep periodontal pockets in >1 tooth surface
	Endo-periodontal lesion in non-periodontitis patient	**Grade 1:** narrow deep periodontal pocket in 1 tooth surface **Grade 2:** wide deep periodontal pocket in 1 tooth surface **Grade 3:** deep periodontal pockets in >1 tooth surface

Endo-periodontal lesion (EPL) with root damage: prognosis is generally 'hopeless'; EPL lesion without root damage: prognosis ranges from favorable to 'hopeless' depending on (a) current periodontal destruction around affected tooth and (b) overall periodontal status. (The aim of the classification is to guide the clinician's decision on whether to retain or extract the tooth, making the new classification more treatment- / prognosis- based, rather than etiology-based.)

CASE-BASED LEARNING EXERCISE

Scenario: A 71-year-old male presented with the chief complaint "My upper tooth is moving." Medical history includes controlled hypertension and allergy-induced asthma. Dental history: referral from an endodontist who diagnosed #3 with pulpal diagnosis of previously root canal–treated teeth and periapical diagnosis of symptomatic apical periodontitis (#3, including a vertical root fracture). The treatment plan generated was extraction of #3 and site development for future implant placement. Ridge preservation was performed and implant was placed subsequently.

Questions

1. Apart from root fracture, what are the other means of infection spread between pulp space and periodontium?
   - **a.** Lateral canals
   - **b.** Apical foramen
   - **c.** Dentinal tubules
   - **d.** All of the above
2. While performing this combined treatment, which of the following anatomic structures was in close proximity to the surgical site?
   - **a.** Zygomatic arch
   - **b.** Nasopalatine duct
   - **c.** Maxillary sinus
   - **d.** Orbital cavity
3. For a primary endodontic lesion with secondary periodontal involvement, the sequence of treatment should be:
   - **a.** Endodontic therapy only.
   - **b.** First perform periodontal therapy and reevaluate in 2–3 months; perform endodontic therapy as needed.
   - **c.** First perform endodontic therapy and reevaluate in 2–3 months; perform periodontal therapy as needed.
   - **d.** Periodontal therapy only.
4. When an endodontic lesion that is progressing coronally joins with a periodontal pocket, it is called a(n):
   - **a.** Primary endodontic lesion with secondary periodontal involvement.
   - **b.** Primarily periodontal lesion with secondary endodontic involvement.
   - **c.** True combined lesion.
   - **d.** Periodontal lesion only.

This chapter was developed from Chapter 46 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Solutions

1. Answer: d

Explanation: All of the anatomic communications mentioned can facilitate the spread of infection between pulp space and periodontium.

2. Answer: c

Explanation: Based on the radiographic presentation, the maxillary sinus was in close proximity for both aspects of the procedure, periodontal and endodontic.

3. Answer: c

Explanation: Considering that the lesion was primarily endodontic, the recommended sequence is to perform endodontic therapy first, then reevaluate in 2–3 months to assess potential need for periodontal therapy.

4. Answer: c

Explanation: When an endodontic lesion that is progressing coronally joins with a periodontal pocket, it is referred to as a true combined lesion.

Reference

1. Herrera, D., Retamal-Valdes, B., Alonso, B., & Feres, M. (2018). Acute periodontal lesions (periodontal abscesses and necrotizing periodontal diseases) and endo-periodontal lesions. *Journal of Periodontology*, *89*(Suppl. 1), S85–S102.

27 Plaque Control

Relevant Terminology

Terminology	Explanation
abrasive	Insoluble inorganic salt that enhances the abrasive action of toothbrushing by as much as 40 times and makes up 20%–40% of dentifrices.
chlorhexidine	A bisbiguanide with antimicrobial properties, used as a prescription-only oral rinse.
disclosing agents	Solutions or wafers that stain bacterial biofilm on the surfaces of the teeth, tongue, and gingiva. Used as an oral hygiene instruction tool.
essential-oil rinses	Contain thymol, eucalyptol, menthol, and methyl salicylate, and have plaque biofilm reduction and gingivitis reduction efficacy.
gingival col	Nonkeratinized interdental gingival surface under the contact area of two adjacent teeth that is most vulnerable to microbial presence.
plaque control record	Index of presence or absence of plaque from teeth surfaces.
subgingival irrigation	Pulsating a stream of water through a narrow nozzle to the root surfaces of difficult-to-reach sites (e.g., furcas).

Fast Facts

Plaque management	Periodontal disease therapy and prevention are based on minimizing the accumulation and removal of plaque biofilm around the periodontium via professional cleaning and self-care.
Microbial plaque and calculus	Microbial plaque growth occurs within hours and can be removed with effective oral self-care at home. If not removed, it will mineralize due to mineral transfer from saliva, and form calculus, which can only be removed by dental instrumentation.
Toothbrush	The main aid for hygiene of straight teeth surfaces. Selection should prioritize ease of use; most designs have equivalent plaque removal efficacy. Hard bristles may cause recessions and should be avoided. Oscillating and rotating powered toothbrushes may have slightly increased efficacy and patient acceptance than manual brushes, especially for children and individuals with reduced motor skills.
Dentifrices	Aid in cleaning and polishing tooth surfaces. Fluoride toothpastes are advantageous for caries control. Toothpastes with coarse abrasives should be discouraged because of the risk for hard and soft tissue damage.
Bass technique	The Bass toothbrushing technique is the preferred method for reaching the gingival sulcus with the bristles. Other toothbrushing techniques: Stillman, modified Stillman, Charters, Fones, Leonard and Scrub (please refer to Chapter 48 of Newman and Carranza's Clinical Periodontology 13th Edition textbook for details).
Interdental cleaning aids	Selection of interdental hygiene aids should be based on ease of use and patient dexterity. While dental floss is the most widely recommended tool for removing biofilm from proximal tooth surfaces, other options should be explored with each person until the ideal oral hygiene aid is identified.
Oral irrigation	Removes nonadherent bacteria and debris from the oral cavity more effectively than toothbrushes and mouth rinses (especially in cases with challenging access), and decreases inflammation.
Chemical plaque control	A large selection of antimicrobial agents are available as adjuncts to mechanical oral hygiene. While they improve plaque and/or gingivitis reduction, their long-term use must be weighed against the risk of adverse events. For example, long-term chlorhexidine use may lead to staining of the teeth, tongue, and restorations, as well as transient impairment of taste perception.

Core Knowledge

Plaque biofilm control has two important purposes in periodontal therapy and maintenance:

1. To minimize gingival inflammation
2. To prevent the recurrence or progression of periodontal diseases and caries (especially root caries in regions of exposed cementum)

Fig. 27.1 briefly reviews the plaque control methods that are currently advocated. The reader is referred to Chapter 48 of *Newman and Carranza's Clinical Periodontology* (13th ed.) for a detailed description of the various methods of plaque control.

CLINICAL CORRELATE

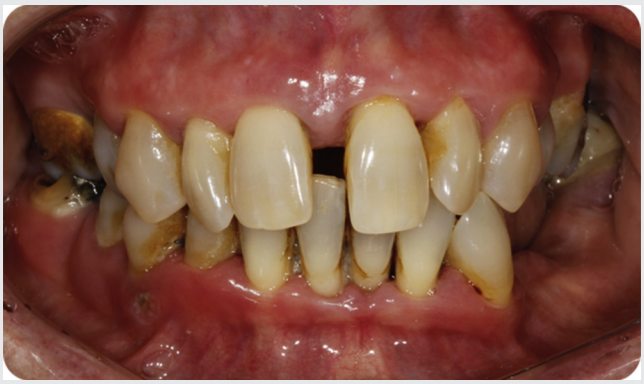
What happens when one ceases to practice oral hygiene measures for a period of time?

Daily plaque biofilm control practices result in improved periodontal and gingival health. Cessation of plaque control practices for 7–21 days results in:

- Accumulation of thick plaque on tooth surfaces
- Reddened gingiva that bleeds easily
- Shift to more virulent gram-negative flora
- Microscopic changes that are completely reversed in about 7 days when plaque control practices are resumed

CASE-BASED LEARNING EXERCISE

Scenario: A 60-year-old female presented with the chief complaint: "I haven't been to a dentist in a long time and I am sure I will need a lot of work." No medical conditions were reported, and the patient was not taking any medications. Clinical findings: generalized heavy deposits of calculus with deeper probing depths (6–8 mm) in posterior sites and generalized bleeding on probing (Fig. A).



Questions

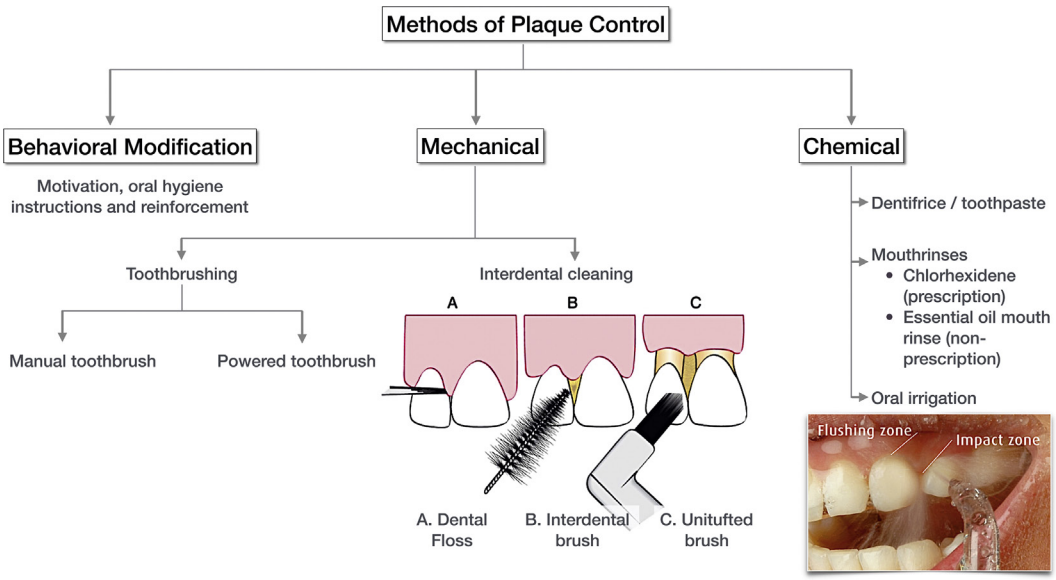
1. The most effective method of preventing periodontal disease in the general population is:
- **a.** Oral hygiene.
- **b.** Low-fat diet.
- **c.** Fluoride therapy.
- **d.** Antibiotics.

2. What is the recommended interval for reevaluation (follow-up) after nonsurgical periodontal treatment of a patient with periodontitis?
- **a.** 0–1 week
- **b.** 2–3 weeks
- **c.** 4–8 weeks
- **d.** > 2 months

3. In general, the chance of complete plaque elimination with scaling and root planing is greater with probing depths of:
- **a.** 4 mm or less.
- **b.** 6 mm or less.
- **c.** 8 mm or less.
- **d.** 10 mm or less.

4. What is the correct angle between terminal shank and tooth surface for efficient scaling using Gracey curette #11/12?
- **a.** 0–19 degrees
- **b.** 20–39 degrees
- **c.** 70–80 degrees

This chapter was developed from Chapter 48 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.



• **Fig. 27.1** Methods of Plaque Control. *Target hygiene* refers to an emphasis on plaque biofilm removal at the dentogingival junction to prevent caries and periodontal disease. The following methods are all used together in various ways to achieve optimal target hygiene.

Behavioral modification: Using aids like face mirrors and disclosing solutions (that preferentially stain plaque on teeth), a patient can be made to understand the inadequacies in their current brushing style and also taught the correct method of brushing to achieve optimal oral hygiene.

Mechanical methods: Tooth brushing: This can be performed using manual or powered toothbrushes

- Recommendations for using manual toothbrushes:
  - Soft nylon bristle toothbrushes can clean effectively and do not traumatize the gingiva or root surfaces as much as hard-bristle toothbrushes do
  - Worn-out brushes should be replaced every 3–4 months
  - If patients perceive a benefit from a particular design of toothbrush, they should use it as long as it is not too stiff and hard
- Recommendations for using powered toothbrushes:
  - Oscillating and rotating powered brushes remove plaque biofilm and reduce gingival bleeding slightly better than manual toothbrushes; patients who want to use powered toothbrushes must be encouraged to do so
  - Patients with limited manual dexterity, children, older adults, and caregivers may particularly benefit from using powered toothbrushes
- Tooth brushing techniques:
  - To be effective, brushing with either a manual or powered toothbrush requires a systematic routine with emphasis on brushing all the surfaces of the teeth
  - The method most often recommended is the *Bass technique*, which emphasizes the sulcular placement of the bristles at the critical cervical and interproximal areas of teeth.
    Inter-dental cleaning: The type of embrasure a patient presents with, dictate the selection of interdental cleaning devices. Example: single tufted brushes clean embrasures without papillae well, while dental floss is effective in cleaning embrasures with intact papillae.

Chemical methods: Dentifrices: These increase the effectiveness of brushing by including additional ingredients like:

- Fluoride and antimicrobial agents, which provide additional benefits for controlling caries and gingivitis.
- Pyrophosphates in a calculus control dentifrice, for use in patients who are at increased risk for supragingival calculus formation.
  Mouth rinses: These should be used as adjuncts to proven mechanical methods of plaque control.
- Chlorhexidine rinses (prescription only) can be used to improve plaque biofilm control during phase I therapy for patients with recurrent disease, after periodontal or oral surgery, and for caries management.
- Essential-oil rinses have fewer side effects and are available without a prescription.
  Irrigation: Gingival irrigation can be a useful adjunct for periodontal patients who have residual pockets and complex dental architecture to clean every day.
- Pulsation and pressure applied to the jet of water from the irrigation device together create a compression-decompression phase that allows both penetration of the irrigant into the sulcus or pocket and the expulsion of bacteria and debris.

(From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

Case-Based Learning Exercise

Solutions

1. Answer: a

Explanation: Oral hygiene is key in the prevention of periodontal disease. This can be acheived using multiple tools and techniques including brushing, flossing, and use of proxy brushes.

2. Answer: c

Explanation: The recommended interval for reevaluation (follow-up) after nonsurgical periodontal treatment of a patient with periodontitis is 4 to 8 weeks. For a patient with gingivitis, improvements can be observed as early as 2 weeks.

3. Answer: a

Explanation: In general, the chances of complete plaque elimination with scaling and root planing are greater in probing depths of 4 mm or less.

4. Answer: c

Explanation: 70–80 degrees is the correct angle between terminal shank and tooth surface for efficient scaling using Gracey curettes.

28 Nonsurgical Periodontal Therapy

Relevant Terminology

Terminology/Abbreviation	Explanation
curette	Fine instrument with rounded toe end, used for subgingival *scaling and root planing*
explorer	Instrument used to locate calculus deposits and caries. Critical for identifying deposits prior to *scaling and root planing* and also assessing effectiveness of removal of deposits on treatment completion.
gracey area-specific curettes	Gracey curettes differ from *universal curettes* in that the blade is not at a 90-degree angle to the lower shank, but is angled at approximately 70 degrees. This unique angulation allows the blade to be inserted in the precise position necessary for subgingival *scaling and root planing*, provided the lower shank is parallel to the long axis of the tooth surface being scaled.
implant instruments	Implant scalers and curettes are usually made of plastic composites or titanium. It is imperative that implant instruments are nonabrasive to metal implants. Conventional steel instruments can cause mechanical damage to the implant surface.
magnetostriction	Refers to a change in the dimensions of a metallic material when subjected to a magnetic field. Examples of magnetostrictive materials: iron, nickel, nickel alloys, and cobalt.
magnetostrictive and piezoelectric ultrasonic instruments	The handpiece of both types of instrument contains a transducer—a device that converts electrical current from the foot pedal into acoustic energy and high-frequency vibrations of the instrument tip for *scaling*. The conversion can be achieved by magnetostrictive or piezoelectric transducers by dimensional changes. In a magnetostrictive scaler, the transducer is a stack of nickel strips, while in piezoelectric scalers, ceramic/quartz disks act as transducers.
periodontal probe	Instrument used to locate, measure, and mark pockets, and determine their course on individual tooth surfaces
piezoelectricity	Change in dimensions of a crystalline material when subjected to an electric field. Examples of piezoelectric materials: quartz, ceramics.
power instruments	Power scalers such as sonic, magnetostrictive, and piezo instruments are mainstays of clinical practice. They can be used alone or in combination with hand instruments for removal of both plaque (biofilm) and calculus. They can make *scaling* less demanding.
scaling and root planing	Scaling is the process by which biofilm and calculus are removed from both supragingival and subgingival tooth surfaces. Root planing removes residual embedded calculus and surface irregularities to produce a smooth, hard, clean root surface.
sickle scalers	Instruments with a sharp pointed tip and cutting edges that facilitate the rapid removal of large supragingival calculus deposits
ultrasonic energy	Sound energy (acoustic energy) above the range of normal human hearing, which is 20 kHz
universal curettes	Universal curettes have cutting edges that may be inserted in most areas of the dentition by altering and adapting the finger rest, fulcrum, and hand position of the operator. The blade size and shank angle and length may vary, but the face of the blade of every universal curette is at a 90-degree angle (perpendicular) to the lower shank when seen in cross section from the tip. This positioning allows the curette to function using both cutting edges.

Fast Facts

Objectives of instrumentation	1. Disruption and removal of subgingival biofilm (plaque) 2. Reduction/removal of plaque-retentive factors (e.g., dental calculus) 3. Conservation of tooth structure 4. Creation of a biologically acceptable root surface 5. Resolution of inflammation
Parts of a typical periodontal instrument	• Working end/blade • Shank • Handle
Instrument balance	A periodontal instrument is said to be balanced if the blade is centered with the long axis of the handle.

Classification of nonsurgical periodontal instruments	• Periodontal probes
	• Explorers
	• Scaling and curettage instruments
	  • Sickle scalers, ultrasonic and sonic instruments
	  • Curettes—fine instruments used for subgingival scaling and root planing
	  • Hoes, chisels, and file scalers
	  • Ultrasonic and sonic instruments
	  • Implant instruments (made of plastic or titanium)
	• Periodontal endoscopes
	• Cleansing and polishing instruments

Periodontal Probing	• Various probe designs are available • It is recommended to keep probe selection consistent within the same patient for reproducibility • When measuring a pocket, the probe is aligned with the long axis of the tooth surface to be probed, for reliability. Angulating the probe over 30 degrees against the long axis of the tooth can lead to overestimation.

Types of curettes	• Universal curettes
	• Area-specific curettes
	  • *Gracey* curette—numbered 1 to 18
	  • *Curette For Deeper Pockets*—extended shank curette, 3 mm longer shank than Gracey curettes for probing depth >5 mm
	  • *Mini Five* curette—half the blade length of Gracey/After Five curette, allows for use in deep, narrow pockets
	  • *Micro Mini Five* curette—20% thinner blade than Mini Five curette
	  • *Gracey* curette—50% shorter blade than Gracey curette, blade curves upward
	  • *Langer* curette—combination of Gracey curette shank and Universal curette blade designs
	  • *Quetin* curette—for furcations

Determinants of successful instrumentation	• Ideal instrument selection, proper patient and operator positioning • Good visibility and clean field with frequent flushing • Condition and sharpness of instruments • Proper instrument grasp and finger rest • Proper adaptation, correct tooth-blade relationship, correct amount of lateral pressure and working stroke
Mechanism of action of ultrasonic debridement	Involves: 1. **Mechanical** disruption of deposits by vibratory action of oscillating instrument tip; depends on frequency (number of times tip oscillates) and amplitude (distance traveled by oscillating tip in motion) 2. **Irrigation** and flushing out of debris by **acoustic streaming** (unidirectional flow of water out of handpiece) and **acoustic turbulence** (swirling effect caused by moving tip within streaming water) 3. **Cavitational effect**—acoustic turbulence creates bubbles that "implode" or collapse into themselves, leaving cavities within the liquid and creating shock wave ripples that disrupt microflora

Core Knowledge

Introduction

A great deal of periodontal treatment time is spent in nonsurgical elimination of plaque and its contributory factors, with the aim of controlling inflammation. It is important for a practicing dentist to be aware of the distinctions between individual treatment modalities (e.g., scaling and root planing), and the nuances surrounding the objectives of each one. The time has come to interpret the paradigm shifts in our understanding of

periodontal disease etiopathogenesis in the context of how best to treat and manage our patients (Table 28.1).

Objectives of Instrumentation

The host response against subgingival plaque is sometimes a frustrated attempt to eliminate the effects of the pathologic biofilm because:

- The biofilm within the gingival sulcus effectively shields pathogens from immune clearance by host defense mechanisms; it also affects the ability of antibiotics to destroy them.
- At the same time, plaque mass is held in close enough proximity to host tissues within pockets to trigger host inflammatory responses.

CLINICAL CORRELATE

Considering the current paradigm shifts in our understanding of periodontal disease etiopathogenesis, what is the main objective of initial periodontal therapy? How can it best be achieved?

Current understanding of periodontal pathogenesis indicates that "control of inflammation" is most vital for treating periodontal disease. This depends on the cornerstones of periodontal therapy, namely:

1. Plaque control by the patient
2. Instrumentation aimed at biofilm disruption by the clinician

For these reasons, the physical removal/disruption of plaque biofilm by some form of instrumentation still remains the starting point of periodontal therapy.

While the earlier prevailing belief was that the removal of every speck of calculus, endotoxin, and contaminated/necrotic cementum achieved the primary therapeutic endpoints of periodontal instrumentation, this is no longer the case. The objectives of modern instrumentation (Fig. 28.1), based on a deeper understanding of the etiology of periodontal disease, are more realistic and include[1]:

1. Disruption and removal of subgingival biofilm
2. Reduction/removal of plaque-retentive factors (e.g., dental calculus, restoration overhangs)
3. Conservation of tooth structure
4. Creation of a biologically acceptable root surface
5. Resolution of inflammation

Nonsurgical Treatment Modalities

Nonsurgical therapy can be both "preventive" and "therapeutic" in nature; hence it is one of the initial treatment phases (Phase 1) undertaken in the management of the periodontal patient. Practically speaking, one could think of this phase in terms of three categories based on the existing clinical condition (Table 28.2):

- Primary prevention—in clinically healthy periodontium
- Secondary prevention—in cases of clinical gingivitis
- Initial/Causal therapy—in cases showing true pocketing and attachment loss.

The various nonsurgical treatment modalities employed in the past and present in Phase 1 (nonsurgical) periodontal therapy include:

1. **Scaling**—aims at:
   - disruption and removal of plaque biofilm
   - reduction/removal of dental calculus
   - resolution of inflammation
2. **Root planing**—aims at:
   - removal of root-adherent calculus and cementum (and hence endotoxins)
   - creation of smooth, hard root surface
3. **Curettage**—aims at conversion of a chronic lesion into an acute surgical wound to promote better healing by:
   - marginal gingiva shrinkage
   - epithelial adhesion to tooth surface
4. **Root surface debridement (RSD) or root detoxification**—aims at:
   - disruption and removal of subgingival biofilm
   - reduction/removal of plaque-retentive factors (e.g., dental calculus)
   - conservation of tooth structure
   - creation of a biologically acceptable root surface
   - resolution of inflammation

Scaling and root planing (SRP) are often combined and instrumentation is performed in the same appointment under local anesthesia. Fig. 28.2 depicts the relevance of the various treatment options for meeting the objectives of nonsurgical periodontal therapy.

Classification of Periodontal Instruments

Periodontal instruments may be classified based on their purpose as:

1. **Periodontal probes**—used to locate and measure pocket depths and determine pocket configurations
2. **Explorers**—used to detect calculus and caries, and to check smoothness of tooth surface after instrumentation
3. **Periodontal therapy instruments**—used for scaling and debridement of tooth and root surfaces
4. **Endoscopes**—used to visualize deep into pockets and furcations to detect deposits
5. **Cleansing and polishing instruments**—used to clean and polish tooth surfaces, implants, and restorations

Fig. 28.3 discusses in detail the various instruments used for nonsurgical periodontal therapy. All of them have the common purpose of scaling and debridement of tooth/implant surfaces and periodontal pockets. The reader is referred to *Newman and Carranza's Clinical Periodontology* (13th ed.) for detailed descriptions of all instruments.

General Principles of Instrumentation

Certain fundamental prerequisites for effective instrumentation are common to all periodontal instruments:

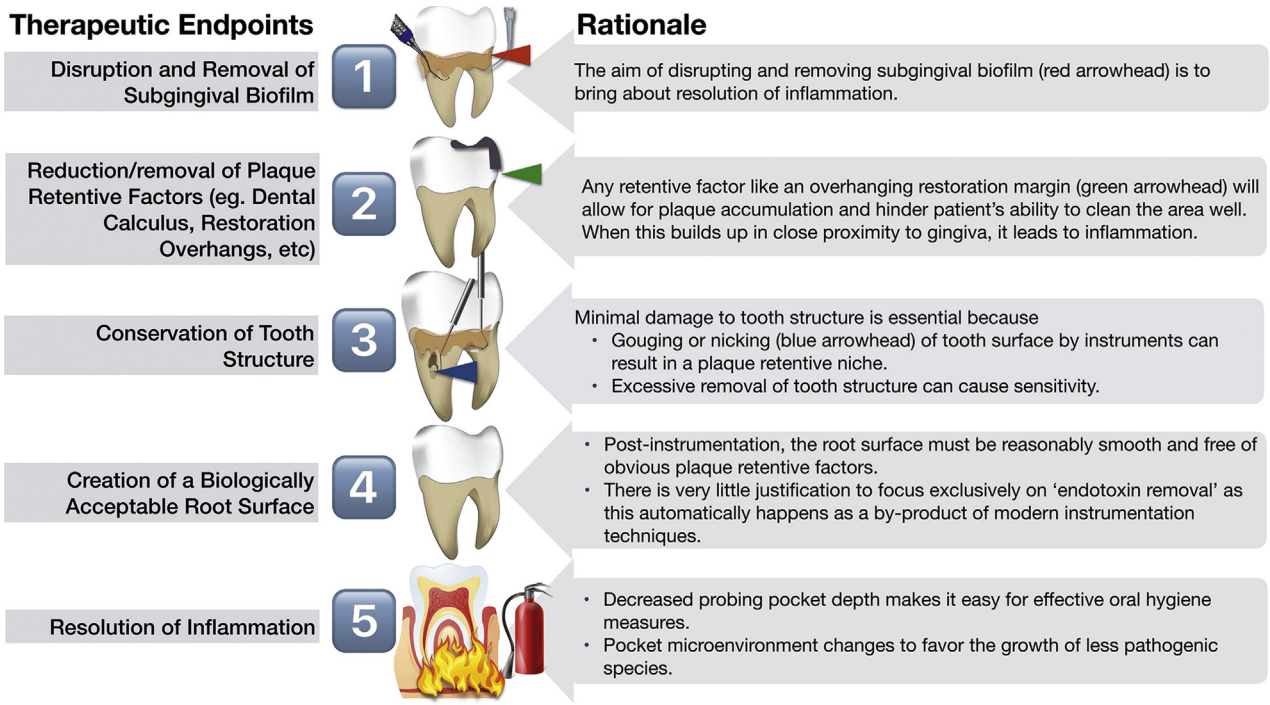
1. **Accessibility**—depends on positioning of both patient and operator as well as proper retraction.

(Text continues on p. 6)

TABLE 28.1 Paradigm Shifts in Periodontal Etiopathogenesis: Impact on Nonsurgical Treatment Modalities[4,5]

Traditional Concepts	Current Concepts
Dental calculus and endotoxins are the main drivers of periodontal disease.	Dental plaque biofilm is the driver of periodontal inflammation.
Endotoxin (LPS from cell wall of gram-negative bacteria) is **firmly bound** to the outer layers of cementum under subgingival plaque biofilm. This LPS-containing cementum is considered "infected," "necrotic," or "contaminated."	Endotoxin/LPS is **loosely associated** with cementum.
Removal of calculus and outer layer of cementum by root planing eliminates endotoxins and is responsible for achieving periodontal health.	It is now known that endotoxins can be removed by gentler techniques (e.g., washing with water, polishing root surfaces, brushing). Hence the systematic and extensive removal of cementum by root planing as a procedure aimed at removal of endotoxins is now somewhat controversial, although still accepted and practiced.
Periodontal disease results from direct tissue damage caused by bacterial toxins and noxious products.	Periodontal disease is initiated by plaque bacteria, but tissue damage is mainly the result of dysregulated and disproportional host inflammatory response triggered by specific microbes in the subgingival biofilm.

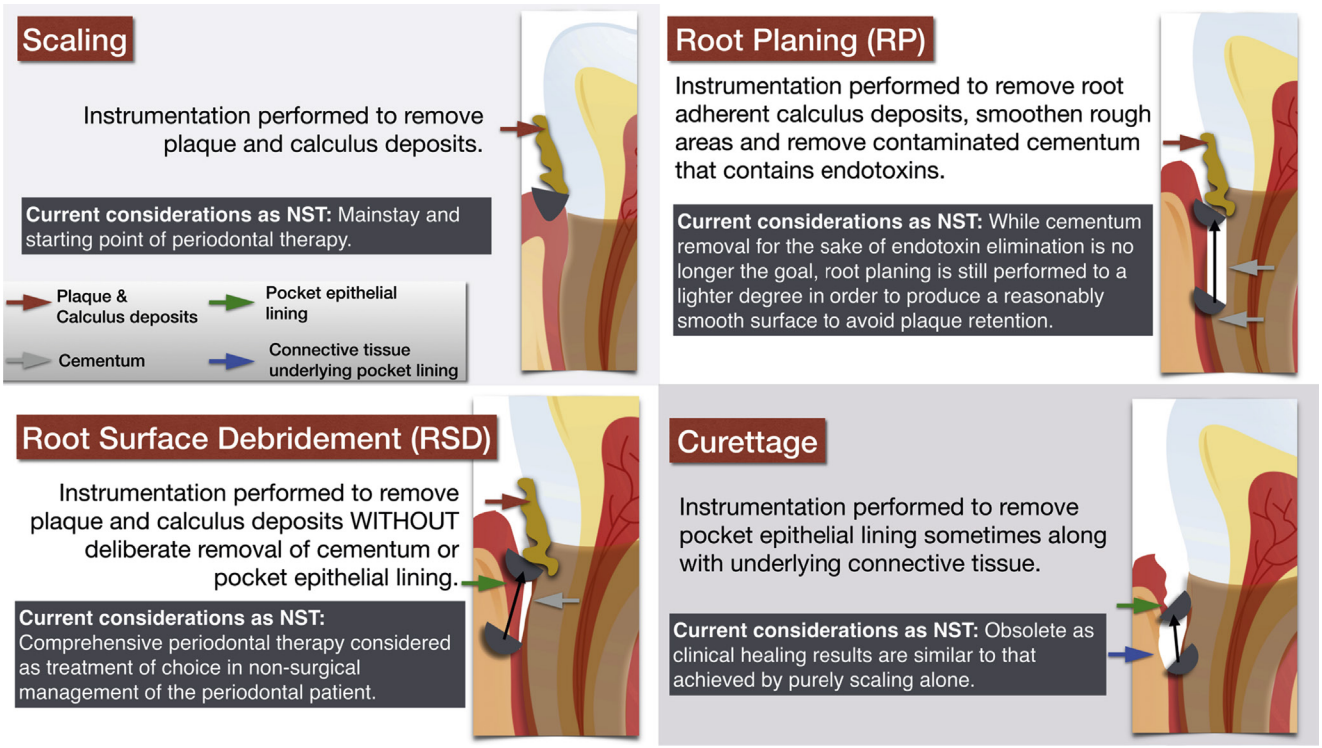
LPS, lipopolysaccharide.



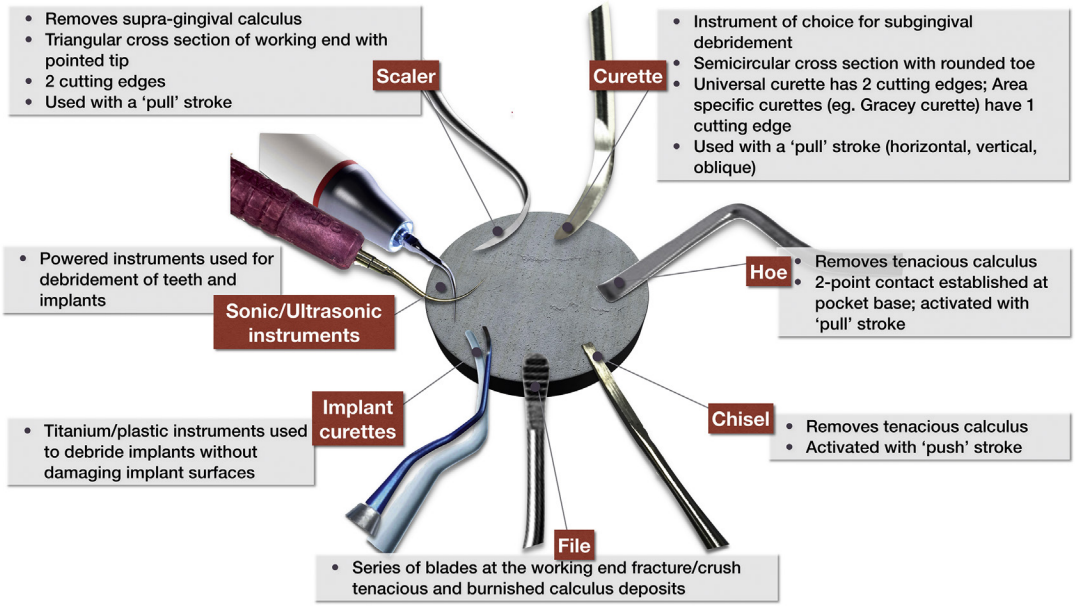
• **Fig 28.1** Objectives of Modern Periodontal Instrumentation. The therapeutic objectives of modern periodontal instrumentation mainly revolve around producing a reduction in periodontal inflammation by removal of plaque biofilm and plaque-retentive factors in order to enhance a healthy biologic tooth-periodontium interface. The rationale behind each of the main therapeutic endpoints is discussed in the figure.[1]

TABLE 28.2 Nonsurgical Periodontal Therapies[6]

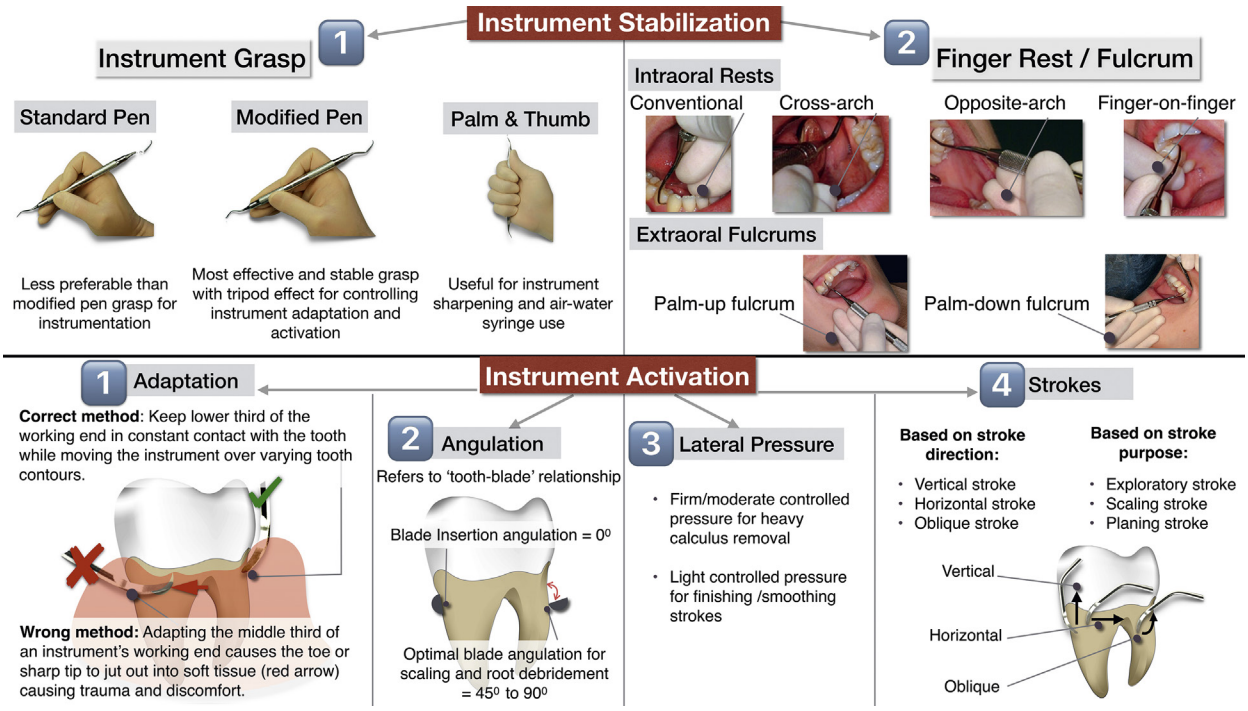
	Preventive		Therapeutic
	Primary Prevention	Secondary Prevention	Initial/Causal Therapy
Clinical Presentation	Health	Gingivitis	Periodontitis
Therapeutic Strategies	Toothbrushing and other oral hygiene measures (prescribed as appropriate for local conditions, e.g., diastema, crowding) Regular scaling and polishing every 6 months. Achieved usually by hand or ultrasonic instruments	Professional removal of plaque biofilm by scaling and irrigation (in addition to debridement and oral hygiene strategies discussed under primary prevention)	Closed subgingival treatments for root surface debridement and removal of plaque-retentive factors (e.g., subgingival calculus, overhanging restorative margins), in addition to debridement and oral hygiene strategies discussed under primary and secondary prevention



• **Fig 28.2** Nonsurgical Periodontal Therapy: Current Considerations. Subgingival scaling and root planing (SRP) are classically undertaken as two components of the same nonsurgical therapeutic appointment. Root planing and curettage—in their traditional forms, as separate procedures to remove infected cementum and pocket lining, respectively—are no longer the primary objectives of instrumentation. The current opinion is that scaling (the starting point of therapy) and root surface debridement (comprehensive periodontal therapy without intentional removal of cementum for the purpose of endotoxin removal) using manual or ultrasonic instruments, lasers, and so on are the more effective forms of nonsurgical periodontal therapy.[2,3] NST, nonsurgical periodontal therapy.



• **Fig 28.3** Instruments Used for Nonsurgical Periodontal Therapy. This figure depicts the different instruments used for nonsurgical therapy. They may be classified as (1) manual instruments—scalers, curettes, hoes, chisels, files, implant scalers/curettes—and (2) powered instruments—sonic/ultrasonic scalers.
Note: Instrument tips are not depicted to scale.
From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.



• **Fig 28.4** Instrument Stabilization and Activation. Instrument stabilization depends on **(a) Instrument grasp:** modified pen grasp is optimal for controlled instrumentation, while the palm and thumb grasp is least favored; and **(b) Finger rests (intraoral) or Fulcrums (extraoral**). Intraoral finger rests can be established adjacent to the working area (conventional), on the contralateral side of the same arch (cross-arch), on the opposite arch, or on the index finger/thumb of the nonoperating hand for improved stability. Extraoral fulcrums use the back (palm-up) or front (palm-down) surfaces of fingers of the operating hand on the face for greatest stability. Instrument activation depends on:

(a) **Adaptation—**only the lower third of the working end, the last few millimeters from the tip/toe of the blade, must be always in contact with the tooth surface while instrumenting. This prevents the sharp ends from traumatizing soft tissue, especially in tight or narrow pockets.

(b) **Angulation—**the blade must be flush with the tooth surface while inserting into the gingival sulcus; that is, the tooth-blade angle must be 0 degrees, and opened out to between 45 and 90 degrees for instrumentation. If the tooth-blade angle is greater than 90 degrees (too obtuse) or less than 45 degrees (too acute), ineffective calculus removal results.

(c) **Lateral pressure—**the pressure applied by the instrument on the tooth surface depends on the nature of the deposits (tenacious calculus/plaque biofilm/loose debris) and the intended purpose of the stroke (scaling or smoothing of tooth surface).

(d) **Strokes—**"push" or "pull" strokes in the vertical, oblique, or horizontal directions are used for exploration, scaling, and planing. The "exploratory" stroke is a light "feeling" stroke used to evaluate pocket dimensions or to detect calculus/surface irregularities. The "scaling" stroke is a short, powerful pull stroke used to remove calculus. The "planing" stroke is a light to moderate pull stroke used for final smoothening and planing of root surface.

Note: Diagrammatic representations are not drawn to scale but are aimed at concept understanding.

From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

2. **Visibility**—depends on proper retraction of tongue, cheeks, and so on, and good illumination of field of operation (either directly from dental light, fiber-optic instrument or operator's headgear, or indirect reflected light from mouth mirror).
3. **Sharp instruments**—allow for efficient instrumentation and effective calculus removal; dull instruments cause unnecessary trauma due to the excessive force applied to compensate for their ineffectiveness.
4. **Clean field of operation**—depends on proper suctioning of saliva, blood, and debris by saliva ejectors or wiping/blotting with gauze squares.
5. **Instrument stabilization**—depends on instrument grasp and finger rest (which should provide a firm "fulcrum" that prevents injury/laceration of gingiva by poorly controlled instruments). The index finger/thumb of the nonoperating hand is often used on the instrument handle/shank to add reinforcement to rests/fulcrum for better control.
6. **Instrument activation**—depends on instrument working end adaptation, angulation of shank and blade, lateral pressure applied during instrumentation, and push/pull strokes that can be used in different directions with different objectives in mind.

Fig. 28.4 elaborates on the various types of instrument grasps, finger rests/fulcrums, and instrument strokes that allow for effective instrumentation by the operator.

Manual and Ultrasonic Instruments in Nonsurgical Periodontal Therapy

In most cases, hand and ultrasonic instruments are used in conjunction with each other to bring about effective periodontal debridement. The extent to which one or the other is used may depend on individual preference, expertise, and operator training. The reader is referred to *Newman and Carranza's Clinical Periodontology* (13th ed.) for detailed discussion on various instruments, their design, purposes, and handling.

The differences in the handling of hand instruments and power-driven instruments must be well understood; using them both in the same way might not be effective and may traumatize the periodontium and/or damage the instruments. Ultrasonic scalers should not be used as "hand scalers with power." The principles governing correct usage are different for both types of instruments and the nuances are discussed briefly in Fig. 28.5.

CLINICAL CORRELATE

What is the major design difference between manual and ultrasonic instruments that an operator must keep in mind during instrumentation?

Manual scaling instruments and technique are designed to break the bond between tooth and calculus at the tooth–deposit interface. They work to debride by manually stroking the working end on the tooth surface, starting from the apical edge of the deposit. In contrast, ultrasonic instruments, although they sometimes work at the tooth–deposit interface, mainly remove calculus deposits by ablation/attrition. They remove the plaque/calculus deposits from the outside surface using vibratory and biophysical forces (acoustic streaming, turbulence, and cavitation). Thus the overlapping strokes used with ultrasonic instruments do not require the firm controlled pressure commonly needed with hand instruments for debridement; instead, lighter pressure and grasps work more effectively.

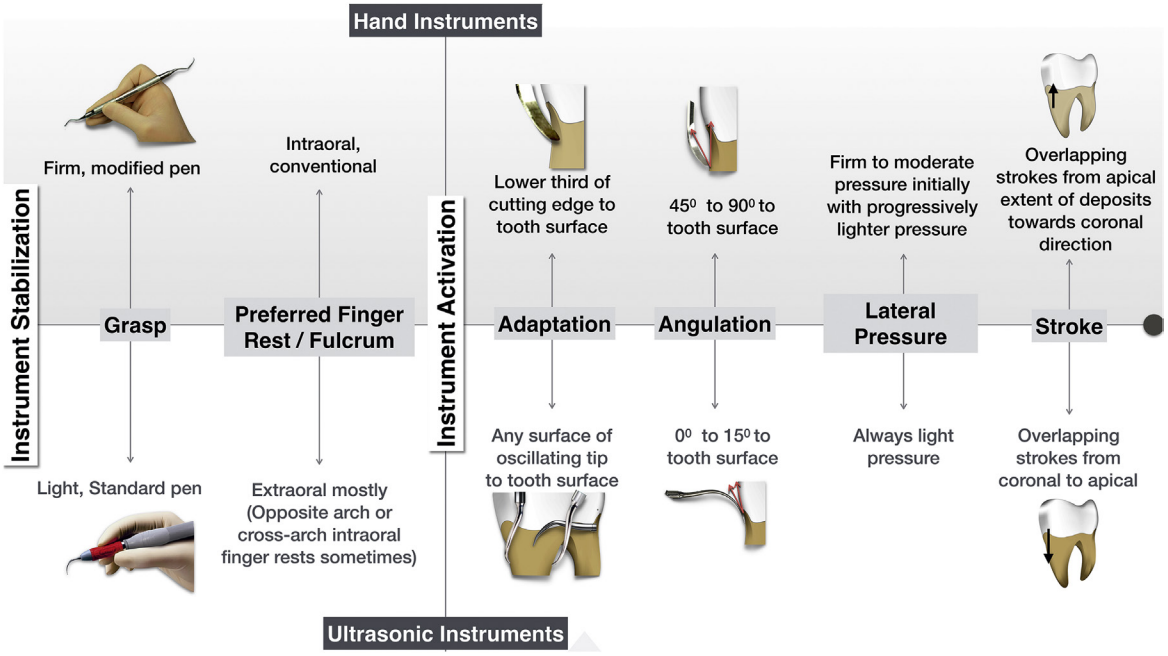
Limitations of Nonsurgical Periodontal Therapy

While a nonsurgical approach to periodontal therapy is a conservative and effective way to address the causes of periodontal disease, certain limitations exist to this approach:

- **Restricted vision of, and access to, subgingival deposits in deep pockets**—the operator needs to depend mostly on tactile sensations to assess whether all deposits have been effectively removed from the root surface, and this can be somewhat unreliable.
- **Lesser reduction in pocket depth than with surgical therapy when probing depth > 6 mm**—greater reductions can be obtained by surgical approaches than nonsurgical ones in the case of pockets with probing depths greater than 6 mm.
- **Inability to perform regenerative procedures**—regenerative procedures (e.g., guided tissue regeneration) cannot be performed using nonsurgical approaches; most of the gain in clinical attachment in nonsurgical therapy is generally attributed to healing by repair/reattachment rather than "new attachment" formation.
- **Side effects of recession and sensitivity**—soft tissue recession and dentin hypersensitivity may result after nonsurgical periodontal therapy, though perhaps to a much lesser degree than with surgical periodontal therapy.

Conclusions

There are several accepted nonsurgical treatment modalities in practice today, with some confusion surrounding their use and nomenclature. A clinician's choice of periodontal

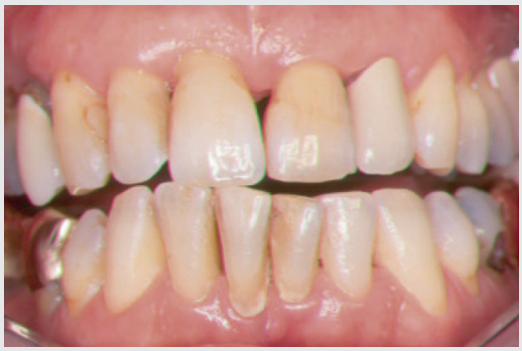
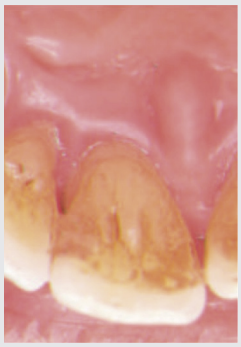
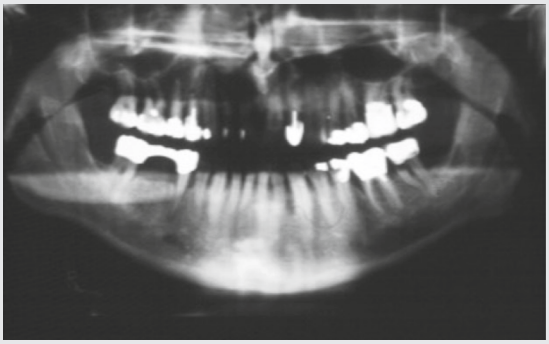


• **Fig 28.5** Principles of Instrument Stabilization and Activation. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

therapy in a given scenario should be backed by sound justifications for its use, whether as the main procedure or as an adjunct to others. The success of periodontal therapy hinges on the healing response within the periodontium, not merely on whether complete calculus removal has been achieved. This chapter with its focus on therapeutic relevance discusses the rationale behind various nonsurgical treatment modalities in today's practice based on current understanding of this disease.

CASE-BASED LEARNING EXERCISE

Scenario: The patient was a 55-year-old mechanical engineer, diagnosed with diabetes 5 years ago after his dentist noticed severe periodontitis with multiple abscesses, which were treated at that time. He reported taking 20 units of insulin every morning and was also taking glyburide, atorvastatin (Lipitor), and losartan. The patient was overweight, had hypertension, and reported that his fasting blood sugar was sometimes over 200, even with medication. His chief complaint in the clinic was pain in tooth #5 and discomfort on chewing with tooth #18. He had not had any periodontal treatment since he underwent scaling 5 years earlier and was requesting a "recall" scaling. Findings: Several molars had furcation invasions with severe bone loss and suppuration; generalized bleeding on probing but no signs of active periodontal abscesses. Poor oral hygiene, generalized severe periodontitis (generalized periodontitis, stage III, grade B) with probing depths of 4 to 9 mm, and moderate to heavy calculus throughout the mouth.

Questions

1. For this patient, which of the following curettes would be the most effective for scaling a thin sheet of tenacious calculus in the 6-mm pocket on the palatal surface of the maxillary right central incisor (#8), which has very firm, tight tissue?
- **a.** Gracey #7-8, Gracey #13-14
- **b.** Gracey #11-12, Gracey #5-6, Gracey #7-8
- **c.** Gracey #13-14, Mini Five #13-14, Micro Mini #13-14
- **d.** Gracey Curette Sub-0, Mini Five #5-6, or Micro Mini #1-2

2. In which of the following areas would you select an extraoral fulcrum for use with Gracey curettes?
- **a.** Labial of maxillary anteriors with tight tissue
- **b.** Lingual of mandibular anteriors with heavy calculus
- **c.** Buccal of mandibular molars with furcations
- **d.** Mesial of maxillary molars with deep pockets

3. The maximum width of the active stroke of any ultrasonic tip when it is properly adapted to the tooth or root surface is:
- **a.** 1–2 mm
- **b.** 2–4 mm
- **c.** 4–6 mm
- **d.** 6–8 mm

4. As an alternative to string floss, the water flosser has been found to be _____ at reducing plaque and bleeding:
- **a.** As effective
- **b.** Less effective
- **c.** More effective

5. When re-evaluating the results of scaling and root planing after 4 weeks, the best indication of success is:
- **a.** Reduction of pocket depth.
- **b.** Lack of bleeding on probing.
- **c.** Root smoothness.
- **d.** Absence of plaque.

This chapter was developed from Chapters 50 and 51 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Solutions

1. Answer: d

Explanation: All three of the mini-bladed Gracey curette designs listed in (d) are better choices for scaling tenacious calculus on a deep palatal root surface with tight tissue than the others. All the other choices include standard Gracey curettes, which would be too large for use on this palatal surface.

2. Answer: d

Explanation: An extraoral fulcrum would work best for the mesial surfaces of the maxillary molars. Intraoral fulcrums would work best for the other areas listed, and these areas should be scaled with mini-bladed Gracey curettes.

3. Answer: a

Explanation: Only 1–2 mm of any ultrasonic tip or hand instrument will adapt to the tooth during any stroke because of the curvature of the tooth or root surface. This is true regardless of the design or length of the ultrasonic tip.

4. Answer: c

Explanation: Emerging studies have demonstrated that the water flosser is more effective at reducing bleeding and gingivitis than string floss is. Many factors likely contribute to this outcome. For many people, the water flosser is easier to use than string floss, and it has been shown to reduce bacteria up to 6 mm; this may benefit patients with pockets or hard-to-access areas.

5. Answer: b

Explanation: Lack of bleeding on probing is the most reliable indication of success after scaling and root planing. Presence of bleeding on probing is almost always a sign that residual subgingival calculus remains on the root surface, especially in areas of pocket depth. Reduction of pocket depth, root smoothness, and absence of plaque are all positive outcomes, but they are not significant if bleeding on probing persists.

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29 Antibiotics and Host Modulation for Periodontal Diseases

Relevant Terminology

Terminology/Abbreviation	Explanation
amoxicillin	Semisynthetic penicillin with an extended anti-infective spectrum that includes gram-positive and gram-negative bacteria
bactericidal agent	Pharmacologic agent that actually kills bacteria
bacteriostatic agent	Pharmacologic agent that inhibits the growth of bacteria
combination drug therapy	Refers to administration of many pills each containing one drug or single pill that contains several drugs combined together; may include serial administration (drugs not given at the same time, but one after the other) or parallel administration (drugs given at the same time).
disclosing agents	Solutions or wafers that stain bacterial biofilm on the surfaces of the teeth, tongue, and gingiva. Used often as patient education and oral hygiene motivation tool.
essential-oil rinses	Contain thymol, eucalyptol, menthol, and methyl salicylate; efficacy in the reduction of plaque biofilm and gingivitis
host modulation	Treatment intended to alter host response to pathogenic stimuli A good example is sub-antimicrobial-dose doxycycline (SDD). At the employed dose, SDD inhibits matrix metalloproteinases (enzymes involved in collagen degradation) and thereby modulates the host response to microbial infection.
metronidazole	A nitroimidazole with antiprotozoal efficacy. Often used to treat anaerobic bacteria in combination with other antibiotics.
nonsteroidal anti-inflammatory drugs (NSAIDs)	Drugs that inhibit prostaglandin synthesis via cyclooxygenase inhibition, thus conferring an anti-inflammatory effect

Fast Facts

Antibiotics against periodontal biofilms	Because oral biofilms are highly resilient and protect bacteria against antibiotics, very high antibiotic concentrations are necessary to have any effect. Therefore mechanical removal of local factors around teeth surfaces is essential to disrupt the biofilm; antibiotics are employed mainly as treatment adjuncts, either systemically or locally.
Adverse effects of commonly presecribed antibiotics	Amoxicillin: anaphylactic reaction, gastrointestinal upsets Metronidazole: severe cramps, nausea, and vomiting when alcohol is ingested (disulfiram-like effect) Clindamycin: pseudomembranous colitis Azithromycin: can alter the electrical activity of the heart, which may lead to a potentially fatal heart rhythm known as prolonged QT interval

Continued

Fast Facts—cont'd

Full mouth disinfection protocol	It involves: 1. Removal of all plaque and calculus completed in two appointments within a 24-hour period 2. tongue brushing with 1% chlorhexidine gel 3. Pocket-irrigation with a chlorhexidine solution (1%)
Extracrevicular reservoirs	A number of periodontopathogenic bacteria (e.g., *Aggregatibacter actinomycetemcomitans*) have the ability to survive within periodontal tissues, which enables them to persist after mechanical plaque removal, thus requiring adjunctive antibiotic usage
Resistance	In addition to person-related adverse events around the use of antibiotics (e.g., diarrhea), a major factor that necessitates their judicious use is the possibility of developing resistant bacterial strains. Because bacterial cross-talk through gene transfer and various other mechanisms is widespread, the overuse or misuse of antibiotics can confer region-specific resistance not only to periodontal infection but to various other systemic infections.
Antibiotic selection	Selection of antibiotic use for periodontitis is based on various factors, including the specific microbiota composition, bioavailability of specific drugs in the gingiva, adverse events, medical history, and cost. Ideally, selection should be based on sensitivity testing. While tetracyclines had various advantages for use in periodontitis, their use has been limited because of multiple resistant strains. Currently, the combination of amoxicillin (with or without clavulanate) and metronidazole is the most commonly utilized systemic antibiotic regimen against periodontal bacteria.
Interactions	Because bactericidal antibiotics are effective against actively growing bacteria, concurrent use of bacteriostatic antibiotics (which inhibit bacterial growth) should be avoided. Examples of antibiotics that should be given serially and NOT in combination are Amoxicillin(bactericidal) & Tetracycline(bacteriostatic).
Antibiotics for acute conditions	Antibiotic use is not recommended for acute periodontal conditions (e.g., necrotizing gingivitis) unless there are signs and symptoms of systemic involvement such as fever, swollen lymph nodes, or malaise
Locally delivered antimicrobials	An array of formulations, including chips and injectable gels, are available for local delivery of antimicrobials and controlled release of medications within the periodontal sulcus. Their advantage is targeted delivery that bypasses adverse events associated with oral/systemic administration.
Chemical plaque control	A large selection of antimicrobial agents are available as adjuncts to mechanical oral hygiene. While they aid in plaque and/or gingivitis reduction, their long-term use must be weighed against adverse events. For example, extended use of chlorhexidine as a mouthrinse may lead to staining of the teeth, tongue, and restorations, as well as transient impairment of taste perception.

Core Knowledge

Systemic Antibiotics in Periodontics

The nonsurgical phase of periodontal therapy typically involves mechanical therapy aimed at the removal of dental plaque and other deposits from the root surface and gingiva-tooth interface in order to reduce the microbial burden. Since dental plaque matrix provides a protective environment for pathogens against antimicrobial agents, disruption of plaque during mechanical therapy, combined with optimal supragingival plaque control by the patient, will enhance microbial susceptibility to antibiotics. In a select group of patients with periodontitis, adjunctive use of systemic antibiotics was shown to improve clinical outcomes of mechanical therapy.[1] A treatment decision tree employed to assess the need for antibiotic use in periodontics is shown in Fig. 29.1.

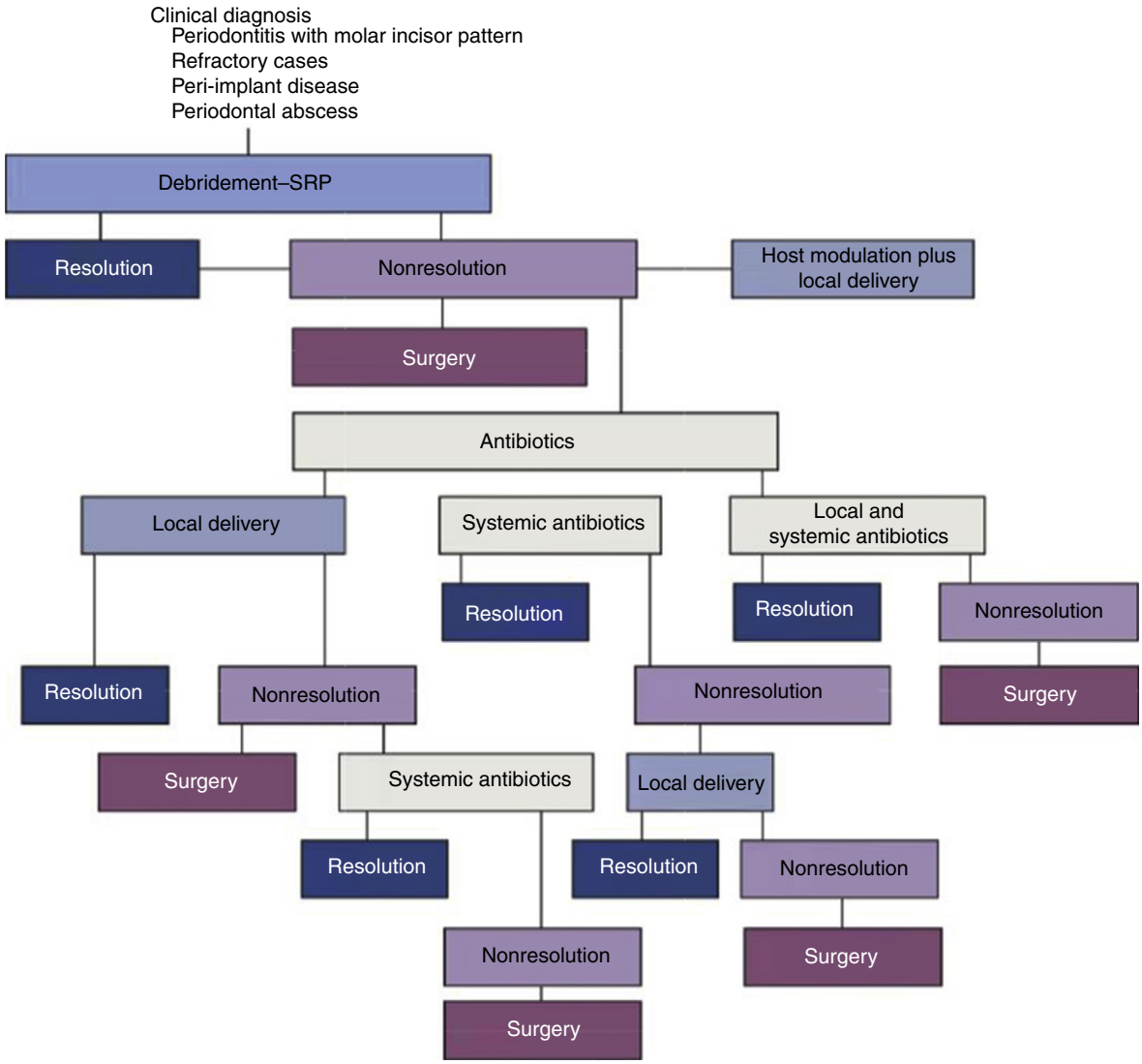
CLINICAL CORRELATE

What are the characteristics of periodontitis patients who are believed to benefit from the use of systemic antibiotics?

Periodontitis patients with particular profiles are believed to benefit from adjunctive systemic antibiotic therapy:[1]

- Patients with higher levels of periodontal pathogens (e.g., *Porphyromonas gingivalis*) in their subgingival biofilm
- Patients with rapid progression of disease (continuous loss of clinical attachment) in spite of adequate scaling and root planing
- Patients presenting with severe periodontitis

Antibiotics used to treat periodontal diseases and their major features are listed in Tables 29.1 and 29.2 and



• **Fig. 29.1** A Decision Tree for the Selection and Timing of Antibiotic Therapy in Periodontics. *SRP*, scaling and root planing.
From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

indications for the use of systemic antibiotics in different periodontal conditions are listed in Table 29.3. Table 29.2 highlights those antibiotics that are available in locally deliverable formulations.

Locally Delivered Controlled-Release Antibiotics in Periodontics

In this approach, instead of patients taking the medications orally (as in systemic delivery), the antibiotic is injected directly into the pockets. The active ingredient then gets released in a controlled and sustained fashion to deliver an effective dose in the local environment (see Table 29.2 for details on available LDD systems). This offers the following advantages to local delivery over systemic administration of antibiotics:

1. Capacity to deliver higher concentrations of the medication locally
2. Helps to overcome systemic, off-target adverse effects and hence associated with better compliance
3. Reduces the risk of promoting microbial resistance associated with systemic use of antibiotics

After scaling and root planing, the locally delivered antibiotics are typically injected into the periodontal pocket at the site of interest (Fig. 29.2). Potential indications for the use of locally delivered antibiotics include (a) as an adjunctive therapy (along with nonsurgical or surgical pocket reduction therapy) and (b) for management of sites with peri-implantitis.

Host Modulation

Though periodontal disease has infectious etiology, the tissue destruction involved in disease outcomes (e.g., clinical attachment loss) is host-mediated. Therefore both pathogens and destructive host responses are involved in the initiation and progression of periodontitis. The concept of host modulation relies on the idea that successful management of periodontal disease may require an approach that integrates therapies to address

TABLE 29.1 Antibiotics Used to Treat Periodontal Diseases

Category	Agent	Major Features	Available for Local Delivery* (Present/Past)
Penicillin	Amoxicillin	Extended spectrum of antimicrobial activity; excellent oral absorption; used systemically	
	Augmentin (amoxicillin + clavulanate potassium)	Effective against penicillinase-producing microorganisms; used systemically	
Tetracyclines	Minocycline	Effective against a broad spectrum of microorganisms; used systemically and applied locally (subgingivally)	Yes
	Doxycycline		Yes
	Tetracycline	Effective against a broad spectrum of microorganisms; used systemically and applied locally (subgingivally)	Yes
		Chemotherapeutically used in sub-antimicrobial doses for host modulation (Periostat)	
		Effective against a broad spectrum of microorganisms	
Quinolone	Ciprofloxacin	Effective against gram-negative rods; promotes health-associated microflora	
Macrolide	Azithromycin	Concentrates at sites of inflammation; used systemically	
Lincomycin derivative	Clindamycin	Used in penicillin-allergic patients; effective against anaerobic bacteria; used systemically	
Nitroimidazole	Metronidazole	Effective against anaerobic bacteria; used systemically and applied locally (subgingivally) as gel	Yes

**Chlorhexidine, an antiseptic, is also available in local delivery format.*
Adapted from Table 52.1 in Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). Newman and Carranza's Clinical Periodontology (13th ed.). Philadelphia: Elsevier.

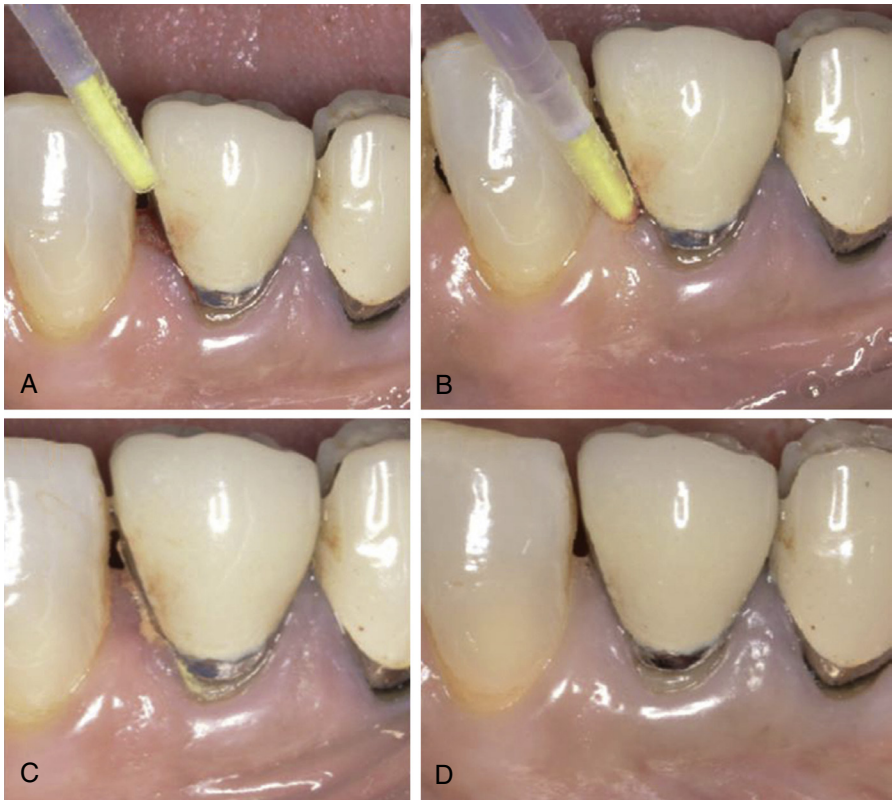
TABLE 29.2 Locally delivered antibiotics used in the management of periodontitis

	Minocycline	Doxycycline	Chlorhexidine
Commercial name	Arestin	Atridox	Periochip
Carrier	Polymeric microspheres	Biodegradable polymer	Degradable gelatin chip
Drug concentration	2% minocycline in bioresorbable microspheres	10% doxycycline (a gel system used with syringe)	2.5mg of chlorhexidine is incorporated in a gelatin matrix
Action against microbes	Bacteriostatic	Bacteriostatic	Bacteriostatic/bactericidal

GCF, gingival crevicular fluid
Note: An ethylene or vinyl acetate copolymer fiber containing the antibiotic tetracycline (actisite), was the first product introduced into the U.S. market in the early 1990s and was the prototypic system. The tetracycline fiber is no longer commercially available in the United States.

both the pathogens and the host response. In high-risk patients, integrating host modulation therapy with mechanical therapy can contribute to favorable clinical outcomes. Table 29.4 lists some systemically and locally administered host modulation agents that have been assessed for their efficacy in periodontal research. As a host modulation therapeutic agent, sub-antimicrobial-dose doxycycline is the only systemically administered agent currently approved for use as an adjunct to scaling and root planing for treating periodontitis.

Clinical Use Locally Delivered, Controlled-Release Antimicrobials



• **Fig. 29.2** Clinical Use of Locally Delivered, Controlled-Release Antimicrobials. (A and B) Local administration of antibiotic using a syringe. (C) Immediately after application. (D). 1 week after application.
From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

TABLE 29.3 Clinical Indications of Systemic Antimicrobial Agents

Disease	Systemic Antimicrobial Agents
Gingival diseases	Antibiotic use not recommended
Necrotizing gingivitis	Antibiotic use not recommended unless there are systemic complications (e.g., fever, swollen lymph nodes)
Periodontitis	Limited benefit; antibiotic use not recommended
Periodontitis with molar incisor pattern	Antibiotic use recommended; for greatest benefit, therapeutic levels of antibiotics should be achieved by the time scaling and root planing are completed (all debridement should be completed within a week); the optimal antibiotic type, dose, frequency, and duration have not been identified
Necrotizing periodontitis	Antibiotic use dependent on the systemic condition of the patient
Periodontitis as a manifestation of systemic disease	Antibiotic use dependent on the systemic condition of the patient
Periodontal abscess	Antibiotic use not recommended

Adapted from Table 52.3 in Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). Newman and Carranza's Clinical Periodontology (13th ed.). Philadelphia: Elsevier.

TABLE 29.4 Use of Host Modulation Agents in Periodontics

Route of Administration	Class of Medication	Examples
Systemically administered	Nonsteroidal anti-inflammatory drugs Bisphosphonates Sub-antimicrobial-dose doxycycline	Indomethacin, naproxen, and flurbiprofen Alendronate Doxycycline
Locally administered	Nonsteroidal anti-inflammatory drugs Enamel matrix proteins, growth factors, and bone morphogenetic proteins (BMP)	Ketorolac mouth rinse and ketoprofen Recombinant human platelet-derived growth factor and recombinant human BMP-2

CLINICAL CORRELATE

What are the clinical considerations for the adjunctive use of local drug delivery (LDD) systems in periodontitis?

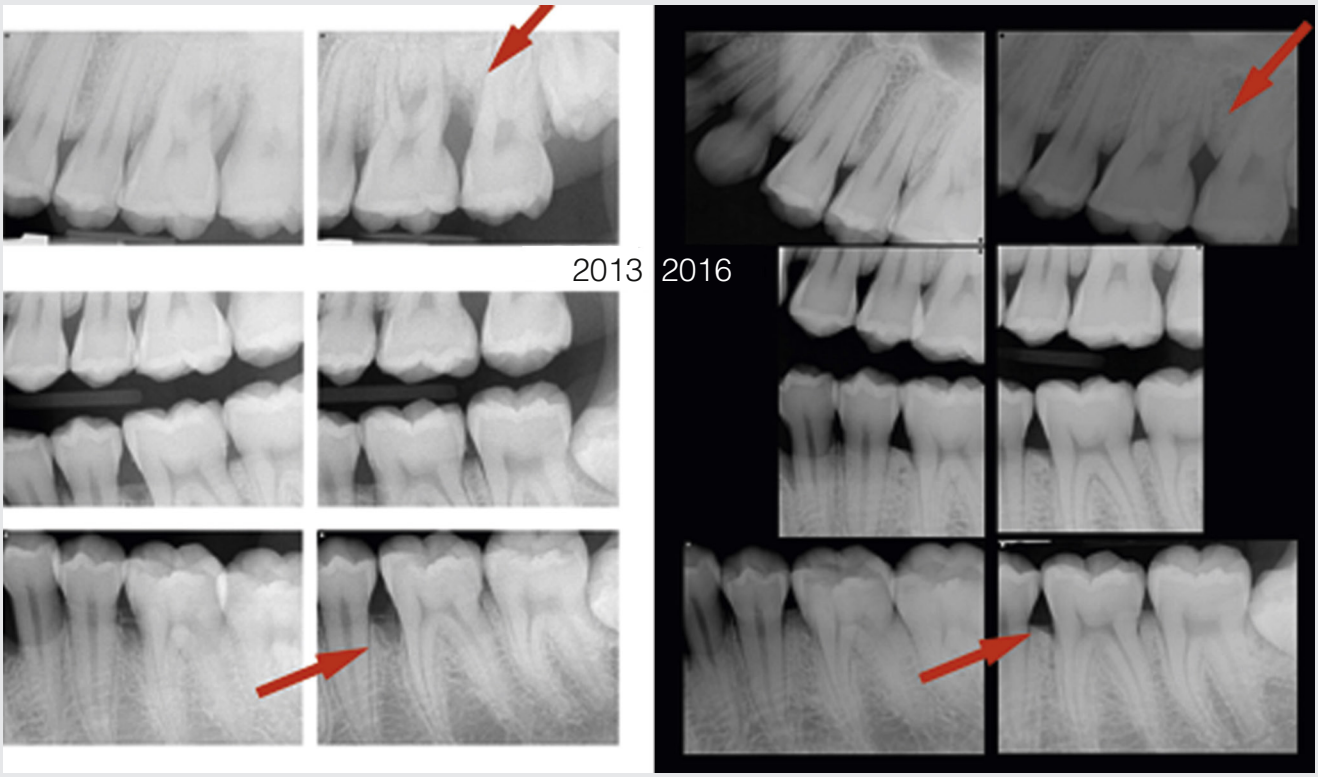
Indication: Quadrants in which localized recurrent and/or residual probing depths greater than or equal to 5 mm (with inflammation) is present following conventional periodontal therapies.

Therapies other than local drug delivery system should be considered in the following scenarios:

1. Multiple deep pocket depths (greater than or equal to 5 mm) within the same quadrant
2. Use of LDD has failed to control periodontitis
3. Presence of anatomical defects that require surgical correction. (reference #2 - PMID: 29539170)

CASE-BASED LEARNING EXERCISE

Scenario: A 17-year-old female was diagnosed with localized, rapidly progressing periodontitis with molar-incisor pattern (previously called aggressive periodontitis), and was treated nonsurgically with scaling and root planing and adjunctive systemic antibiotic therapy. The images show the positive radiographic changes 3 years after the initial therapy was initiated (red arrows). The antibiotics used during treatment were amoxicillin and metronidazole.



Questions

1. Based on the case presentation, which is the most likely periodontal pathogen involved in the etiology of the condition?
   a. *Aggregatibacter actinomycetemcomitans*
   b. *Actinomyces viscosus*
   c. *Fusobacterium nucleatum*
   d. *Treponema pallidum*
2. What is the advantage of using locally delivered antibiotics over systemic antibiotics?
   a. Better clinical result
   b. Reduced risk for bacterial resistance
   c. Direct effect of medication on calculus
   d. Cheaper
3. What is the main purpose of prescribing sub-antimicrobial-dose doxycycline (Periostat)?
   a. Bactericidal to selective group of periodontal pathogens
   b. Bacteriostatic to a broad range of periodontal pathogens
   c. Suppression of arachidonic acid pathway
   d. Inhibition of collagenase activity
4. All of the following are active ingredients in commercially available locally delivered antibiotics, except one. Which is the exception?
   a. Amoxicillin
   b. Minocycline
   c. Doxycycline
   d. Chlorhexidine

This chapter was developed from Chapters 52, 53, and 54 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Solutions

1. Answer: a

Explanation: The clinical presentation indicates that the patient has a clear molar-incisor pattern (formerly known as localized aggressive periodontitis). ***Aggregatibacter actinomycetemcomitans*** is the most likely periodontal pathogen as it is commonly associated with this condition.

2. Answer: b

Explanation: Locally delivered antibiotics have a reduced risk for inducing bacterial resistance compared with systemic antibiotics.

3. Answer: d

Explanation: Inhibition of collagenase (matrix metalloproteinases) activity is the main purpose of prescribing doxycycline at sub-antimicrobial doses.

4. Answer: a

Explanation: Amoxicillin is not commercially available for local delivery, but the rest are.

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30 Occlusal Evaluation and Therapy

Relevant Terminology

Terminology	Explanation
absolute anchorage	Use of mini implants for anchorage (temporary anchorage devices, TAD) in orthodontic therapy
anterior guidance	When protrusive movements are performed with teeth in opposing arches still in contact, the movement of the mandible is mainly guided by the palatal surfaces of maxillary anterior teeth. This constitutes the "anterior guidance" and ideally should result in immediate disclusion of all posterior teeth.
bruxism	Involuntary and/or unconscious grinding, gnashing, or clenching of teeth. It usually happens during sleep.
canine guidance	When laterotrusive movements are made with teeth in opposing arches still in contact with each other, the palatal surface of the canines on the working side guides the mandibular movement. This ideally should result in immediate disclusion of all posterior teeth and anterior teeth other than the specific canine guiding the movement.
centric occlusion	When teeth in opposing arches are contacting in maximum intercuspal position (tooth-to-tooth relationship)
centric relation	Position of the mandible when both condyle-disc assemblies are in their most superior positions in their respective glenoid fossae and against the slope of the articular eminences of each respective temporal bone (bone-to-bone relationship)
disclusion during excursive movements	Loss of contact/occlusion between teeth in opposing arches
excursive movements	Any movement of the mandible from centric positions is considered excursive; this includes: • Protrusion—mandible moves forward from maximum intercuspal position (MIP). Patient can establish this movement • Retrusion—mandible moves backward from MIP. This requires forceful maneuvering by the clinician and is performed for diagnostic purposes • Laterotrusion—mandible moves to the right or left side from MIP. The side toward which the mandible moves is called a "working side." The side away from mandibular movement is called the "nonworking" or "balancing" side • Mediotrusion—mandible moves back into MIP from laterotrusion
forced eruption	Eruption of the tooth coronally using orthodontic forces
fremitus	Vibration or micromovement of a tooth felt when patients tap their teeth together
group function	When laterotrusive movements are made with teeth in opposing arches still in contact with each other, the palatal inclines of the buccal cusps of maxillary teeth on the working side guide the mandibular movement. While this may be acceptable in stable occlusions, it is not considered an ideal occlusal scheme.
interference	Any occlusal contact in the centric relation closure arc or in any excursion that prevents the remaining occlusal surfaces from achieving stable contact or functioning harmoniously, or that which encourages masticatory system disharmony. Also called an *occlusal discrepancy.*

Relevant Terminology—cont'd

Terminology	Explanation
occlusal appliance	Custom-fitted appliance that provides bilateral simultaneous contact of all opposing posterior teeth in centric relation, shallow anterior guidance, and the immediate disclusion of all posterior teeth in every excursive movement
oral parafunctional habit	Normal function of teeth is mastication of food at the time of eating. An oral parafunctional habit is one that involves using teeth for activities other than mastication (e.g., bruxism, clenching, excessive gum chewing, lip or fingernail biting, thumb sucking).
periodontally accelerated osteogenic orthodontics	Clinical procedure that utilizes selective decortication with/without particulate bone grafting in the presence of orthodontic forces that relies on *regional acceleratory phenomenon* to accelerate tooth movement
regional acceleratory phenomenon	Reaction of hard and soft tissues to a noxious stimulus (such as decortication) that eventually enhances their healing capacity. Decortication is a procedure by which the cortical bone is perforated to allow bone forming cells in the bone marrow to make its way through the perforations into the overlying bone graft to enhance bone regeneration.
root proximity	Occurs when the roots of teeth are too close together, causing inability to perform oral hygiene. Progression of periodontal disease is rapid at these sites.

Fast Facts

Occlusal trauma and periodontitis	While periodontitis is caused by host immune response against pathogenic bacteria, occlusal trauma can amplify (not cause) localized loss of attachment from inflammatory bone damage. Thus it is a contributing factor to periodontal destruction.
Occlusion and periodontal inflammation	Because inflammation disrupts the integrity of the attachment apparatus and periodontitis leads to reduced bone support, periodontally compromised teeth have less resistance to forces from opposing teeth. It is important to diagnose whether tooth mobility is the result of excessive forces or active inflammation.
Occlusal adjustment	After inflammation control is established, if the clinician confirms that occlusal interferences correlate with a greater than expected loss of attachment, direct intervention in the patient's occlusion is performed by selective reshaping of the occluding surfaces of the teeth
Orthodontic therapy in periodontal patients	In addition to the therapeutic use of orthodontic alignment for establishing a harmonious occlusal scheme without interferences, orthodontics can be employed to improve inter-tooth alignment to help improve plaque control
Orthodontic treatment of osseous defects	Orthodontic treatment can often aid in the treatment of osseous defects with strategic tooth movement. One example is the resolution of proximal defects associated with proximally tipped teeth that can be eliminated by uprighting.

Core Knowledge

Introduction

All disciplines of dentistry require a comprehensive analysis of occlusal relationships for treatment planning. The reader is referred to Chapter 16 (Fig. 16.3) for a synopsis of occlusal evaluation and occlusal factors that may affect periodontal prognosis. This chapter reviews important aspects of the management of the occlusion specific to a patient's unique susceptibility to periodontitis.

Timing and Goals of Occlusal Treatment in Periodontal Therapy

Treatment of symptoms of occlusal traumatism is appropriate in any phase of therapy, except in cases of acute conditions and periodontitis where:[1]

- Definitive occlusal therapy must be performed only after inflammation reduction is achieved.
- Periodontal regenerative procedures must be attempted only after definitive occlusal therapy is performed satisfactorily.

The goals of occlusal treatment in periodontal therapy are:[2]

- Reduction (or elimination) of tooth mobility
- Establishment or maintenance of stable and physiologically acceptable maximal intercuspal position (centric occlusion)
- Provision of efficient masticatory function with no interferences in excursive movements
- Establishment of painless and comfortable occlusion with acceptable phonetics and esthetics
- Elimination or modification of parafunctional habits (e.g., bruxism)

CLINICAL CORRELATE

What are the clinical indicators of unresolved occlusal traumatism?

The following findings can indicate the need for continued occlusal therapy in a patient treated for trauma from occlusion and in whom other contributory factors (periodontal disease, endodontic infection, etc.) have been addressed effectively:[1]

- Tooth mobility continues to increase
- Tooth migration (away from excessive forces) continues with resultant increased spacing between teeth
- Radiographic changes associated with occlusal traumatism (e.g., widened periodontal ligament spaces, furcation radiolucencies) persist and worsen
- Pain and discomfort during mastication, premature contacts, and occlusal interferences continue to exist
- Persistent parafunctional habits and worsening of temporomandibular joint dysfunction are seen

Occlusal Treatment as a Part of Periodontal Therapy

All efforts in occlusal treatment are focused toward elimination of excessive forces, especially on periodontally affected teeth, and the maintenance of physiologically harmonious functioning of the temporomandibular joint and muscles of mastication. To this end, occlusal therapy may be accomplished using several different approaches:

- Occlusal adjustment (e.g., coronoplasty, selective grinding, coronal reshaping, occlusal equilibration)

CLINICAL CORRELATE

What are the acceptable outcomes/endpoints of occlusal treatment in the periodontal patient?

The desired outcomes following occlusal treatment include:[1]

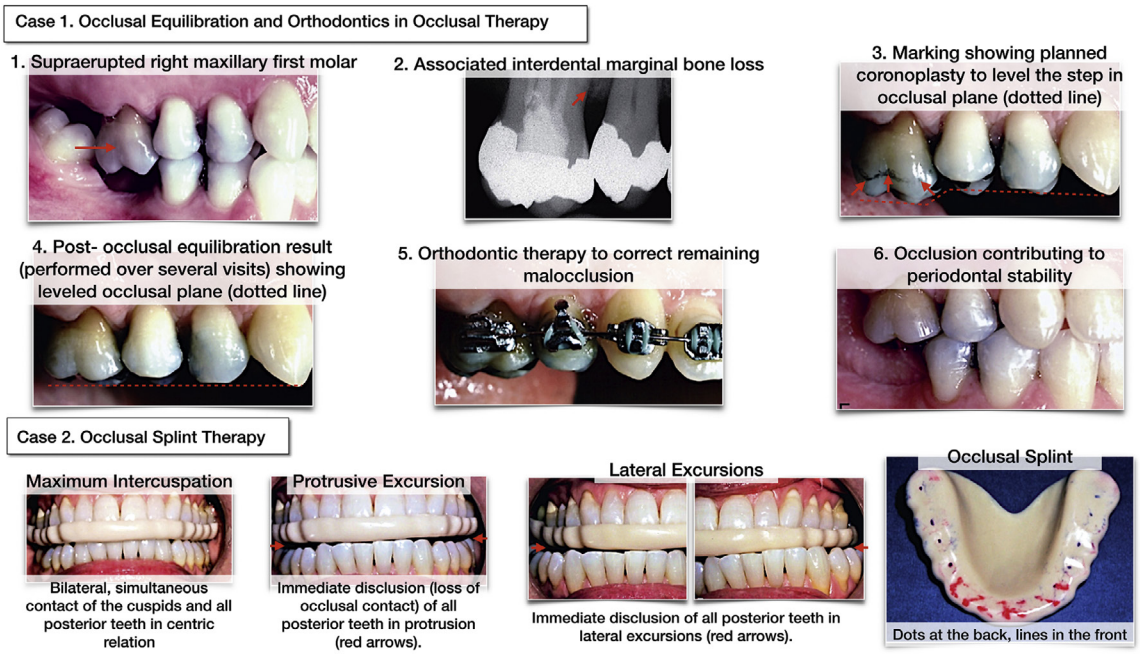
- Diminished/absent of tooth mobility (if present, a decreased mobility pattern that allows the patient to function comfortably without pain or danger of further deterioration is an acceptable endpoint).
- Prevention of further tooth migration (the pathologic migration already suffered by periodontally weak teeth may or may not be resolved due to altered forces from lip, cheek, tongue etc.)
- Stable or diminished changes on follow-up radiographs.
- A stable, physiologically and esthetically acceptable occlusion compatible with periodontal health.

- Management of parafunctional habits using occlusal splints
- Orthodontic tooth movements
- Temporary, provisional or long-term stabilization of mobile teeth using removable or fixed appliances

Fig. 30.1 gives examples of different approaches to treat occlusal problems that have an impact on the periodontium.

Adjunctive Role of Orthodontic Therapy

Orthodontic therapy can provide several benefits to adult periodontal patients. Fig. 30.2 shows examples of different adjunctive uses of orthodontics in the periodontal management of a patient.



• **Fig. 30.1** Examples of Different Approaches Used in Occlusal Therapy.

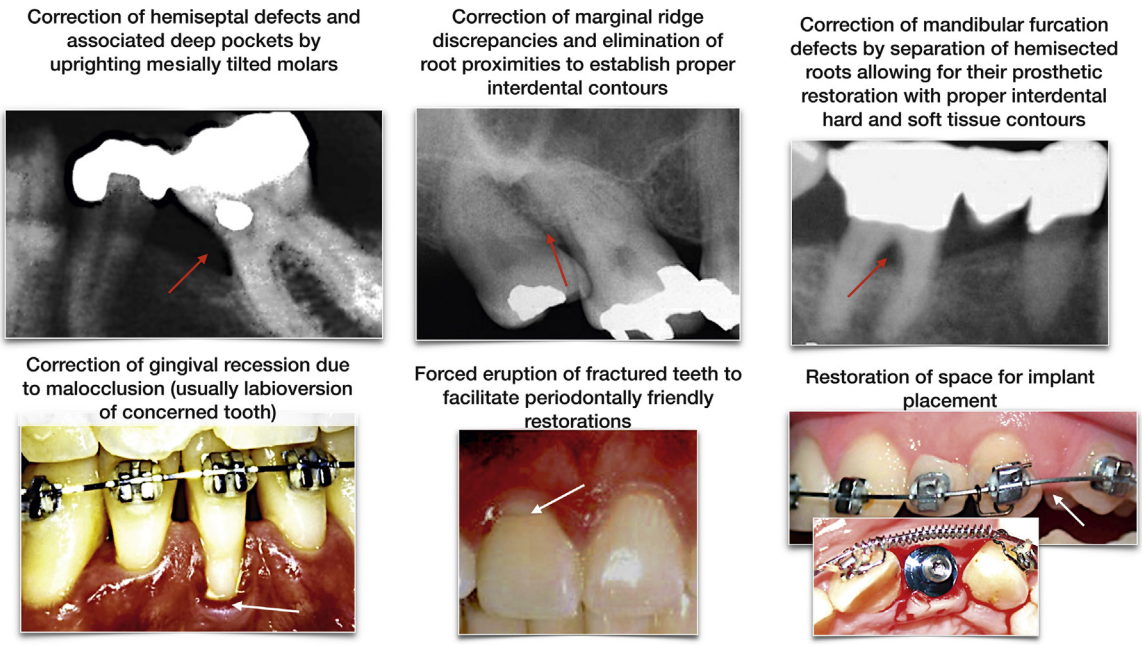
Case 1: Using occlusal equilibration and orthodontics to treat traumatic occlusion and enhance periodontal stability.

- To level the occlusal plane, and to prevent the collapsed bite from adversely affecting the periodontal support of the supraerupted molar tooth, the occlusal surface of the first molar is equilibrated and the malocclusion is corrected orthodontically.

Case 2: Using occlusal appliances to reduce the detrimental effects of parafunctional habits including abnormal tooth mobility.

- A well-designed and accurately fitted appliance can benefit masticatory system function while encouraging loose teeth in both arches to tighten as the supporting periodontium heals.
- Teeth that are opposing an appliance should be loaded as close as possible to their long axis.
- As teeth tighten from consistent use of the appliance, occlusal interferences may become more evident, which are then further subjected to occlusal equilibration for stable results.
- The bilateral simultaneous contact of all opposing posterior teeth in centric relation, shallow anterior guidance, and the immediate disclusion of all posterior teeth in every excursion, are essential elements of maxillary and mandibular occlusal appliances.

From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

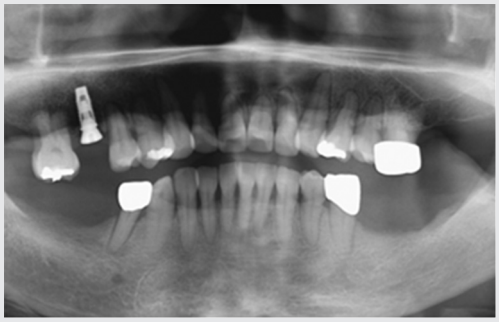


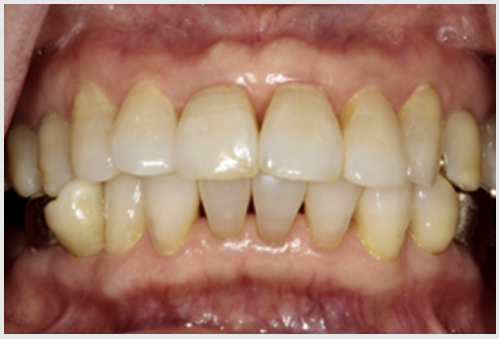
• **Fig. 30.2** Adjunctive Uses of Orthodontic Therapy in Periodontal and Dental Implant Therapy.

From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

CASE-BASED LEARNING EXERCISE

Scenario: A 63-year-old female presented with the chief complaint: "My teeth are shaking and I feel they are going to come out." Dental history: she had a removable partial denture that had just fractured. The implant in site #3 was placed more than 10 years earlier, but had never been restored (Fig. A). Findings: generalized widening of the periodontal ligament with mobility (2 and 3). Generalized probing depths of 1 to 3 mm with bleeding on probing in less than 10% of the sites.





Questions

1. According to the existing definition, the occlusal trauma for this patient can be classified as:
   a. Primary.
   b. Secondary.
   c. Primary after initial therapy and secondary after the osseous surgery.

2. What is characteristic for secondary occlusal trauma?
   a. Secondary refers to permanent dentition, primary is for deciduous teeth.
   b. Secondary refers to a second occurrence of trauma.
   c. Secondary refers to the presence of attachment loss.
   d. Secondary refers to diagnosis from referring specialist, primary refers to diagnosis from a general dentist.

3. This patient will most benefit from which of the following procedures?
   a. Biopsy
   b. Scaling and root planing
   c. Occlusal guard
   d. Open flap debridement

4. All of the following are part of phase 1 therapy, except one. Which is the exception?
   a. Motivational interviewing
   b. Oral hygiene instructions
   c. Occlusal adjustment
   d. Osseous surgery

This chapter was developed from Chapters 26, 55, and 56 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Solutions

1. Answer: a

Explanation: This is a case of primary occlusal trauma. There is minimal attachment loss, but characteristics of occlusal trauma (generalized widening of the periodontal ligament, mobility) are noted, indicating traumatic occlusive forces on a normal periodontium.

2. Answer: c

Explanation: Primary occlusal trauma generally occurs in the absence of attachment loss while secondary occlusal trauma generally occurs in the presence of attachment loss.

3. Answer: c

Explanation: Considering the clinical and radiographic presentation, this patient will most likely benefit from the use of an occlusal guard.

4. Answer: d

Explanation: All options are part of phase 1 therapy, except osseous surgery, which is part of phase 2 therapy.

References

1. Reinhardt RA, Killeen AC. Do Mobility and Occlusal Trauma Impact Periodontal Longevity? *Dent Clin North Am*, 2015;59(4):873–883. PMID: 26427572.

2. American Academy of Periodontology. (2000). Parameter on occlusal traumatism in patients with chronic periodontitis. *Journal of Periodontology*, *71*(Suppl. 5), S873–S875.

31

General Indications and Principles of Periodontal Surgery

Relevant Terminology

Terminology	Explanation
desensitizing agent	Agent applied to control root hypersensitivity
electrocoagulation	Coagulation and hemorrhage control by using the electrocoagulation current at the initial entry into soft tissue
electrosurgery and radiosurgery	Surgical techniques performed on soft tissue using controlled, high-frequency electrical (radio) currents in the range of 1.5– 7.5 million cycles per second
interdental knives	Shaped specifically to aid tissue removal from interdental areas
long junctional epithelium	After therapy, diseased sites may heal by forming a long junctional epithelial attachment to the tooth rather than regenerating a new connective tissue periodontal attachment that supports a short junctional epithelial attachment. This type of healing is considered reparative; it may be a less ideal outcome than true regeneration of the attachment apparatus
needle holders	Used to suture the flap at the desired position after the surgical procedure has been completed
periodontal dressing	Surgical dressing applied to the surgical site with the intention to protect the wound. It can be zinc oxide/eugenol-based (Wonder Pak) or non-eugenol-based (Coe-Pak)
periodontal knives (gingivectomy knives)	Instruments typically used for gingivectomy or tissue removal
periosteal elevators	Used to reflect and move the mucoperiosteal flap away from the underlying bone after the incision has been made for flap surgery (different instrument than extraction elevators)
phase II therapy	The surgical phase of periodontal therapy that follows after patient education / motivation, biofilm control, and thorough mechanical instrumentation of affected roots (i.e., phase I)
pocket elimination	Surgical treatment done with the intention of reducing periodontal pocket depth via resective or regenerative approaches
root hypersensitivity	Pain induced by thermal changes (cold or heat), by citrus fruits or sweets, or by contact with a toothbrush or a dental instrument. Commonly seen in patients after periodontal therapy

Fast Facts

Critical zones in periodontal surgery	• Soft tissue pocket wall • Root surface • Underlying bone • Attached gingiva
Periodontal surgery objectives	• To improve the prognosis for teeth and their replacements • To create a periodontal environment (shallow probing depths) that can be maintained by the patient and the professional • To improve aesthetics
Types of periodontal surgery	• Treating periodontal disease (e.g., pocket reduction surgery) • Enhancing esthetics (periodontal plastic/esthetic surgery procedures) • Aiding preparation for prosthetics (e.g., crown lengthening)
Time period of reevaluation after phase I therapy	• No less than 1–3 months; sometimes as much as 9 months after the completion of phase I therapy. Typically done 4–8 weeks following phase I therapy • A final decision on the need for periodontal surgery should be made only after a thorough evaluation of the effects of phase I therapy • Reevaluation of the periodontal condition includes repeat probing of the entire mouth and evaluation of calculus, root caries, defective restorations, and signs of persistent inflammation
Surgical pocket therapy	Because limited access and vision often hinder calculus and plaque removal from diseased sites, open flap access is a viable treatment option that increases the visibility and accessibility of the root surface for instrumentation
Assessing disease progression	Patient education and nonsurgical periodontal therapy often lead to sustainable results and do not necessitate surgical treatment. To determine site activity and assess the need for additional treatment, it is important to longitudinally assess the level of clinical attachment (i.e., the distance from the cementoenamel junction to the base of the pocket/sulcus).
Key components of periodontal surgery	Premedication only when medically required, behavioral modifications (e.g., smoking cessation), informed consent, universal precautions, appropriate anesthesia/sedation, tissue management, scaling and root planing, hemostasis, postoperative instructions, postoperative assessment
Pain management	It is expected that nearly one-half of periodontal surgical patients experience minimal to no postoperative pain, and fewer than 5% will have severe pain. Ibuprofen (600mg) administered preoperatively followed by postoperative use up to one tablet every 6 hours should be effective for pain management. The combination of acetaminophen and ibuprofen further improves pain management. Opioid prescriptions are not warranted for the vast majority of periodontal surgical procedures and run the risk of misuse

Core Knowledge

Introduction

After completion of nonsurgical periodontal therapy, which consists of patient education, biofilm control, and thorough disinfection and debridement of the tooth roots, the involved periodontal areas are re-evaluated. Sometimes, further treatment in the form of surgical intervention is required as a second phase of periodontal therapy, when better access for complete root planing is required, or correction of anatomic or morphologic defects is necessary. Placement of dental implants can be part of this surgical phase of therapy.

Phase II Periodontal Therapy

The surgical phase of therapy is also referred to as phase II therapy. This phase aims to improve the prognosis for teeth (and their replacements) and to improve aesthetics. In many cases, different therapies are combined to fulfill these objectives, or one therapy may fulfill many objectives (e.g., gingivectomy can improve esthetics as well as reduce pocket depths). Table 31.1 discusses the primary objectives of surgical periodontal therapy and the various techniques used to achieve them.

Surgical Pocket Reduction Therapy

The aims of surgical pocket reduction therapy are:

- To gain access to root surface and underlying bone, and remove irritants and infected tissue under direct vision
- To reduce the probing depth to levels that can easily be maintained free of plaque by patients along with periodic professional cleanings

These aims can be achieved by resecting or displacing the soft tissue wall of the pocket using both the "flap" approach and "gingivectomy" procedures.

TABLE 31.1 **Objectives of Different Types of Periodontal Surgical Procedures**

		Types of Periodontal Surgery				
		Pocket Reduction Surgery		Mucogingival Surgery		Preprosthetic
		Resective	Regenerative	Plastic	Esthetic	
Description of Surgical Approaches		Pocket reduction procedures that involve removal of soft and/or hard tissue wall of the pockets to reduce clinical probing depths	Pocket reduction procedures that involve the use of biomaterials such as bone grafts and membrane to reconstruct the lost periodontal attachment	Techniques to widen attached gingiva	Techniques for root coverage, re-creation of gingival papillae	• Techniques to modify the periodontal and neighboring tissues to receive prosthetic replacements • Implant placement and implant site development
Objectives	Patient plaque control enhancement by pocket reduction therapy	✓	✓			
	Patient plaque control enhancement by correction of morphologic/anatomic defects	✓	✓	✓	✓	✓
	Improvement of esthetics	✓	✓		✓	✓
Examples of Surgical Procedures		• Gingivectomy • Apically displaced flap • Undisplaced flap with or without osseous reduction • Modified Widman flap	• Guided tissue regeneration	• Free gingival grafts • Connective tissue grafts • Soft tissue allografts	• Pedicled flaps • Papilla reconstruction • Connective tissue grafts	• Crown lengthening • Ridge augmentation (e.g. guided bone regeneration, sinus grafting) • Vestibular deepening • Dental implant placement

This table provides a broad categorization that guides basic understanding regarding the various objectives behind commonly performed periodontal surgical procedures. It does not attempt to provide details on the complex decision making process or the considerable overlap in objectives/rationale that is often encountered in real clinical situations.

Rationale for Pocket Reduction Therapy

- Deep periodontal pockets are difficult to keep clean. Plaque accumulation in pockets leads to gingival inflammation, which leads to further pocket deepening. This is a vicious cycle.
- Definitive pocket therapy helps to eliminate or reduce pockets. Both outcomes, along with proper maintenance protocols, help in restoring a sulcus/pocket depth that can be easily maintained and kept free from plaque by the patient.

The **"Critical Probing Depth"** concept is an evidence-based clinical guide. It uses a probing depth (PD) value to decide the type of definitive pocket therapy that will be beneficial in a case and lead to attachment gain. This concept is not a hard-and-fast rule for treatment planning, but it may be used as a guide for decision-making.

Proposed guidelines for decision-making based on this concept are:[1]

- **Critical PD for scaling and root planing (SRP): 2.9 mm.** Below this critical PD of 2.9mm, if SRP is done, attachment loss occurs; when PD > 2.9 mm, SRP results in attachment gain
- **Critical PD for Modified Widman Flap (MWF): 4.2 mm.** Below this critical PD of 4.2mm, if MWF surgery is done, attachment loss occurs; when PD > 4.2mm, MWF results in attachment gain.
- **MWF trumps SRP at 5.5 mm.** Pockets deeper than 5.5mm respond better to MWF than to SRP with more gain in attachment levels.

CLINICAL CORRELATE

What are the indications for periodontal surgery?

Certain findings can indicate the need for a surgical phase of therapy:

- Areas with irregular bony contours, deep craters, etc., that result in gingival contours that are not self-cleansing or are difficult for patients to maintain free of plaque
- Pockets with restricted access to the root surface causing difficulties in complete removal of root irritants by just scaling and closed root planing
- Furcation involvement (grade II or III) including the need for root resection or hemisection
- Intrabony pockets distal to the last molars complicated by mucogingival problems
- Persistent inflammation even after nonsurgical management
- Mucogingival and esthetic problems
- As part of preprosthetic procedures (e.g., crown lengthening)

Results of Pocket Therapy

It is important to understand how periodontal surgical wounds heal following pocket therapy:

- Epithelial response to surgery in the healing period is quicker than that of all other tissues (connective tissue, cementum, or bone) of the periodontium.
- The fate of the postsurgical blood clot depends on its position and the precursor cells that are recruited *temporally* (at the right time) and *spatially* (into the right space).
- Hence periodontal healing by *repair* (where tissues regained are not exactly the same type as those destroyed by disease) or *regeneration* (where tissues regained are exactly the same type as those lost) are both possible outcomes of surgical therapy (Fig. 31.1), and the results depend on the surgical technique used to treat pockets.

Critical Tissue Zones in Pocket Therapy

The choice of periodontal surgical technique in pocket therapy depends on the evaluation of four different critical tissue zones that make up the periodontal pocket (Fig. 31.2):

- Zone 1: Soft tissue pocket wall
- Zone 2: tooth surface
- Zone 3: underlying bone
- Zone 4: attached gingiva

Methods of Pocket Therapy

Table 31.2 categorizes pocket therapy methods into three types based on how they achieve pocket reduction/elimination. They are:

1. New attachment techniques
2. Removal of soft tissue pocket wall
3. Removal of tooth surface of pocket

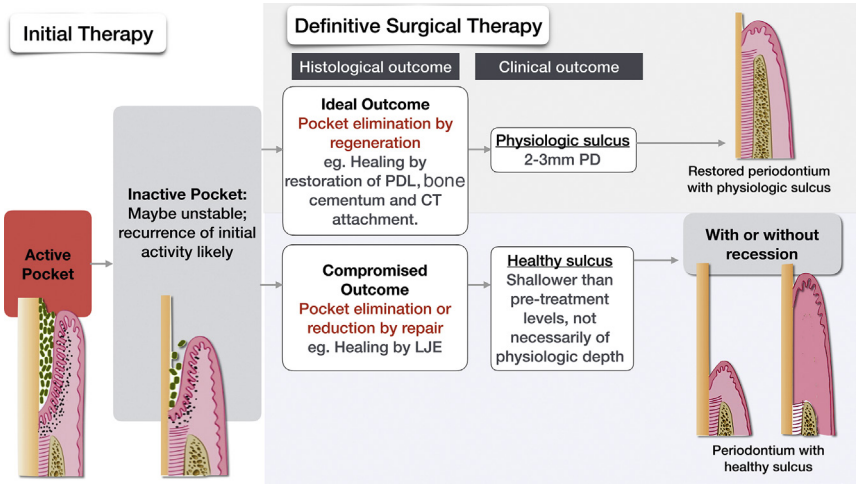
General Principles of Periodontal Surgery

Some general presurgical, surgical, and postsurgical considerations are common to all periodontal surgical techniques. Table 31.3 lists such considerations for phase II periodontal therapy.

Surgical Instruments

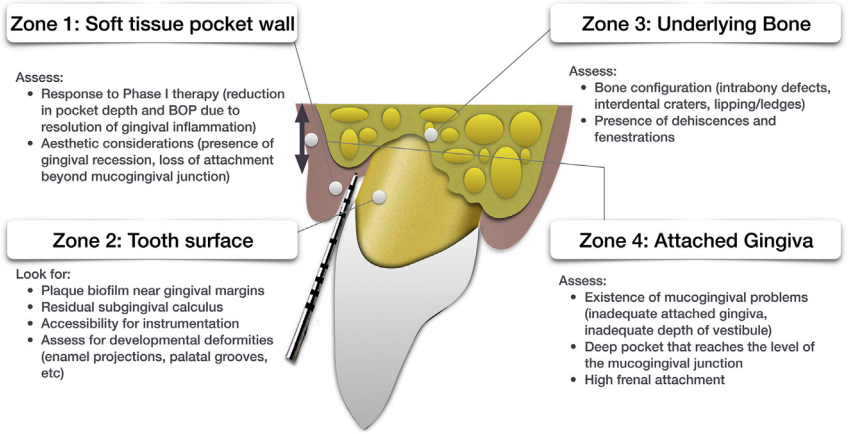
Periodontal surgical instruments are classified as follows (Fig. 31.3):

1. Excisional and incisional instruments
2. Periosteal elevators
3. Surgical curettes and sickles
4. Surgical chisels and files
5. Scissors
6. Hemostats and tissue forceps



• **Fig. 31.1** Possible Outcomes of Surgical Pocket Therapy. Pocket therapy can occur in stages. It is common to perform initial therapy and then, depending on the stability of the results, a particular approach for definitive therapy may be chosen. Here, the transformation of the initial deep, active pocket into a shallower, inactive, maintainable pocket requires some form of definitive pocket therapy and constant supervision thereafter. The results of definitive pocket therapy can be categorized into two possible outcomes that could occur, irrespective of the type of therapeutic approach chosen:

- Ideal outcome—histologic regeneration occurs with clinical PD of 2–3mm (physiologic sulcus)
- Compromised outcome—histologic repair occurs with reduced, but healthy sulcus (with or without recession). Note: Regardless of the surgical technique used for pocket therapy, a certain pocket depth recurs. Maintenance of this depth without any further attachment loss therefore becomes the therapeutic goal. *CT*, connective tissue; *LJE*, long junctional epithelium; *PD*, probing depth. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)



• **Fig. 31.2** Evaluation of Critical Tissue Zones in Surgical Pocket Therapy. The illustration shows the four tissue zones critical for decisions regarding therapeutic approaches to pocket therapy. Each zone is analyzed for various factors, following which a specific technique for definitive surgical therapy is selected. The approach with the highest likelihood to successfully solve the problem with the fewest untoward effects should be selected. *BOP, bleeding on probing*.

TABLE 31.2 Methods of Pocket Therapy

	New Attachment Techniques (Regenerative)	Removal of Pocket Wall (Resective)	Removal of Tooth Surface of Pocket Wall
Probing depth reduction or elimination achieved by:	• Filling in of new bone and regeneration of periodontal ligament, gingival connective tissue attachment and cementum (e.g., guided tissue regeneration and use of growth factors)	Shrinkage of inflamed tissues: • scaling and root planing Surgical removal of pocket wall (may include bone removal in some intrabone defects): • gingivectomy • undisplaced flap • modified Widman flap Apical displacement of pocket wall: • apically displaced flap	Partial tooth extraction: • hemisection • root resection Tooth extraction

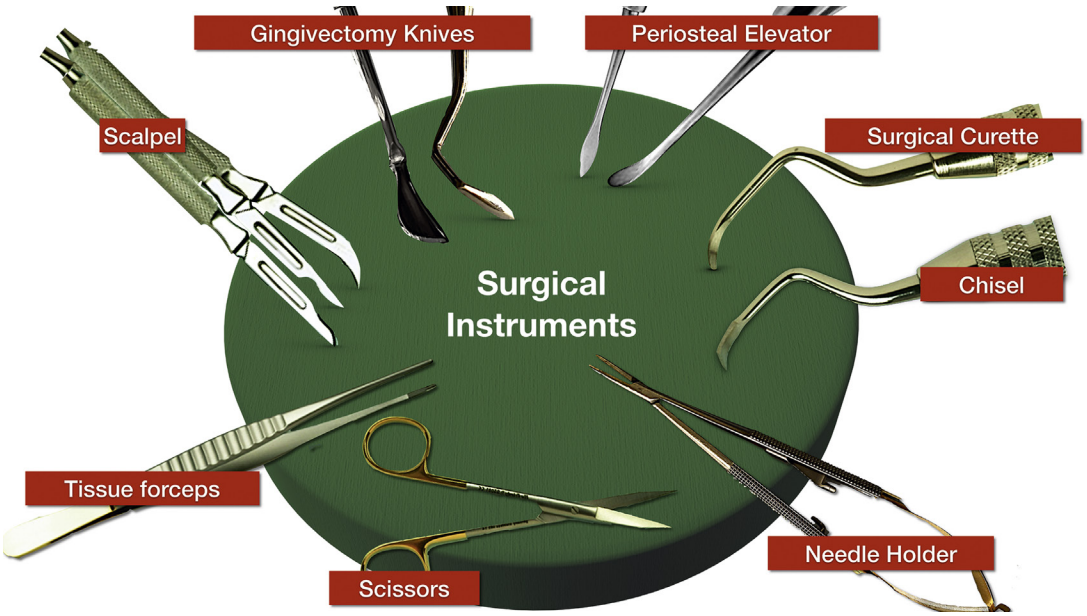
TABLE 31.3 General Considerations in Surgical Periodontal Therapy

Presurgical Considerations	
Patient preparation	• **Reevaluation after phase I therapy:** The reevaluation phase consists of re-probing and re-examining all the pertinent findings that indicated the need for the surgical procedure. The persistence of these findings confirms the indication for surgery. • **Premedication:** Prophylactic antibiotics, pre-emptive analgesics, and antimicrobial preprocedural rinse may be used as indicated. • **Smoking:** Patients should be informed of the deleterious effects of smoking on immunity and wound healing and encouraged to quit smoking completely. • **Control of detrimental systemic influences:** Underlying systemic diseases and conditions (e.g., stress, diabetes, hypertension, hormonal imbalances, hematological disorders, immunosuppression) should be under control; work with the patient's physician if needed. Record the patient's blood pressure and vital signs. • **Informed consent:** Discuss pros and cons, risks and outcomes of proposed surgical treatment, encourage patient to clarify doubts and get them to indicate their agreement to undergo the procedure by signing the informed consent form.
Emergency equipment	Drugs and equipment for emergency use should be readily available at all times.
Sterilization and asepsis	Universal precautions (e.g., protective attire) and barrier techniques must be employed.
Sedation and anesthesia	• Local anesthesia must be effectively administered using regional block injections and/or local infiltration. • Apprehensive and neurotic patients may require special management with antianxiety or sedative-hypnotic agents by inhalation, oral, intramuscular, or intravenous routes. The specific agents and the modality of administration are based on the desired level of sedation, the anticipated length of the procedure, and the overall condition of the patient.
Procedure selection	Once the problem list and treatment goals are finalized, the surgical procedure that accomplishes the required objectives in the most simple, predictable, and efficient manner must be carried out with due considerations for mucogingival and underlying osseous problems, anatomic restrictions (e.g., mental foramen, mandibular canal), physical restrictions (e.g., small mouth, gagging reflexes, restricted mouth opening), age, and systemic factors.
Surgical Considerations	
Choice of instruments	Instruments must be sharp to be effective; dull instruments inflict unnecessary trauma as a result of poor cutting and excessive force applied to compensate for their ineffectiveness.
Incisions	All incisions must be planned and made with clean, smooth, and definite movements. Indecision can result in ragged incisions, which take longer to heal.
Flap design	• Should allow for optimal visibility, accessibility, and preservation of keratinized tissue • Should prevent unnecessary bone exposure • Should allow for healing by primary intention versus secondary intention wherever possible • Should allow for adequate vascularity
Tissue handling	• Tissue manipulation should be precise, deliberate, and gentle; traumatic instrumentation produces excessive tissue injury, causes postoperative discomfort, and delays healing. • Tissue tags must be removed to allow for rapid healing and prevent regrowth of granulation tissue. • Observe the patient at all times; facial expressions, pallor, and perspiration are distinct signs that a patient is experiencing pain, anxiety, or fear. The clinician's responsiveness to these signs can be the difference between success and failure in patient management.
Hemostasis	• Good intraoperative control of bleeding permits accurate visualization of the extent of disease, the pattern of bone destruction, and the anatomy and condition of the root surfaces. It also prevents excessive loss of blood. • After flap reflection and the removal of granulation tissue, bleeding stops or is considerably reduced. • Continuous suctioning of the surgical site with an aspirator and the application of pressure to the wound with moist gauze can help to control bleeding. Intraoperative bleeding that is not controlled with these simple methods may indicate a more serious problem requiring additional control measures. • The use of local anesthesia with epinephrine is also helpful. It is important to remember that its action is effective for a short duration and it should not be used toward the end of the surgical procedure. When the patient is dismissed from the appointment and the effect of the vasoconstriction is no longer present, bleeding can occur during the patient's trip home. • Hemostasis may be achieved with hemostatic agents like absorbable gelatin sponge (Gelfoam), oxidized cellulose (Oxycel), oxidized regenerated cellulose (Surgicel Absorbable Hemostat), and microfibrillar collagen hemostat (Avitene, CollaCote, CollaTape, CollaPlug).

Continued

TABLE 31.3 General Considerations in Surgical Periodontal Therapy—cont'd

Flap stabilization	Using sutures, flap stabilization is done to prevent flap displacement, excessive bleeding, hematoma formation, bone exposure, and possible infection in the healing period.
Dressings	On completion of the procedure, clinicians may elect to cover the area with a surgical dressing (Eugenol or non-Eugenol packs).
Postsurgical Considerations	
Postoperative instructions	Oral and printed postoperative instructions should be given before the patient is dismissed from the chair.
Management of postoperative pain and dentinal hypersensitivity	• For most healthy patients, a preoperative dose of ibuprofen (600–800 mg) followed by one tablet every 8 hours for 24–48 hours is very effective for reducing discomfort after periodontal surgery. Patients are advised to continue taking ibuprofen or acetaminophen thereafter, if needed. • Root sensitivity occurs more frequently in the cervical area of the root, where the cementum is extremely thin. Scaling and root planing procedures remove this thin cementum, thereby inducing dentinal hypersensitivity. Biofilm control and desensitizing agents help to control root sensitivity.



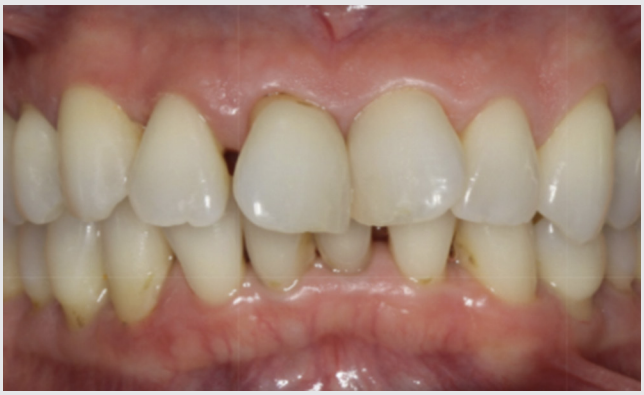
• **Fig. 31.3 Periodontal Surgical Instruments.**

1. Excisional and incisional instruments: includes scalpels (BP blades #15, #15C and #12, #12D) and gingivectomy knives (Kirkland and Orban's knives).
2. Periosteal elevators: double-ended instruments used to reflect and move the flap after the incision has been made for flap surgery (e.g., Woodson and Prichard elevators).
3. Surgical curettes and sickles: larger and heavier curettes and sickles are often needed during surgery for the removal of granulation tissue, fibrous interdental tissues, and tenacious subgingival deposits (e.g., Prichard's surgical curette).
4. Surgical chisels and files: used with a push or pull motion to remove sharp interdental bony projections (widow's peaks at line angles of teeth) in osseous contouring (e.g., back-action chisel, Ochsenbein chisels).
5. Scissors: Scissors (e.g. Goldman-Fox) used in periodontal surgery are effective in trimming flap margins, enlarging incisions to drain periodontal abscesses and removing muscle attachments in mucogingival surgery. Tissue nippers and scissors also help to remove tissue tags during gingivectomy.
6. Hemostats and tissue forceps: the tissue forceps are used to hold the flap during suturing. This instrument is also used to position and displace the flap after it has been reflected (e.g., DeBakey forceps).
7. Needle holders: help to securely suture the flap in the desired position (both when undisplaced or displaced) after completing the surgical procedure. In addition to the regular types of needle holders, the Castroviejo needle holder is used for delicate, precise techniques that require quick and easy grasp and release of the suture needle.

(From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

CASE-BASED LEARNING EXERCISE

Scenario: A 47-year-old Caucasian male presented to a periodontal re-evaluation appointment 5 weeks after four quadrants of scaling and root planing. Clinical examination revealed improvements in probing depth reduction in several areas of the mouth. Deep pockets (>5 mm) remained on the distal aspect of maxillary central incisors and on the maxillary molars. Radiographic exam revealed mainly horizontal bone loss in the areas with deeper probing depths. The patient's oral hygiene had improved significantly since the initial examination. BOP during this appointment was 10%. Patient was a controlled type 2 diabetic and otherwise in good health, and a nonsmoker.



Questions

1. What is the ideal time to perform periodontal reevaluation?
   a. 2 weeks
   b. 4–8 weeks
   c. 3 months
   d. 6 months

2. Is this patient a good candidate for periodontal pocket reduction surgery?
   a. Yes
   b. No

3. Based on the information provided, is the regenerative approach a good selection in this clinical situation to reduce pockets in molars?
   a. Yes
   b. No

4. Which of the following will be a preferred surgical technique to reduce periodontal pockets in maxillary incisors?
   a. Modified Widman flap
   b. Apical displacement flap
   c. Resective osseous surgery
   d. Guided tissue regeneration

Clinical photo attribution: Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

This chapter was developed from Chapters 57 and 59 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Solutions

1. Answer: b

Explanation: 4–8 weeks will provide adequate time for the healing of soft tissues (epithelium and connective tissue) following scaling and root planing. It will also allow sufficient time to assess any improvements in the patient's oral hygiene compliance.

2. Answer: a

Explanation: The patient has demonstrated his commitment to improving his oral hygiene and has no major systemic conditions except for diabetes, which is well controlled.

3. Answer: b

Explanation: The regenerative approach is not indicated in pockets associated with horizontal bone loss.

4. Answer: a

Explanation: Due to the esthetic location involved, a more conservative procedure, such as a modified Widman flap, would be preferred because it would minimize the chance of postoperative recession. Consideration should also be given to preserve papilla, when periodontal surgery is performed in esthetically demanding areas.

Reference

Lindhe, J., Socransky, S. S., Nyman, S., Haffajee, A., & Westfelt, E. (1982). "Critical probing depths" in periodontal therapy. *Journal of Clinical Periodontology*, *9*(4), 323–336.

32 Periodontal and Peri-implant Surgical Anatomy

Relevant Terminology

Terminology	Explanation
alveolar process of maxilla	Part of bone in maxilla that houses the roots of maxillary teeth.
anatomic spaces	Spaces *associated* with critical structures in the oral cavity, that distend and present with pain and/or swelling due to spreading infection (or hemorrhage).
dysesthesia versus paresthesia	Injury to nerves can lead to one of these two complications. There is abnormal sensation in both conditions; an unpleasant sensation is referred to as dysesthesia; an abnormal sensation which is *not* unpleasant is referred to as paresthesia.
exostosis	Normal range of anatomic variation characterized by excessive bone.
ostium	Opening located at the highest point on the mesial wall of the maxillary sinus through which the maxillary sinus drains into the middle meatus of the nasal cavity.
pneumatization	Process by which paranasal sinuses (including maxillary sinus) expand in volume. The volume of the maxillary sinuses increases with age.
retromolar triangle	Triangular area located distal to the third molar that contains glandular and adipose tissue covered by nonkeratinized epithelium.
schneiderian membrane	Membrane that lines the interior of the maxillary sinus cavities. Histologically, it consists of pseudostratified ciliated columnar epithelium.
space separation	Submandibular space is separated from sublingual space by the mylohyoid muscle that attaches to the mylohyoid ridge.
torus	Maxillary torus is commonly located in the midline of the palate and mandibular torus commonly occurs bilaterally on the lingual aspect of canines and premolars.

Fast Facts

Mandibular canal	Contains inferior alveolar nerves and vessels. It divides into two branches, one exiting from the mental foramen as the mental nerve and the other running anteriorly via the incisive canal.
Mental foramen	Located on the buccal aspect of the body of the mandible below the apices of the premolars (closer to second premolar), halfway between the alveolar margin and the lower border of the mandible.
Mental nerve loop	High prevalence (>85%), bilateral, average length of 4 mm (0.5–5 mm).
Processes of maxilla	Alveolar, palatine, zygomatic, and frontal.
Foramens in hard palate	Incisive canal: located anteriorly behind the maxillary incisors, allows nasopalatine nerves and vessels to pass through. Greater palatine foramen: located 3–4 mm anterior to the posterior border of the hard palate. Greater palatine nerves and vessels emerge through this foramen and run anteriorly.

Fast Facts—cont'd

Blood and nerve supply to maxillary sinus	Blood supply is derived from anterior, middle, and posterior superior branches of the maxillary artery. It is innervated by anterior, middle, and posterior superior nerves, and branches of the maxillary nerve.
Maxillary sinus septa	Roughly one-third of maxillary sinuses present with bony septa; they are more common in the middle third of the sinus, compared with anterior and posterior areas. Between the second premolar and first molar seems to be the most common location.
Posterior superior alveolar artery	It can have an intraosseous course on the lateral aspect of the maxillary sinus and should be avoided during a lateral (direct) sinus augmentation procedure.
Sublingual space	Infection in this anatomic space (located in the anterior aspect of the floor of the mouth) can raise the tongue, leading to breathing difficulties; it can therefore be life-threatening.
Muscles encountered in periodontal surgeries	Mentalis, incisivus labii inferioris, depressor labii inferioris, depressor anguli oris, and buccinator.

Core Knowledge

Introduction

Several vital anatomic structures in close proximity to the teeth and jaws are at risk of injury or damage during periodontal and implant surgical procedures. A clinician must possess a sound knowledge of the anatomy of the periodontium and jaw to determine the scope and feasibility of periodontal and implant surgical procedures, and to minimize the risks associated with damaging vital structures.

- **Neurovascular structures** are vulnerable to injury from sharp incisions and dissections, and from drilling for implant site osteotomies and bone augmentation procedures.
- **Fascial spaces** such as the sublingual and submental spaces, if entered inadvertently, pose an immediate risk of bleeding and a subsequent risk of infection.

This chapter reviews only the crucial anatomic structures of the maxilla, mandible, and surrounding tissues that are critical to recognize when planning and performing periodontal and implant surgical procedures. The reader is referred to Chapter 58 in *Newman and Carranza's Clinical Periodontology* (13th ed.) for further detailed reading of the topic.

Mandible

The mandible, a horseshoe-shaped bone connected to the skull by the temporomandibular joints, presents several landmarks of great surgical importance for both periodontal and implant surgical procedures (Fig. 32.1).

Maxilla

The maxilla, a paired bone that contains the maxillary sinus and the nasal cavity, has the following four processes:

- The *alveolar process* contains the sockets for the maxillary teeth.
- The *palatine process* extends horizontally from the alveolar process to meet its counterpart from the opposite maxilla at the midline intermaxillary suture, and extends posteriorly with the horizontal plate of the palatine bone to form the hard palate.
- The *zygomatic process* extends laterally from the area above the first molar and determines the depth of the vestibular fornix in that region.
- The *frontal process* extends upward and articulates with the frontal bone at the frontomaxillary suture.

Fig. 32.2 shows the important anatomic landmarks that require consideration during periodontal and implant surgery.

Anatomic Spaces

Anatomic spaces are compartments found close to the operative field of periodontal and implant surgery sites containing loose connective tissue that is easily distended by hemorrhage, inflammatory fluid, and infection. Surgical invasion of these areas may result in dangerous hemorrhage (intraoperative complication) or infections (postoperative complication) and should be carefully avoided.

Table 32.1 lists the important anatomic landmarks that require consideration during periodontal and implant surgery.

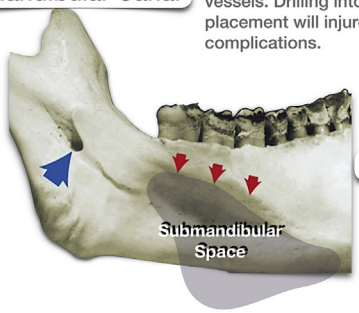
CLINICAL CORRELATE

What is Ludwig angina?

- Ludwig angina is a life-threatening fascial space infection involving the submandibular, sublingual, and submental spaces.
- It is characterized by extraoral swelling and edema of the lower face and neck, with intraoral swelling that raises the floor of the mouth and tongue.
- If not treated urgently, it can lead to airway obstruction requiring tracheostomy due to edema of the neck and glottis.
- Infection can spread to other fascial spaces of the head and neck, including the retrosternal space.
- Although the bacteriology of these infections has not been completely determined, they are presumed to be mixed infections with an important anaerobic component.

Mandibular Canal

(Blue arrow) Contains the inferior alveolar nerve and vessels. Drilling into the canal during implant placement will injure these structures leading to complications.

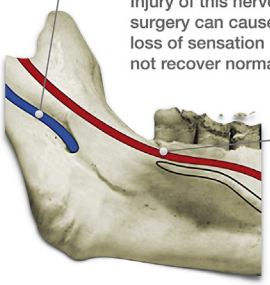


Mylohyoid Ridge

(Red arrows) Excessive lingual flap retraction or too lingual an implant placement in the posterior mandible might violate the highly vascular submandibular space under the mylohyoid ridge, leading to hemorrhagic complications.

Inferior Alveolar Nerve

Injury of this nerve during periodontal or implant surgery can cause chronic pain, altered sensation or loss of sensation postoperatively that may or may not recover normal impulse conduction.

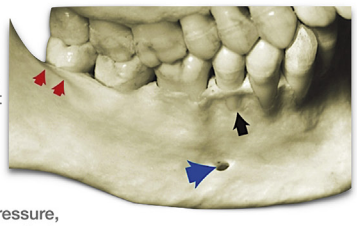


Lingual Nerve

The lingual nerve lies close to the surface of the oral mucosa in the third molar area. It can be damaged during anesthetic injections, when a periodontal partial-thickness flap is raised in the third molar region or when releasing incisions are made in the area.

External Oblique Ridge

(Red arrows) Resective osseous therapy may be difficult or impossible in this area because of the amount of bone that must be removed distally toward the ramus to achieve optimal results.

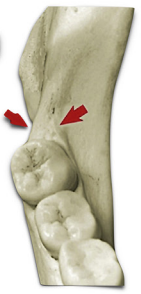


Mental Foramen

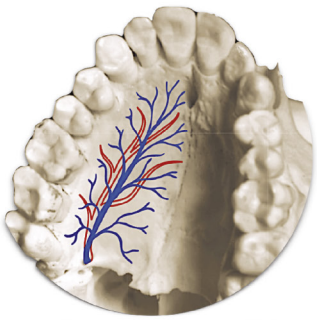
(Blue arrow) Surgical trauma, including pressure, manipulation, accidental nicking or postsurgical swelling of the mental nerve tissues, may result in transient or permanent paresthesia of the lip.

Retromolar Triangle

This region (red arrows) is occupied by glandular and adipose tissue and normally covered by unattached, nonkeratinized mucosa. If sufficient space exists distal to the last molar, a band of attached gingiva may be present; **only** in such a case can a distal flap procedure be performed effectively.



• **Fig. 32.1** Surgical Anatomy: Considerations in the Mandible. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

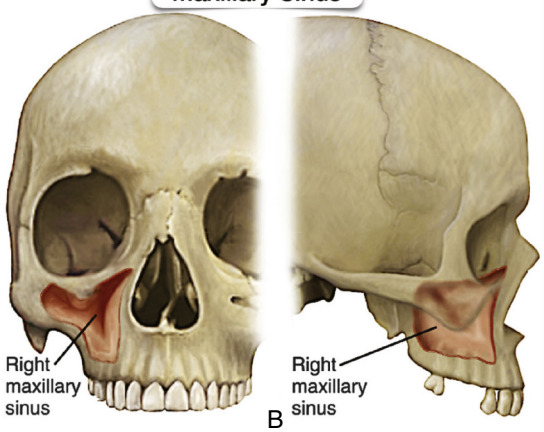


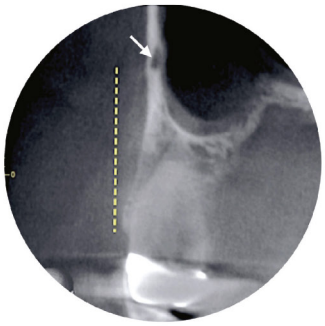
Greater Palatine Foramen, Nerves and Blood Vessels

- Palatal flaps for gingival and connective tissue grafts should be carefully performed and donor sites carefully selected to avoid invading these areas because profuse hemorrhage may ensue.
- Vertical incisions in the molar region should be avoided

- The ability to perform periodontal osseous surgery in the posterior maxilla may be limited when the sinuses are severely pneumatized.
- When there is no bone to maintain integrity of the maxillary sinus floor, extraction of teeth with roots exposed into the maxillary sinus, surgical procedures for bone augmentation or the placement of implants in edentulous areas in this region can result in an oroantral communication.

Maxillary Sinus





Intraosseous Branch of Posterior Superior Alveolar Artery

When considering a lateral window approach to sinus floor elevation and bone augmentation for implant site devlopement, the loaction of this artery (white arrow) in relation to the position of the lateral window for sinus augmentation presents a risk for bleeding complications.

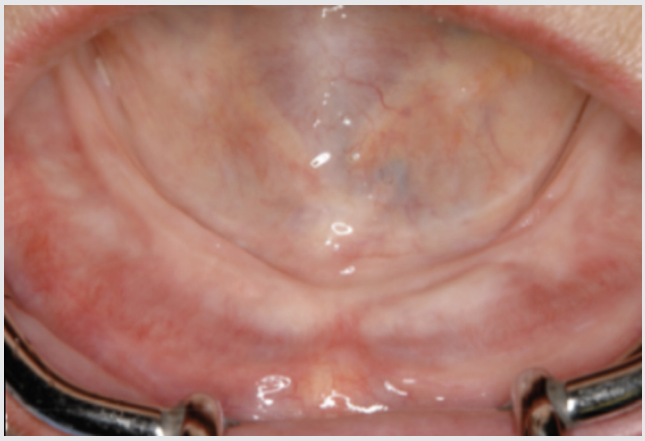
• **Fig. 32.2** Surgical Anatomy: Considerations in the Maxilla. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

TABLE 32.1 Anatomic Spaces: Complications in Periodontal and Implant Surgery

Space	Complications
Canine fossa	Infection of this area results in: • Swelling of the upper lip, which obliterates the nasolabial fold • Swelling of the upper and lower eyelids, which closes the eye
Buccal space	Infection of this area results in: • Swelling of the cheek that may extend to the temporal space • Swelling of the submandibular space, with which the buccal space communicates
Mental space	Infection of this area results in a large swelling of the chin, extending downward
Masticator space	• Infection of this area results in swelling of the face, severe trismus, and pain • If the abscess occupies the deepest part of this compartment, facial swelling may not be obvious, but the patient may complain of pain and trismus • Patients may also have difficulty and discomfort when moving the tongue and swallowing
Sublingual space	Infection of this area raises the floor of the mouth and displaces the tongue, which results in pain and difficulty swallowing, but there is little facial swelling.
Submental space	Infections of this area arise from the region of the mandibular anterior teeth and result in swelling of the submental region; infections become more dangerous as they proceed posteriorly.
Submandibular space	Infections of this area originate in the molar or premolar area and result in swelling that obliterates the submandibular line and causes pain on swallowing.

CASE-BASED LEARNING EXERCISE

Scenario: A 58-year-old Caucasian female presented with the chief complaint: "The lower denture that I have is not stable in my mouth and sometimes I feel pain in the lower jaw upon biting when I'm using my denture." She quit smoking 20 years earlier and was known to have hypertension, controlled with lisinopril. All her teeth had been extracted approximately 6 years earlier because they were not restorable due to severe caries. Palpation revealed ridge resorption and knife-edged ridge (mainly in the mandibular anterior sextant).



Clinical photo attribution: Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

Questions

1. What are the key anatomic landmarks in the mandible that should be considered in treating this patient?
   a. Inferior alveolar canal
   b. Mental foramen
   c. Lingual nerve
   d. All of the above

2. If multiple implants are planned, what is the ideal imaging modality to assess the anatomic landmarks?
   a. Panoramic radiographs
   b. Periapical radiographs
   c. Cone beam computed tomography
   d. Occlusal radiographs

3. The pain the patient feels on the mandibular ridge upon biting (when wearing the denture) is probably due to:
   a. Denture-induced pressure on the mental nerve
   b. Denture-induced pressure on the inferior alveolar nerve
   c. Denture stomatitis
   d. All of the above
4. When performing surgery, vertical incision should be avoided on the lingual aspect of mandibular third molars.
   a. True
   b. False

Case-Based Learning Exercise

This chapter was developed from Chapter 58 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Solutions

1. Answer: d

Explanation: Depending on the extent of the procedure involved, all the mandibular anatomic landmarks mentioned should be considered.

2. Answer: c

Explanation: Of the listed imaging modalities, cone beam computed tomography (CBCT) will provide the maximum information for the surgeon to avoid damaging anatomic structures intraoperatively.

3. Answer: a

Explanation: In completely edentulous patients the mental foramen will be very close to the superior aspect of the ridge, due to ridge resorption. In such cases, the pressure induced by a denture during chewing can cause discomfort or pain. CBCT is very valuable in diagnosing this condition.

4. Answer: a

Explanation: It is important to avoid vertical incisions on the lingual aspect, to avoid damage to the lingual nerve.

33

Pocket Reduction Surgery: Resective Approach

Relevant Terminology

Terminology	Explanation
flat architecture	Interdental bone remains at the same level as the buccal or lingual/palatal bone.
gingivectomy	Surgical excision/removal of gingiva.
gingivoplasty	Surgical reshaping of gingiva performed to establish a more physiologic contour.
ideal bone architecture (positive or scalloped architecture)	Interdental bone is coronal to the buccal or lingual/palatal bone.
negative (or reversed) architecture	Interdental bone is apical to the buccal or lingual/palatal bone.
ostectomy	Process that involves the removal of tooth-supporting bone.
osteoplasty	Process that involves reshaping of bone without the removal of tooth-supporting bone.
widow's peaks	Peaks of bone that remain on the facial and lingual/palatal aspect of teeth and interproximal line angles during an osseous surgery procedure. It is important to remove widow's peaks in subsequent steps during the surgery to establish a smooth flowing bony architecture that will be followed by the overlying gingival tissues after healing.

Fast Facts

Rationale for pocket reduction	Pocket reduction procedures are aimed at reducing or eliminating periodontal pockets (deepened gingival sulci) that harbor periodontal pathogens and make self-care and professional periodontal care challenging.
Resective (subtractive) approach	Eliminating pockets by procedures that involve removal of the soft and/or hard tissue wall of the pockets.
Regenerative (additive) approach	Eliminating pockets by procedures that allow restoration of the lost attachment apparatus.
Indications for resective procedures	• Multiple deep probing depths (>5 mm) in a quadrant at the time of periodontal reevaluation (after phase I therapy) with horizontal bone loss • Furcation-involved molars with defect(s) not amenable to regeneration • Gingival (pseudo) pockets (an indication for gingivectomy)
Contraindications for resective osseous procedures	• Multiwalled intrabony defects or furcation defects amenable to regeneration • Severe bone loss and/or tooth mobility • If pocket reduction by resection would compromise the periodontal support of adjacent teeth • If the patient is not a good candidate for any periodontal surgical procedure (e.g., has poor oral hygiene or complex medical condition)
Expected adverse postoperative outcomes following resective approaches	• Exposure of more tooth structure and/or gingival recession (attachment loss) • If more of the dentin is exposed, dentinal hypersensitivity is expected • Increase in tooth mobility

Continued

Fast Facts—cont'd

Types of suture materials	Nonabsorbable (nonresorbable): • Silk—braided • Nylon—monofilament (Ethilon) • ePTFE—monofilament (Gore-Tex) • Polyester—braided (Ethibond) Absorbable (resorbable): • Surgical—gut • Plain gut—monofilament (30 days) • Chromic gut—monofilament (45–60 days) Synthetic: • Polyglycolic—braided (16–20 days) • Vicryl (Ethicon) • Dexon (Davis & Geck) • Polyglycaprone—monofilament (90–120 days) • Monocryl (Ethicon) • Polyglyconate—monofilament (Maxon)
Criteria for choice of surgical therapy in treating gingival enlargements	For gingivectomy: • Small areas of enlargement (up to 6 teeth) • No attachment loss or bone loss • Abundance of keratinized tissue For flap surgery: • Large areas of enlargement (>6 teeth) • Presence of osseous defects • Limited keratinized tissue
Osseous resection technique	Involves four steps: 1. Vertical grooving 2. Radicular blending 3. Flattening interproximal bone 4. Gradualizing bone margins

Core Knowledge

Introduction

Periodontal access surgery, an adjunct to nonsurgical periodontal therapy, has the following objectives:

- **Primary objective**—to obtain access for root instrumentation to thoroughly remove plaque biofilm and calculus from the root surfaces.
  - Both gingivectomy and flap surgery provide access for root instrumentation.
- **Secondary objective**—to obtain pocket reduction via soft and hard tissue resection or periodontal regeneration, to facilitate home care and long-term maintenance.
  - Gingivectomy achieves pocket reduction only by resection of the suprabony soft tissue pocket.
  - Periodontal flap surgery achieves pocket reduction via soft tissue resection, osseous resection, or periodontal regeneration. (Periodontal regeneration using flap surgery is reviewed in Chapter 34.)

Table 33.1 compares gingivectomy and flap surgery procedures.

CLINICAL CORRELATE

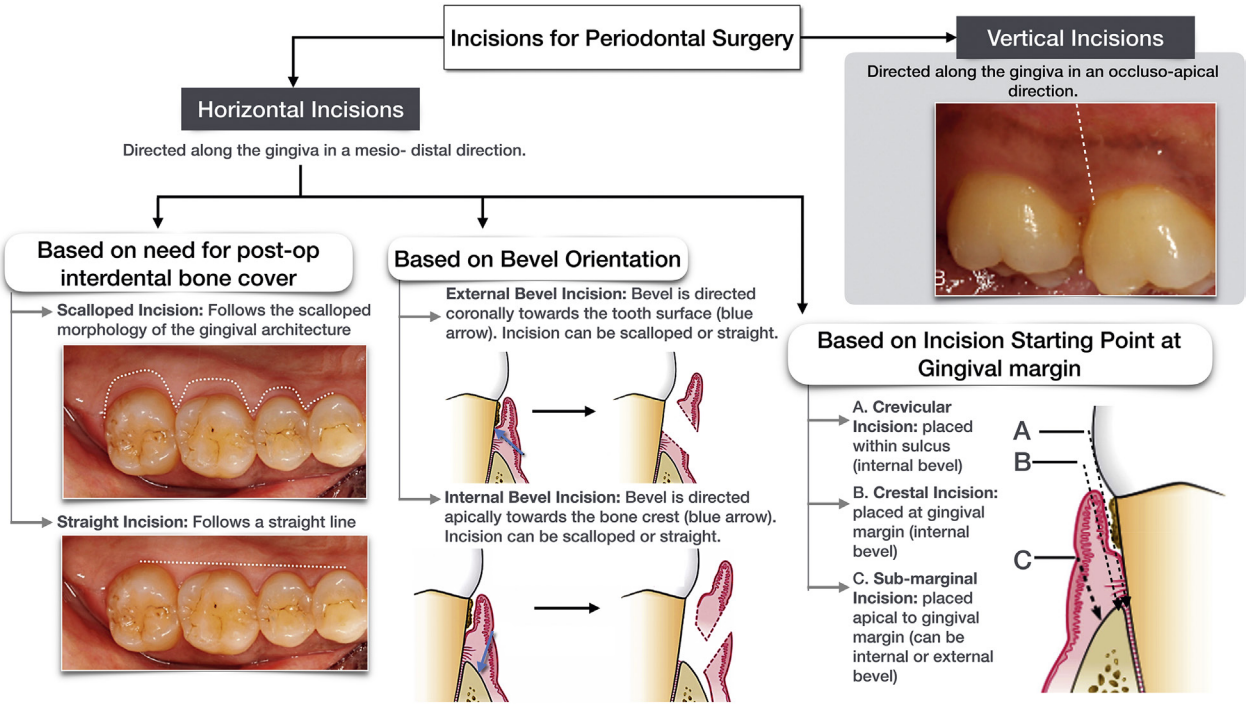
How may flap procedures be classified?

Periodontal flap procedures may be classified based on bone exposure after flap reflection, placement of the flap, or management of the papilla.

- Based on bone exposure after flap reflection:
  - Full-thickness flap—also called *mucoperiosteal* flap; the incision must cut through the periosteum up to bone, and the entire mucosa and periosteum is elevated off bone. This is achieved by blunt dissection using a periosteal elevator. This complete exposure of and access to the underlying bone is indicated when resective or regenerative osseous surgery is contemplated.
  - Partial-thickness flap—also called a *mucosal* or *split-thickness flap*; the incision stops short of the periosteum. Using a scalpel blade for sharp dissection, the mucosa (epithelium with some connective tissue) is elevated off the underlying connective tissue and periosteum. No bone is exposed. The partial-thickness flap is indicated when the flap is to be positioned apically, or when exposure of bone is not desired (when the crestal bone margin is thin, or when dehiscences or fenestrations are present).
- Based on placement of the flap after surgery:
  - Undisplaced flap—the flap is placed and sutured in its original position.
  - Displaced flap—flaps are placed apically, coronally, or laterally to their original position. Both full-thickness and partial-thickness flaps can be displaced. (Note: Palatal flaps cannot be displaced due to the absence of a mucogingival junction and mobile elastic tissue.)
- Based on management of the papilla:
  - Conventional flap—the papilla may be thinned or split beneath the contact point while placing the incision (before flap elevation).
  - Papilla preservation flap—in regenerative therapy and in aesthetic cases, the papilla preservation technique, which retains the entire papilla, is favored. This requires an adequate width of interdental space to allow the intact papilla to be reflected with one side (either the facial or lingual-palatal side) of the flap.

TABLE 33.1 Comparison of Gingivectomy and Flap Surgery Procedures for Pocket Reduction Therapy

	Gingivectomy	Flap Surgery
Access for root instrumentation	Adequate	Very good
Pocket reduction	Achieved by resection of suprabony soft tissue pocket	Achieved by soft tissue resection, osseous resection, or periodontal regeneration
Type of pocket treated	Pseudopocket	Gingival (pseudo) and periodontal (true) pockets
Postoperative bleeding	Often present as mild oozing	Minimal
Postoperative healing and comfort	Healing is by secondary intention; significant discomfort is experienced.	Healing is by primary intention and hence minimal discomfort is experienced.
Feasibility of performing osseous surgery to treat bony irregularities and defects	Not possible	Possible
Commonly used first (submarginal) incision	External bevel incision (beveled bleeding surface of gingiva faces away from tooth surface)	Internal bevel incision (beveled bleeding surface of gingiva faces tooth surface)
Implication for keratinized gingiva	Preservation of existing keratinized gingiva not possible	Existing width of keratinized gingiva can be preserved



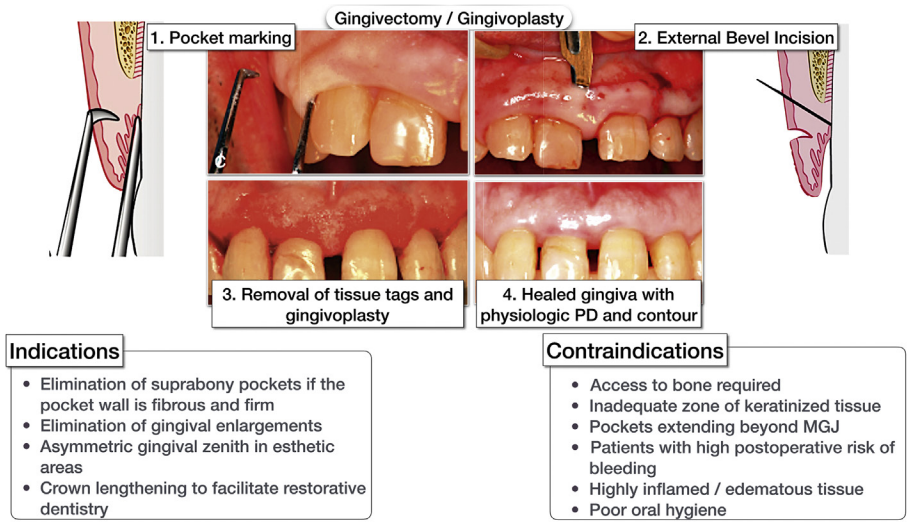
• **Fig. 33.1** Incisions Used in Periodontal Surgery. Incisions can be horizontal (running in the mesio-distal direction) or vertical (running in the occluso-apical direction). Horizontal incisions that are scalloped allow the interdental bone to be covered once the flap is adapted back over the bone and tooth surface. This enhances patients' comfort and allows for faster closure of the wound due to healing by primary intention; hence scalloped incisions are usually preferred. Vertical incisions or oblique releasing incisions should neither split a papilla at the center nor be placed over a root prominence; they should be placed at tooth line angles so they may completely include or exclude the interdental papilla in the flap design. Vertical incisions must extend beyond the mucogingival junction to reach the alveolar mucosa to allow for flap displacement coronally, apically, or laterally. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

Incisions

The #15, #15C, and #12 surgical blades are used most often to make incisions during periodontal surgery. Fig. 33.1 shows various types of incisions that may be used in flap surgery and gingivectomy.

Gingival Surgery

Periodontal surgery limited to the gingival tissues only without the use of periodontal flaps includes gingival curettage, excisional new attachment procedure (ENAP), gingivectomy, and gingivoplasty:



• **Fig. 33.2** Gingivectomy and Gingivoplasty. The major reasons to perform gingivectomy/gingivoplasty are to eliminate soft tissue pockets for accessibility for root instrumentation and to establish physiologic gingival contours. The major steps involved in this procedure include pocket marking (using a Crane-Kaplan pocket marking probe), continuous or discontinuous external bevel incisions (using scalpels or gingivectomy knives), removal of excised pocket tissue, irrigation, scaling and root planing of exposed root surfaces if required, removal of tissue tags, and finally placement of periodontal pack/dressing. Complete epithelial repair takes about 1 month, while complete repair of the connective tissue takes about 7 weeks. The reader is referred to Chapter 60 in *Newman and Carranza's Clinical Periodontology* (13th ed.) for a detailed description of the procedure. *MGJ*, mucogingival junction; *PD*, probing depth. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

- **ENAP** (a definitive subgingival curettage procedure performed with a knife) for the sake of pocket reduction therapy is considered obsolete and unnecessary.
- **Gingival curettage** (scraping of the gingival wall of a periodontal pocket to remove the chronically inflamed tissue) is also considered obsolete and unnecessary. A closed procedure, it should not be confused with the use of curettes to eliminate granulation tissue during flap surgery (an open procedure). The reason for the latter is to remove bleeding tissue that obstructs visualization, to allow the necessary examination of the root surface and the bone. Thus the removal of granulation tissue by curettage during surgery is accomplished for technical rather than biologic reasons.
- **Gingivectomy** refers to excision of soft tissue pocket wall for the sake of achieving pocket reduction or elimination.
- **Gingivoplasty** refers to reshaping the gingiva to allow for a physiological contour (also called as 'positive' architecture that shows a gradual rise of interproximal tissue with a gradual fall of labial/lingual tissue). One may perform gingivectomy and gingivoplasty at the same time (Fig. 33.2).

Access Flap Surgery

Periodontal access surgery is an adjunct to nonsurgical periodontal therapy, and should occur only once the patient has demonstrated effective biofilm control. Currently, five different flap techniques are mainly used: the modified Widman flap, the undisplaced flap, the apically displaced flap, the papilla preservation flap, and the distal terminal molar flap. The reader is referred to Chapter 60 of *Newman and Carranza's Clinical Periodontology* (13th ed.) for detailed descriptions of various types of flap procedures.

Table 33.2 reviews the goals accomplished by these procedures.

CASE-BASED LEARNING EXERCISE

Scenario:

A 46-year-old male presented for resective osseous surgery. He had been seen earlier for initial evaluation followed by four quadrants of scaling and root planing. At the time of reevaluation, due to the presence of residual deep probing depths in the mandibular right posterior sextant (associated with slight to moderate radiographic horizontal bone loss), a decision was made to pursue resective pocket reduction surgery. As part of the surgery, after making submarginal incisions on the buccal aspect, buccal and lingual full-thickness flaps were reflected (Fig. A).

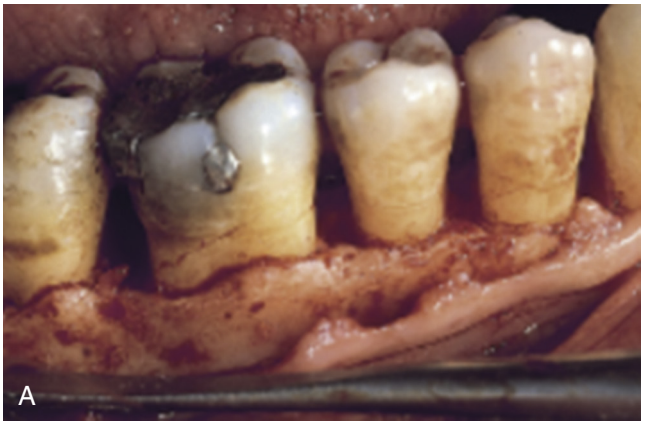


TABLE 33.2 Goals Accomplished by Various Flap Procedures

Goals	MWF	Undisplaced Flap	Apically Displaced Flap			
			Full thickness	Partial Thickness	Distal Wedge	PPF
Root surface access	✔	✔	✔	✔	✔	✔
Pocket reduction at the time of surgery		✔	✔	✔	✔	
Ability to address osseous defects (resection/ regeneration)		✔	✔		✔	✔

MWF, modified Widman flap; PPF, papilla preservation flap.

Questions

1 Based on the figure, what type of bone architecture is visible?
- **a.** Positive
- **b.** Negative
- **c.** Flat
- **d.** None of the above

2. When performing buccal flap reflection in the vicinity of mandibular molars, what anatomic structure should be avoided?
- **a.** Nasopalatine foramen
- **b.** Apical foramen
- **c.** Mental foramen
- **d.** Greater palatine foramen

3. The process of removing supporting alveolar bone during resective osseous surgery is called:
- **a.** Osteoplasty.
- **b.** Ostectomy.
- **c.** Alveolectomy.
- **d.** Enameloplasty.

Clinical photo from Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

This chapter was developed from Chapters 60 and 61 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: c

Explanation: The crestal bone on the interproximal areas is almost at the same level as buccal bone levels, making it a flat architecture.

2. Answer: c

Explanation: Mental foramen is located near the apices of mandibular premolars on the buccal aspect. While reflecting the buccal flap at this location, care should be taken to avoid this foramen and its contents.

3. Answer: b

Explanation: Osteoplasty involves reshaping of bone without the removal of supporting alveolar bone. Alveolectomy is not an accepted terminology; enameloplasty, as the name suggests, is performed on enamel and not on bone.

34

Pocket Reduction Surgery: Regenerative Approach

Relevant Terminology

Terminology/ Abbreviation	Explanation
allograft	Graft obtained from a different donor of the same species (i.e., a human donor for humans).
alloplast	Synthetic bone substitute manufactured to chemically resemble hydroxyapatite and bone minerals.
ankylosis	Denotes a repair phenomenon when tissue continuity is re-established during wound healing by direct bone -to- root attachment without intervening PDL tissue.
autograft	Graft obtained from the patient's own bone. Can be procured from either extraoral or intraoral sites.
biomodification of root surface	Topical use of chemical agents (e.g., tetracycline) on the prepared root surface with the intent of removing local factors that may interfere with new attachment or preferentially encouraging connective tissue adhesion/attachment.
curettage	Removal of the sulcular epithelium using a curette. It is very similar to root planing, except that the blade of the instrument is directed toward the soft tissue wall of the pocket rather than the root surface. This procedure is now considered obsolete and unnecessary because when the root is thoroughly scaled and planed, and the biofilm and calculus removed, the inflammation in the tissue automatically resolves without the need for tissue curettage.
enamel matrix derivative	Biologic mediator composed of enamel matrix proteins, effective in the treatment of intrabony defects with histologic evidence of regeneration.
guided tissue regeneration	Placement of a barrier membrane over the intrabony defect; used for the prevention of epithelial migration along the cemental wall of the pocket and for maintaining space to allow clot stabilization.
laser-assisted new attachment procedure (LANAP)	Use of a neodymium-doped yttrium aluminum garnet (Nd:YAG) laser with a potential to lead to periodontal regeneration. The role of lasers in periodontal therapy remains controversial and lacks scientific validation.
long junctional epithelium (LJE)	A form of periodontal healing by repair, characterized by epithelial attachment on a previously contaminated root surface.
new attachment	Healing by replacement with epithelial and/or connective tissue that matures into various nonfunctional types of scar tissue.
osteoconduction	A property of grafts and biomaterials that passively supports the formation of bone on their surfaces.
osteogenicity	A property of autologous grafts to directly contribute to new bone formation; attributed to the presence of living cells capable of osteogenesis within the graft.
osteoinduction	A property of grafts that contribute to new bone formation by facilitating the recruitment of progenitor cells and the stimulation of these cells to develop into pre-osteoblasts. Only autologous grafts and certain allografts are osteoinductive.
radiographic bonefill	Radiographic description of treated periodontal defect that shows signs of bone tissue formation within the defect. (Note: this term does not commit to histological evidence of true periodontal regeneration)'

Relevant Terminology—cont'd

Terminology/ Abbreviation	Explanation
recombinant human platelet-derived growth factor (rhPDGF)	A potent mitogenic and chemotactic factor that has been successfully applied in the regenerative treatment of human intrabony defects.
regeneration	Healing occurs through the reconstitution of a new periodontium; this involves the formation of alveolar bone, functionally aligned periodontal ligament, and new cementum on a previously diseased root surface.
xenograft	Graft obtained from a different species. Most frequently of bovine or porcine origin.

Fast Facts

Rationale for periodontal regenerative therapy	Intrabony and furcation defects are sequelae of periodontal disease. Some of these defects are amenable to regeneration using approaches that include bone replacement grafts, guided tissue regeneration (GTR), or a combination of both.
Regenerative biomaterials	Bone graft substitutes: • Autologous bone (e.g., osseous coagulum) • Allograft (e.g., freeze-dried bone allografts) • Xenografts (e.g., bovine bone grafts) • Alloplasts (e.g., ceramics) Membranes: • Resorbable (e.g., collagen membrane) • Nonresorbable (e.g., expanded polytetrafluoroethylene membrane) Biologics: • Enamel Matrix Derivative • Recombinant Human Platelet-Derived Growth Factor
Flap management for regenerative procedures	The intent of regenerative surgical approaches is to regenerate the components of the periodontium that were lost due to periodontitis; flap management is as conservative as possible to retain soft tissues that will aid in supporting the intended regeneration. Hence sulcular or marginal incisions are performed (in contrast with resective procedures, where the use of submarginal scalloped incisions is more common).
Defect management	The most common form of regenerative periodontal procedure entails the placement of bone grafts, which act as scaffolds for healing, in combination with barrier membranes that prevent epithelial migration and enable periodontal connective tissue and bone cells to gain access to the defect area and regenerate the components of the periodontium.
Treatment considerations	• Passive flap closure for encasement of the graft materials • Flap design to allow tension-free suture placement • A deeper defect is correlated with increased clinical attachment level gain and reduction in probing depth • Narrow, circumferential defects with a three- or two-walled configuration are more favorable than wide, one-walled defects • Presurgical splinting in case of tooth mobility • Endodontic treatment should be carried out prior to regenerative treatment
Evaluation of results	Following surgery, the area should not be probed for 6 months. Radiographic evidence of bone fill is usually present after 6 months and should continue over the course of 1 year.
Assessment of periodontal wound healing	Accomplished by: 1. Histology 2. Clinical methods 3. Imaging methods 4. Surgical reentry

Core Knowledge

Introduction

Traditional methods of pocket reduction therapy include surgical debridement and resective procedures. Although clinical parameters such as bleeding on probing, probing depth, and clinical attachment loss show improvement, the regeneration of lost periodontal structures is minimal and not predictable in the resective approach. Two well-documented techniques of periodontal regeneration are osseous grafting (bone grafting) and guided tissue regeneration (GTR). This chapter reviews the biologic basis and clinical applicability of these regenerative approaches.

Periodontal Wound Healing

Table 34.1 reviews the complex topic of periodontal wound healing in a simplified manner and compares the possible responses of all periodontal tissues during healing after pocket reduction therapy.

The Biologic Basis of Various Regenerative Modalities

Periodontal regeneration refers to the complete reconstitution of functional supporting tissues, which includes new alveolar bone, cementum, and the periodontal ligament.[1] Several options exist for performing periodontal regenerative surgery. Table 34.2 reviews the biologic basis of various regenerative treatment modalities.

Guided Tissue Regeneration

The GTR technique is used for the prevention of epithelial migration along the cemental wall of the pocket, and for maintaining space for clot stabilization. By preventing the fast migrating epithelial and gingival connective tissues from 'prematurely' occupying the wound space under the periodontal flap, the GTR membrane attempts to 'guide' precursor cells from the bone and ligament to preferentially occupy this same wound space. The prevention of epithelial downgrowth into the wound, and exclusion of gingival connective tissue is achieved using predominantly barrier techniques. This allows cells with regenerative potential from the periodontal ligament (PDL) and bone to repopulate the wound first, thereby contributing to regeneration of ligament fibers and bone. Fig. 34.1 reviews this procedure, in which bone grafting is combined with the use of barrier membrane.

Bone Grafting

The use of graft materials was once thought to provide a regenerative inductive effect; however, current opinion is that it should be viewed primarily as providing a scaffold for healing. In addition to bone graft materials, many non-bone graft materials have been used in an attempt to restore the periodontium. The selection of graft materials depends on the individual clinician's preference and expertise, the nature of the intrabony defect (e.g., furcation versus intrabony defect), and finally the type of healing sought (i.e., regeneration, new attachment, or repair). Table 34.3 reviews the various types of bone grafting materials used for regenerative approaches in pocket reduction therapy.

TABLE 34.1 Healing Periodontal Tissues: Histologic Outcomes Following Pocket Reduction Therapy

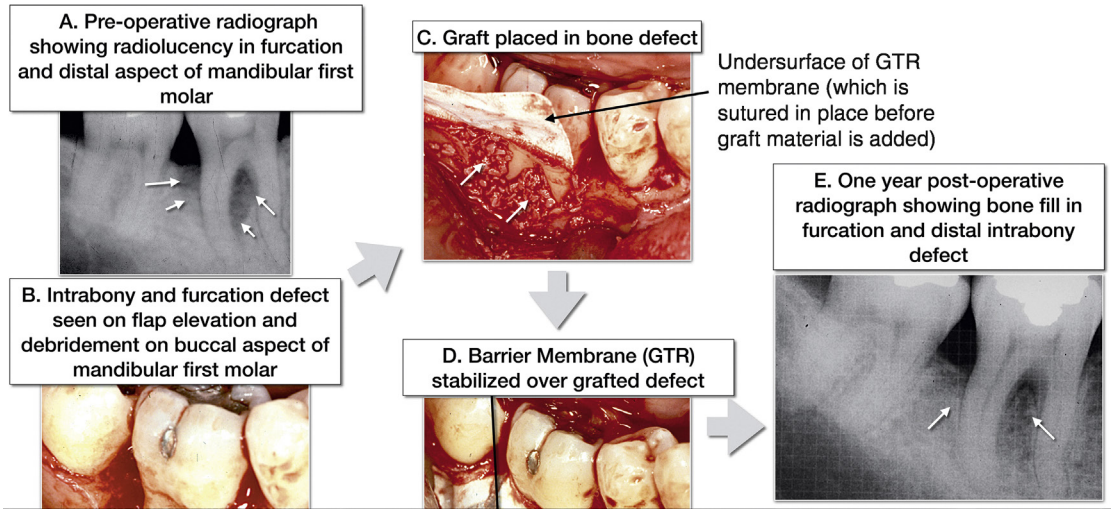
	Gingival Epithelium	Gingival Connective Tissue	Bone	Cementum & PDL
Regeneration	JE is restored to original form (i.e., short and not long JE) and function	Complete regeneration of form and function of connective tissue occurs	Alveolar bone fill occurs	Formation of new acellular extrinsic-fiber cementum with inserted new PDL fibers
Repair	Restoration of tissue continuity in the wound/defect area by LJE	There is no regeneration of form and function of original intact tissues in these tissue components.		
New attachment	Newly formed LJE adheres to previously diseased root surface	Formation of new connective tissue attachment on previously diseased and exposed root surface may occur	Alveolar bone fill may occur	Formation of new cementum with inserted PDL fibers may occur
Reattachment	Epithelial *reattachment* is not possible because tissue continuity is always re-established by new epithelial cells from the basal layer	This occurs in the deeper areas of the pocket. The bond between connective tissue and remaining vital components of the tooth, like cementum and PDL, is reestablished on previously healthy root surface that is unexposed to periodontal disease		

JE, junctional epithelium; LJE, long junctional epithelium; PDL, periodontal ligament.
Some confusion may arise between the terms *regeneration* and *new attachment* because both types of healing involve all periodontal tissue types. Remember:
- Healing by replacement with new epithelial and/or connective tissue that matures into various nonfunctional types of scar tissue is termed *new attachment*.
- Healing by the reconstitution of a new periodontium, which involves the formation of alveolar bone, functionally aligned PDL, and new cementum, is called *regeneration*.

TABLE 34.2 Rationale Behind Various Regenerative Approaches in Pocket Reduction Therapy

Treatment Modality	Rationale
SRP without additional bone grafts or membranes (nonsurgical/closed and surgical/open flap debridement approaches)	• A biocompatible root surface, intentional removal of diseased junctional and pocket epithelium by excision, wound stabilization, and clot protection help in regenerating some bone and connective tissue at the base of the defect • However, new cementum or new functional PDL fibers are not formed, and healing is by formation of long junctional epithelium (e.g., reparative healing after MWF procedure)
Root conditioning	• Root biomodification is thought to bring about surface detoxification, removal of smear layer, and exposure of collagen fibrils that prevent epithelial migration over treated root surfaces, thereby promoting preferential fibroblast cell attachment to root surfaces • This attempt to induce cementogenesis and attachment of collagen fibers has yielded controversial results in humans, relegating this modality to an adjunctive regenerative procedure (e.g., root biomodification using citric acid, tetracycline, fibronectin)
Bone grafts	• Various bone grafting materials are available for use in bony defects, and result in decreased probing depths and improved attachment levels • Bone grafting does not result in regeneration of ***all*** periodontal tissues; rather, it acts as a space maintainer or scaffold for bone regeneration • Healing is by LJE formation when only bone grafts are used for defect filling. This is also considered a therapeutic success due to improvement in clinical parameters like BOP, PD, and CAL
Barrier membranes: GTR	• GTR consists of placing barriers of different types (membranes) to cover the bone and PDL, temporarily separating them from the gingival epithelium and connective tissue • This regenerative method is based on the assumption that excluding the epithelium and gingival connective tissue from the root surface during the postsurgical healing phase not only prevents epithelial migration into the wound but also favors repopulation of the area by cells from the PDL and bone. • When sufficient time is given for deeper periodontal tissues to repopulate the clot under a membrane, healing by new periodontal connective tissue attachment and bone fill (without an intervening LJE) can potentially occur, making this modality one of the better choices for regenerative pocket reduction therapy
Tissue engineering	• Here, manipulation of the wound healing process usually involves one or more of the three key elements: 1. signaling molecules (e.g., growth factors like rhPDGF-BB, biologic modifiers like EMD) 2. scaffold or supporting matrices (eg. β-TCP) 3. cells • These three elements "intertwine" with each other and stimulate regeneration using a different mechanism from the *bioexclusion* principle, as demonstrated with the use of GTR membranes • The use of biologic agents can stimulate recruitment of stem cells to the intrabony or furcation defect site, and their proliferation and differentiation into a newly regenerated periodontal apparatus
Combined techniques	• It is logical to assume that combining the various modalities described above can enhance the regenerative potential of the periodontium. To this end, various combinations are currently being researched • It is important to note that patient selection (general health and attitude), case selection (defect characteristics), treatment modality, choice of material, and surgeon's training and experience all play a vital role in choosing the correct combination of regenerative modalities for a particular case

BOP, bleeding on probing; β-TCP, beta tricalcium phosphate; CAL, clinical attachment loss; EMD, enamel matrix derivative; GTR, guided tissue regeneration; LJE, long junctional epithelium; MWF, modified Widman flap; PD, probing depth; PDL, periodontal ligament; rhPDGF, recombinant human platelet-derived growth factor; SRP, scaling and root planing.



• **Fig. 34.1** Guided Tissue Regeneration in Combination With Bone Grafting.[2] The main steps in the guided tissue regeneration (GTR) procedure, include elevating a full-thickness flap, defect debridement, trimming and adapting a membrane over the bony defect such that the membrane extends 2–3 mm beyond defect margins, optional placement of graft material or biologic modifier in the defect to support the membrane, membrane stabilization using mainly sutures to achieve stability, and flap closure using sutures. GTR procedures are equally effective when using resorbable (e.g., collagen membrane) and non-resorbable (e.g., titanium-reinforced expanded polytetrafluoroethylene) membranes.

Objectives of using a barrier membrane in the GTR procedure:

- Gingival epithelial and connective tissue exclusion
- Cell/tissue repopulation control
- Space maintenance
- Clot stabilization in wound space

Indications for GTR:

- Narrow two- or three-wall intrabony defects
- Circumferential / moat-shaped defects
- Class II molar furcations (especially mandibular molar)
- Recession defects (not a part of pocket reduction therapy, but a mucogingival procedure)

Contraindications for GTR:

- Poor oral hygiene
- Heavy smoking
- Width of attached gingiva ≤ 1 mm
- Generalized horizontal bone loss
- Intrabony defects < 4 mm deep.

(From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

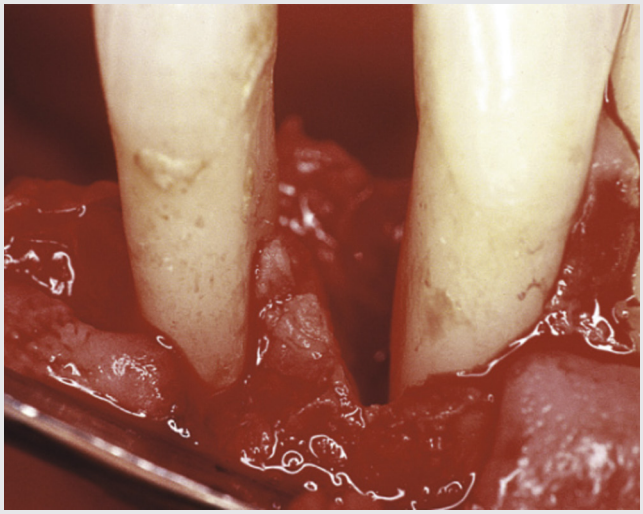
TABLE 34.3 Bone Graft and Bone Replacement Materials

	Autograft	Allograft	Xenograft	Alloplast
Description	Bone obtained from the same individual	Bone obtained from a different individual of the same species from commercial tissue banks	Bone obtained from a different species from which all cellular and protein material has been removed	Synthetic, biocompatible, and sometimes bioactive bone substitute
Examples	• Intraoral source: Osseous coagulum bone blend • Extraoral source: Cancellous bone marrow from iliac crest	• Demineralized freeze-dried bone allograft (DFDBA) • Freeze-dried bone allograft (FDBA)	• Bovine derived hydroxyapatite • Porcine derived • Equine derived	• **Organic:** Dentin, cementum, collagen, corals • **Inorganic:** Plaster of Paris, bioceramics (hydroxyapatite, tricalcium phosphate), bioactive glass, polymers (PMMA/HEMA)
Role in bone regeneration	Osteogenic Osteoinductive Osteoconductive	Osteoconductive (DFDBA–possibly osteoinductive)	Osteoconductive	Osteoconductive

PMMA/HEMA, polymethylmethacrylate/hydroxyethylmethacrylate.
Osteoconductive: the ability of a graft material to provide a scaffold or passive matrix for bone formation.
Osteoinductive: the ability of a graft material to promote recruitment of immature cells and the stimulation of these cells to develop into preosteoblasts (bone-forming cells); that is, the graft stimulates cells from outside grafted material to form bone.
Osteogenic: the inherent ability of a graft material to form bone by itself; that is, cells within the graft form bone.

CASE-BASED LEARNING EXERCISE

Scenario: A 65-year-old male presented to the periodontics clinic for pocket reduction surgery. Prior to this appointment, the patient had been seen for reevaluation following scaling and root planing. He was a nonsmoker with no known systemic conditions. During reevaluation, it became clear that the majority of sites improved, but with localized areas of residual deep probing depths of 6 mm or more. One such site was the distal aspect of the mandibular right canine. Radiographic evaluation of this site revealed a distal vertical defect, and clinically, the tooth had physiological mobility. At the time of surgery, buccal and lingual flaps were reflected and granulation tissue present in the bony defect (distal to canine) was removed.



Questions

1. Which term most closely describes the morphology of the bony defect (seen distal of canine) in Fig. A?
   a. Angular defect
   b. Horizontal defect
   c. Incipient defect
   d. Crater defect
2. Which approach would be better in this situation?
   a. Resective
   b. Regenerative
3. Which of the following is true?
   a. The number of bony walls has no effect on regenerative outcomes.

b. Tooth mobility has no impact on regenerative outcomes.
c. The number of bony walls directly affects regenerative outcomes.
d. Smoking has no effect on regenerative outcomes.

4. The use of barrier membrane in guided tissue regeneration allows for epithelium exclusion and _____
a. Antiinflammation
b. Space maintenance
c. Blood vessel exclusion

Clinical photo attribution: Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

This chapter was developed from Chapter 63 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

CLINICAL CORRELATE

What is the difference between "regeneration" and "bone fill" in periodontal regenerative therapy?

- Regeneration is the restoration of both structure and function of the lost native tissue by newer tissue. Histology is the gold standard to validate regeneration.
- Bone fill describes the clinical restoration of bone tissue in a treated periodontal defect. However, the term does not give any idea about the presence or absence of epithelial or connective tissue *new attachment* (i.e., the union of new epithelium or connective tissue with a previously diseased root surface which was deprived of its original periodontal ligament) or *reattachment* (reunion of connective tissue with a root surface on that viable periodontal ligament tissue is present).[2] Bone fill may be confirmed clinically or radiographically.
  - *Radiographic bone fill* is the term used to denote positive radiographic changes following regenerative therapy. Unlike histology, radiographs cannot delineate regeneration from repair.
  - Surgical reentry, a process that involves reflecting a flap to evaluate the outcome, is another method employed, but again cannot delineate regeneration from repair. In this case, any positive improvement is called "*clinical bone fill.*"

CLINICAL CORRELATE

What are the roles of bone grafting materials and substitutes?

Roles of bone grafts and substitutes include:

- Active formation of new bone (osteogenesis) if cells are present
- Induction of bone formation (osteoinduction)
- Creating a passive scaffold for bone formation (osteoconduction)
- Providing mechanical obstruction for epithelial cells to occupy wound site (space maintenance and contact inhibition)
- Acting as a carrier vehicle for biologic mediators (e.g., growth factors)

Solutions

1. Answer: a
Explanation: Due to the sloping nature of the bony defect, this will be classified as an angular defect.
2. Answer: b
Explanation: Since this angular defect is well contained, a regenerative approach would be preferable.
3. Answer: c
Explanation: The greater the number of bony walls, the better the prognosis; the higher number supplies more bone regenerative cells and aids in graft containment.
4. Answer: b
Explanation: In addition to preventing epithelial cells from entering the defect, space maintenance is another important function of the barrier membrane.

References

1. Rosenberg, E., & Rose, L. F. (1998). Biologic and clinical considerations for autografts and allografts in periodontal regeneration therapy. *Dental Clinics of North America*, *42*(3), 467–490.
2. Wang, H. L., & Cooke, J. (2005). Periodontal regeneration techniques for treatment of periodontal diseases. *Dental Clinics of North America*, *49*(3), 637–659.

35 Management of Furcation Involvement

Relevant Terminology

Terminology/Abbreviation	Explanation
accessory pulp canals	Channels located in the furcation area that lead to the dental pulp; sometimes hypothesized to link pulpal and periodontal disease processes at the furcation involved site.
cervical enamel projections (CEP)	Developmental anomaly characterized by extension of the enamel toward the furcation entrance. They hinder plaque removal, eventually leading to furcation involvement. More common in maxillary and mandibular second molars.
CEP classification	Masters and Hoskins (1964) classified CEP into three grades:[1] • Grade I—CEP extending from cementoenamel junction toward furcation • Grade II—CEP approaching the entrance to the furcation • Grade III—CEP extending horizontally and entering into the furcation
enamel pearls	Ectopic globules of enamel located at the furcation site of molar roots that contribute to plaque retention; they are devoid of soft tissue attachment and associated with furcation involvement.
furcation arrow	Radiographically, mesial or distal furcation involvement of the maxillary molar that appears as a radiolucent triangle.
furcation	Area between roots of a multirooted tooth
furcation entrance	Point where undivided root trunk meets root cones
fornix	Roof of furcation
hemisection	If a mandibular molar is sectioned and crowned separately as two premolars instead of resecting and removing one root, the process is called hemisection or premolarization / bicuspidization. In order to perform root resection or hemisection, the tooth has to be endodontically treated first.
intermediate bifurcation ridges	Ridges that are primarily cementum in nature; common in mandibular molars. They run from the mesial surface of the distal root, via the bifurcation, and end high on the mesial root. Like CEP and enamel pearls, they hinder plaque removal.
regenerative approach	Process of regaining lost attachment by regenerating periodontal tissues at the furcation site using techniques such as guided tissue regeneration.
resective approach	Process of eliminating furcation or minimizing furcation deleterious effects of defects by reshaping or recontouring the existing bone in and around the furcation.
root complex	Consists of root trunk and root cones
root cone	Portion of root apical to furcation entrance upto root apex
root divergence	Distance between root cones
root resection / root amputation	In some cases, in a tooth with furcation involvement, removal of one root will improve the prognosis and longevity of that tooth; that procedure is called root resection.
root trunk	Portion of undivided root between CEJ and furcation
tunnel preparation	In some cases of advanced furcation involvement in mandibular molar tooth, the furcation is intentionally made through and through and exposed to the oral cavity to enhance the hygienic maintenance of the site. This is called “tunnel” preparation.

Fast Facts

Furcation involvement and periodontal disease progression	Furcation involvement in a multirooted tooth leads to the accumulation of dental plaque. The anatomy of the furcation makes it harder for the patient and clinician to efficiently remove plaque and calculus (by nonsurgical means), leading to progression of periodontal disease.
Furcation and tooth loss	Furcation involved teeth are more likely to be lost than non–furcation involved teeth.
Etiology of furcation involvement	Host response to dental plaque microorganisms and their by-products results in bone loss at the furcation site. A variety of local factors contribute to plaque retention.
Furcation entrance (FE) from cementoenamel junction (CEJ)	On average, FEs in maxillary molars are located 3.6, 4.2, and 4.8 mm apical to the CEJ on the mesial, facial, and distal sites, respectively.[2] In the maxillary first premolar, FE is located on average 7.9 mm from the CEJ.[3]
Local anatomic factors that influence furcation involvement	Root trunk length, root morphology, interroot distance, and local anatomic anomalies (e.g., CEPs, intermediate bifurcation ridges). Shorter root trunk length and presence of anomalies increases the probability of furcation involvement.
FE and cleaning	Studies identified the FE diameter to be < 1 mm, making it difficult for instruments (curettes) to enter and remove plaque and calculus from the furcation effectively. Using a narrow ultrasonic tip or a special curette such as DeMarco curettes and Mini Five Gracey Curettes. See Chapter 50 of Newman and Carranza's Clinical Periodontology 13th edition textbook for a discussion on these instruments.

Core Knowledge

Introduction

Involvement of the furcation area of multirooted teeth (maxillary first premolar, maxillary, and mandibular molars) by periodontal disease presents both prognostic and therapeutic dilemmas. This is because the morphology of the multirooted tooth presents unique challenges in periodontal therapy. This chapter reviews the major considerations for management of the periodontally involved furcation.

Diagnosis and Classification of Furcations

The diagnosis of furcation defects is based mainly on:
- Periodontal probing using a Nabers probe;
- Dental radiographs (periapical, bite-wing, panoramic).

Fig. 35.1 shows the classification of furcation defects.

CLINICAL CORRELATE

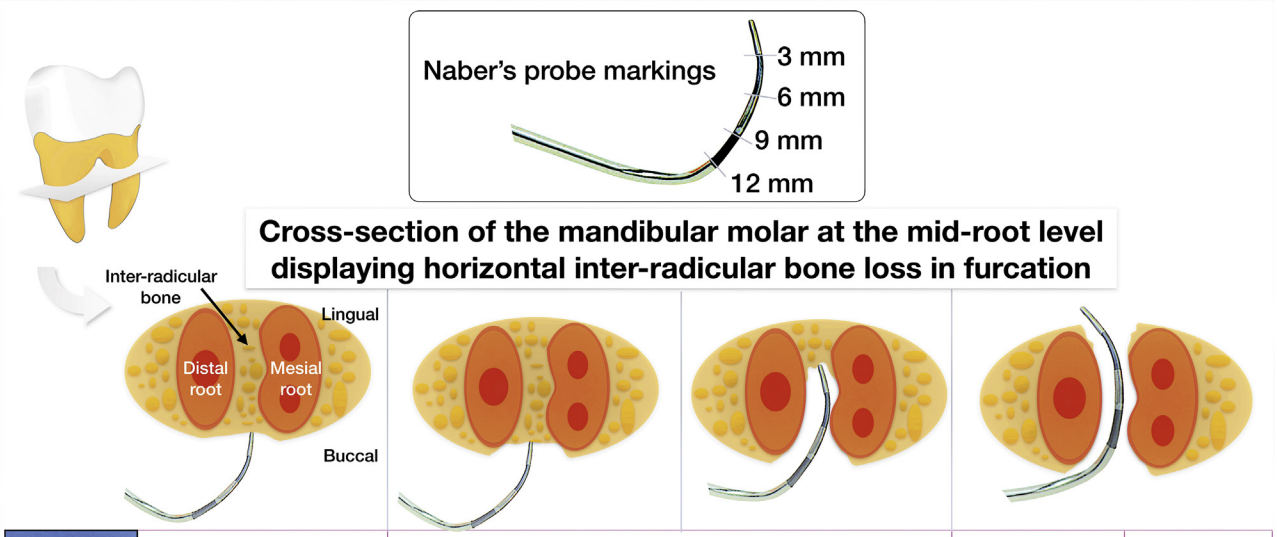
What are the unique challenges to periodontal diagnosis and therapy presented by furcation defects?

Diagnostic challenges:[4]
- Initial furcation involvement can go undetected in radiographs. Even moderately severe lesions may be hidden due to superimposition of structures (especially roots), and only slight alterations in trabecular radiodensity may be present as subtle clues indicating their presence.
- Initial furcation involvement usually depends on periodontal probing for detection. Entry of the probe depends, however, on the absence of obstructing soft tissue, the width of osseous defects, root divergence, furcation ridges, etc. Consequently, furcations (especially maxillary distal furcations) may not be accessible to probing if they are too narrow or tortuous, leading to *underdiagnosis.*

Therapeutic challenges:[4]
- Unlike intraosseous defects, which have mostly osseous walls bordering them, the majority of walls in a furcation defect are actually nonosseous in nature (i.e., the root surfaces and the furcation dome). These are covered usually by cementum, sometimes dentin or even enamel (in the case of enamel pearls and cervical enamel projections). This reduced area of vascularity with its limited source of bone precursor cells is a challenge for periodontal regenerative therapy.
- Cervical enamel projections have shown great complexity in recent electron microscopic evaluations. The complexities exist in the form of pouch-like openings which can house oral biofilms that may resist even the most stringent of oral hygiene measures, thus contributing to the progression of furcation defects.
- Sometimes the furcation fornix is so narrow that once inflammatory periodontal disease exposes it to the oral environment, it becomes virtually impossible for the patient to maintain oral hygiene in the narrow niche. Professional debridement can also be extremely challenging due to lack of proper access into the furcation area.

Etiology of Furcation Involvement in Periodontal Disease

When attachment and bone loss progress apically in inflammatory periodontal disease, they can eventually involve the furcations of multirooted teeth. The various cofactors that predispose toward involvement of furcations in periodontal disease include:
- **Local anatomic factors**—enamel pearls, cervical enamel projections (CEPs), root concavities in furcal areas
- **Trauma from occlusion**
- **Endodontic-periodontal disease**—patent accessory and lateral canals opening from the pulp into the periodontal ligament space can cause infection and inflammation of



Glickman, 1953	**Grade I:** Pocket formation involves the furcation flute with intact interradicular bone	**Grade II:** Loss of inter-radicular bone of varying depths into furcation, but not completely probeable to opposite side of tooth.		**Grade III:** Complete loss of inter-radicular bone	**Grade IV:** Same as grade III with gingival recession
Hamp et al, 1975	-	**Degree I:** Horizontal PD < 3mm	**Degree II:** Horizontal PD > 3mm but not probeable through and through	**Degree III:** Through and through bone loss	

• **Fig. 35.1** Classification of Furcation Defects. Several classification systems exist for furcation defects, most of which are based on horizontal probing depth into the furcation. (Note: horizontal bone loss in a furcation is bucco-lingual or mesio-distal in direction; horizontal bone loss everywhere else is occluso-apical in direction.) This figure discusses two popular classification systems: Glickman's classification[5] and the Hamp, Nyman, and Lindhe classification.[6] In addition to the Hamp *et al.* horizontal classification system, Tarnow and Fletcher's subclassification was proposed to measure 'vertical' probing depth (probing performed in the occluso-apical direction) from the roof of the involved furcation.[7]

The subclasses they proposed are:

- A—probeable vertical depth of 1–3 mm
- B—probeable vertical depth of 4–6 mm
- C—probeable vertical depth of 7 mm or more

Furcations would thus be classified as IA, IB, and IC; IIA, IIB, and IIC; and IIIA, IIIB, and IIIC
These subclassifications aid in prognosis and treatment planning
PD, probing depth. From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier

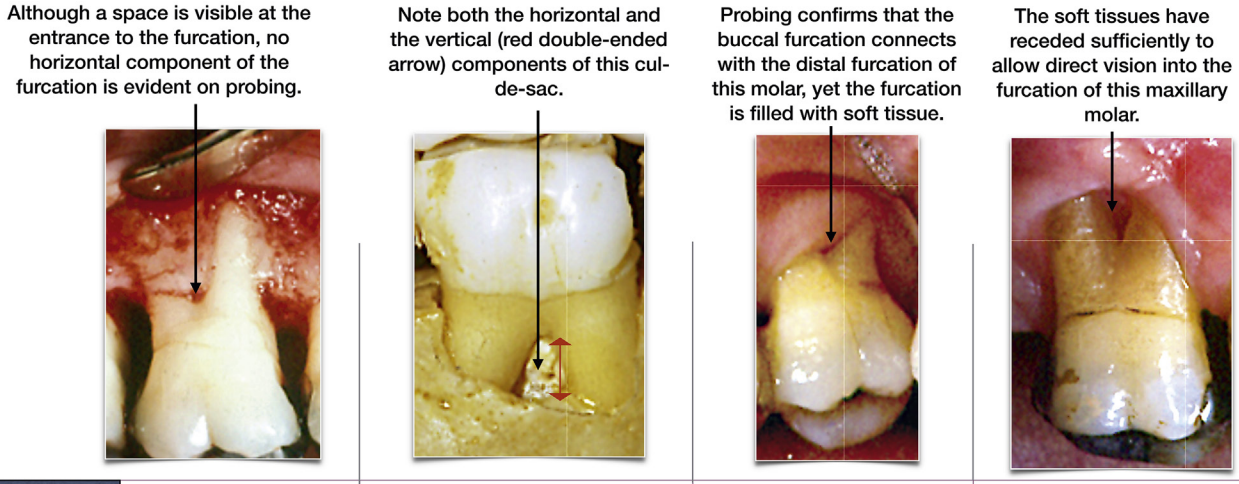
pulpal origin within the periodontium (see Chapter 26 for details on management of such lesions)

- **Root fractures extending into furcations**—usually results in rapid localized alveolar bone loss within furcations
- **Iatrogenic cofactors**—endodontic perforations, overhanging restorations, violation of biologic width while placing the finishing lines for restorations, etc.

Treatment Considerations in Furcation Management

The popular classification systems and related treatment considerations are listed in Fig. 35.2. However, classification systems, though generally a guide for therapy, need to be bolstered by an assessment of the following factors for correct prognosis and treatment planning:

1. **Root divergence**—closely approximated or fused roots can preclude adequate instrumentation during scaling, root planing, and surgery. Teeth with widely separated roots present more treatment options and are more readily treated.
2. **Root trunk length** (distance between cementoenamel junction and furcation area)—once the furcation is exposed, teeth with short root trunks may be more accessible to maintenance procedures.
3. **Root length and amount of remaining bone support**—teeth with long root trunks and short roots may have lost most of their support by the time the furcation becomes involved, affecting the prognosis of any treatment modality used.
4. **Root proximity to adjacent teeth**—this presents the same problems as inadequate root divergence.
5. **Pattern of bone loss**—the treatment response in deep, multiwalled bony defects is different from that in areas of horizontal bone loss. Complex multiwalled defects with deep, interradicular vertical components may be candidates for regenerative therapies.



Glickman	Grade I	Grade II	Grade III	Grade IV
Hamp et al	-	Degree I	Degree II	Degree III
Therapeutic classification	Early defects	Moderate defects	Advanced defects	
Treatment recommended	• SRP • Odontoplasty • Gingivectomy	• Odontoplasty • Osteoplasty • Ostectomy	• Periodontal flap surgery: odontoplasty, osteoplasty, grafting, GTR, root resection, hemisection, tunnel preparation. • Extraction and placement of Implants	

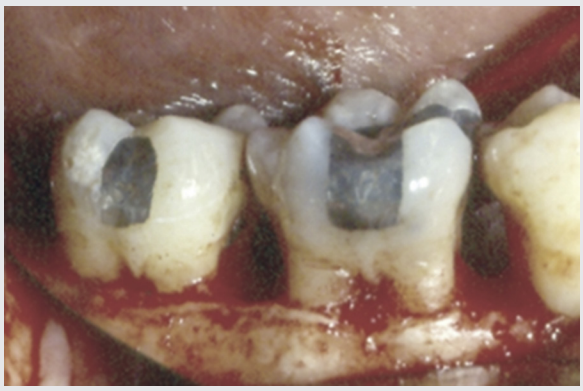
• **Fig. 35.2** Treatment Considerations in the Management of Furcation Defects. The objectives of furcation therapy are to facilitate maintenance, prevent further attachment loss, and obliterate furcation defects as a periodontal maintenance problem.

Therapeutic Classes of Furcation Defects:

- Early defects—incipient or early furcation defects are amenable to conservative periodontal therapy. Any thick overhanging margins of restorations, facial grooves, or CEPs should be eliminated by odontoplasty, recontouring, etc. The resolution of inflammation and subsequent repair of the periodontal ligament and bone are usually sufficient to restore periodontal health.
- Moderate (early Grade II) defects—once a horizontal component to the furcation has developed (class *II / cul de sac*), therapy becomes more complicated. Shallow horizontal involvement without significant vertical bone loss usually responds favorably to localized flap procedures with odontoplasty, osteoplasty, and ostectomy.
- Advanced (late Grade II, III and IV) defects—the development of a significant horizontal component to one or more furcations of a multirooted tooth, or of a deep vertical component to the furca, poses additional problems. Nonsurgical treatment is usually ineffective because the ability to instrument the tooth surfaces adequately is compromised. Periodontal surgery (resective/regenerative), endodontic therapy, and adjunctive procedures like hemisection, root resection, or tunnel preparation may be required. Regenerative approaches such as GTR are primarily indicated in Grade II/degree II furcation defects of mandibular molars. Sometimes the treatment is more predictable when extraction and dental implant placement are chosen. *CEP,* cervical enamel projection; *GTR,* guided tissue regeneration; *SRP,* scaling and root planing. From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

CASE-BASED LEARNING EXERCISE

Scenario: A 47-year-old male presented to the periodontics clinic for pocket reduction surgery. Prior to this appointment, he had been seen for reevaluation following scaling and root planing. The patient was a nonsmoker with no known systemic conditions. During reevaluation, it became clear that the majority of sites had improved, but with localized areas of residual deep probing depths of 6 mm or more with furcation involvements in mandibular molars. Radiographic evaluation revealed involvement of furcations. At the time of surgery, buccal and lingual flaps were reflected, which revealed the unusual (pointed) extension of enamel into furcation sites on both the molars



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Questions

1. What terminology is used to describe the enamel extension?
   - **a.** Cementoenamel projection
   - **b.** Cervical enamel projection
   - **c.** Cervical enamel junction
   - **d.** Dentinoenamel projection
2. Based on the Masters and Hoskins classification, what grade cervical enamel projection is shown in the clinical photo?
   - **a.** Grade I
   - **b.** Grade II
   - **c.** Grade III
3. Which grade **of** furcation (Glickman classification) in mandibular molars has the best prognosis with regenerative therapy?
   - **a.** Grade I
   - **b.** Grade II
   - **c.** Grade III
   - **d.** Grade IV
4. Removal of cervical enamel projections is important in achieving treatment success in this clinical situation.
   - **a.** True
   - **b.** False

This chapter was developed from Chapter 64 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: b

Explanation: The extensions are called cervical enamel projections and are a local factor that contributes to gingivitis and periodontitis. They are more commonly observed in mandibular second molars.

2. Answer: c

Explanation: Because both cervical enamel projections enter into the furcations, they are classified as grade III projections.

3. Answer: b

Explanation: Grade II furcation allows for good graft containment and favors regeneration better than other furcation grades in mandibular molars.

4. Answer: a

Explanation: At the time of surgery, in order to prevent recurrence, cervical enamel projections should ideally be eliminated by odontoplasty.

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36 Periodontal Plastic and Esthetic Surgery

Relevant Terminology

Terminology	Explanation
acellular dermal matrix	Soft tissue graft alternatives that are allografts (i.e., from a human source), processed to eliminate epidermis and cells; meant for use in surgeries without risk of graft rejection.
active versus passive eruption	Eruption of teeth toward the occlusal plane is active eruption; passive eruption occurs mainly by apical migration of gingiva. When passive eruption is affected (as in delayed passive eruption), it can lead to unesthetic short crowns that may require a crown-lengthening surgical procedure.
black triangle	Loss of interdental papillae, mainly in the esthetic areas. One of the most difficult and unpredictable esthetic problems to address surgically.
creeping attachment	Coronal migration of the gingival margin during the healing period following free autogenous grafting. Up to ~1 mm of creeping attachment can be expected in the 12 months following surgery.[1]
frenectomy versus frenotomy	Complete surgical removal of frenum (including its attachment of underlying bone) is frenectomy; relocation of frenum to a more apical location is frenotomy.
keratinized gingiva	Comprises attached gingiva and free gingival margin. Separated from the alveolar mucosa by the mucogingival junction.
pedicle flaps	Pedicle flaps are not completely detached from the donor site and therefore retain their blood supply (e.g., laterally displaced or coronally displaced flaps).
supracrestal tissue attachment (previously called biologic width)	The circumferential space (in relation to every tooth) between the crestal bone and the cementoenamel junction (clinically averages 2 mm) that provides the space for the junctional epithelium and connective tissue fibers to attach.
tunneling procedure	Procedure in which the recipient bed is prepared by creating a tunnel without detaching and elevating the papillae. The donor tissue is then carefully inserted into the created tunnel and secured in place with sutures.
Vestibuloplasty	Surgical procedure intended to deepen the vestibule; usually done in conjunction with free autogenous grafting technique.

Fast Facts

Periodontal plastic surgical procedures	Include crown lengthening, ridge augmentation, surgical exposure of unerupted teeth, coverage of denuded roots, papillae reconstruction, and esthetic surgical corrections around teeth and implants.
Minimal width of attachment gingiva and gingival health	In patients with optimal oral hygiene, gingival health can be maintained in spite of lack of attached gingiva; however, patients with inadequate oral hygiene will benefit from the presence of attached gingiva and a deeper vestibule.
Papilla fill and underlying bone	When the interdental crestal bone level is ≤5 mm apical to the contact point of the adjacent crowns, papilla will be present 100% of the time.[2]
Shallow vestibular depth	Makes brushing difficult and therefore hinders effective plaque control.

Fast Facts—cont'd

Common causes of gingival recession	Traumatic tooth brushing, periodontal disease, orthodontic tooth movement, and aberrant frenum/muscle attachments.
Potential consequences of gingival recession	Dentinal hypersensitivity, unesthetic appearance, marginal gingivitis (due to avoidance of brushing), susceptibility to noncarious cervical lesions and root caries.
Techniques to increase the band of attached gingiva apical to recession	• Free gingival autografts • Free connective tissue autografts • Autograft substitutes (e.g., acellular dermal matrix) • Apically displaced flap
Techniques to increase the band of attached gingiva coronal to recession (root coverage)	• Free gingival autograft • Free connective tissue autograft • Coronally positioned flap; includes semilunar pedicle (Tarnow method) • Subepithelial connective tissue graft (Langer method) • Laterally (horizontally) positioned pedicle flap • Guided tissue regeneration (GTR) • Pouch and tunnel technique
Palatal neurovascular bundle	On average, the greater palatine neurovascular bundle is located 12 mm from the gingival margin toward the palatal midline. This number ranges from 7 mm (in shallow palate) to 17 mm (in deep palate).[3]
Factors positively influencing success of periodontal plastic surgeries	• Lack of biofilm, calculus, and inflammation at the surgical site • Adequate blood supply to the donor tissue • Anatomy of donor and recipient sites • Grafted tissue stability at the recipient site • Minimal trauma to the surgical site
Tissue engineering products used in periodontal plastic surgeries	• Enamel matrix derivative • Platelet-derived growth factor • Cell therapy (e.g., bilayered cell therapy, human fibroblast–derived dermal substitutes) • GTR (barrier membranes) • Acellular dermal matrix

Core Knowledge

Introduction

Not all periodontal surgical procedures are aimed toward pocket reduction therapy. Sometimes, extra measures are required to ensure the establishment of a healthy periodontal complex that can endure the stresses due to mastication, tooth brushing, trauma from foreign objects (e.g., tongue and lip piercings), tooth preparation for crowns and bridges, orthodontic tooth movement, frenum pull, inflammation and subgingival restorative margins.[4]

Periodontal plastic surgery refers to the *surgical* procedures performed to correct or eliminate anatomic, developmental, or traumatic deformities of the gingiva or alveolar mucosa; for example, increasing the width of attached gingiva, vestibular deepening, frenectomy. Note that *mucogingival therapy* is a broader term that includes *nonsurgical* procedures such as papilla reconstruction by means of orthodontic or restorative therapy.

Periodontal esthetic surgery is defined as surgical procedures performed to enhance esthetics; for example, crown lengthening to correct gingival margin discrepancies, recession coverage procedures, etc.

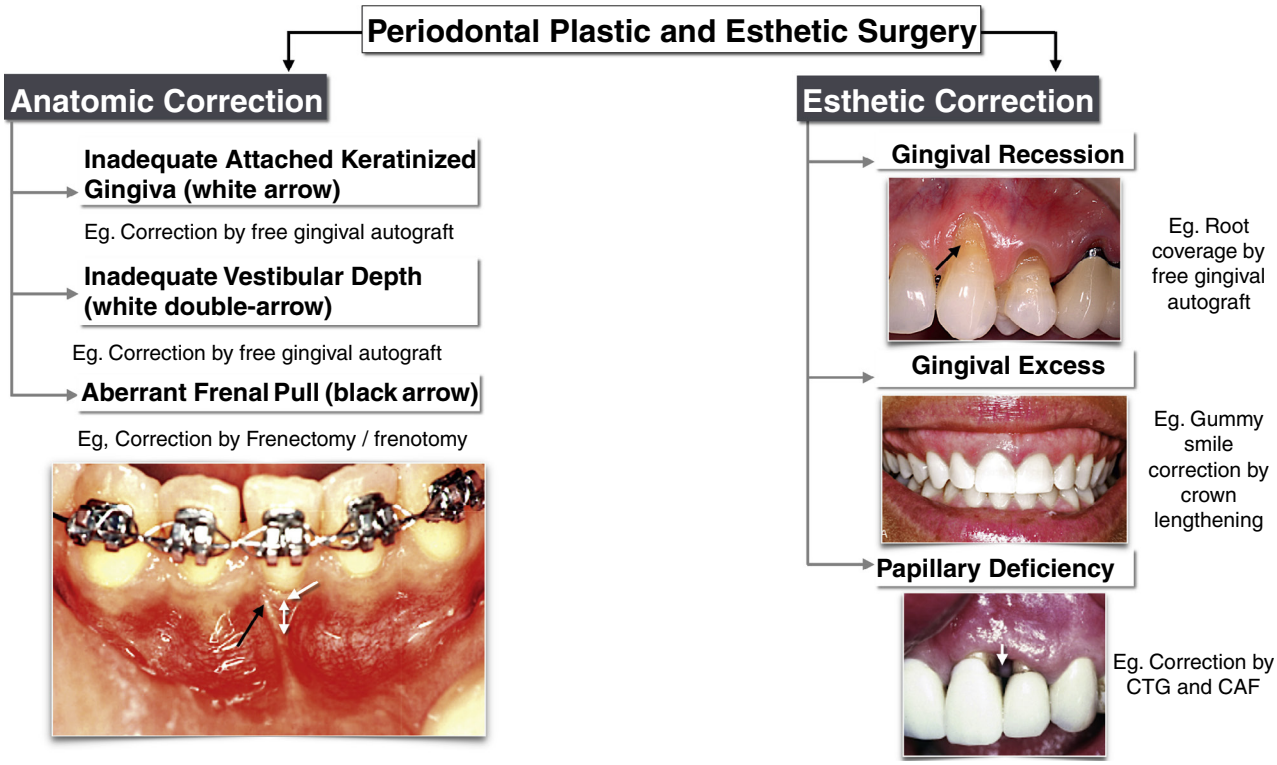
Classification of mucogingival deformities and conditions:[5]

1. gingival/soft tissue recession
2. periodontal phenotype
3. lack of keratinized gingiva
4. aberrant frenum/muscle position
5. decreased vestibular depth
6. abnormal color
7. gingival excess

Objectives of periodontal plastic and esthetic surgery:[4]

1. to maintain a healthy and stable mucogingival complex
2. to cover exposed root surfaces to tackle esthetic or hypersensitivity concerns
3. to establish an optimal zone of attached keratinized gingiva
4. to provide adequate vestibular depth
5. to eliminate aberrant muscle/frenal pull
6. to overcome the complications of subgingival margins in prosthetic dentistry.

The etiologies of problems in the mucogingival complex often overlap. For example, high frenal attachment may coexist with inadequate width of attached gingiva or vestibular depth or reduced height of residual alveolar ridge,



• **Fig. 36.1** Classification of Periodontal Plastic and Esthetic Surgery Procedures. *CAF*, coronally advanced flap; *CTG*, connective tissue graft. This figure provides a very simple classification of periodontal plastic surgical procedures based on goals of therapy, with one example of each procedure. Note that there is considerable overlap in goals/objectives of each type of therapy, and the decision-making process involves taking into account various clinical parameters. The reader is referred to Chapter 65 of *Newman and Carranza's Clinical Periodontology* (13th ed.) for detailed reading on this subject.

and contribute to a mobile gingival sulcus that subsequently becomes plaque retentive and difficult to maintain. In such cases, the goals and objectives of various periodontal surgical modalities overlap and it becomes important to tailor the treatment to the unique problem. This involves the combination of many surgical procedures to achieve one goal, or even the use of one surgical procedure to achieve many goals. The fundamentals of this complex topic are reviewed in this chapter. The reader is referred to Chapter 65 of *Newman and Carranza's Clinical Periodontology* (13th ed.) for detailed discussion of various procedures. Some aspects of periodontal plastic surgery, such as periodontal prosthetic surgery and esthetic surgery around implants, are covered in Chapter 38 and 45 of this book.

CLINICAL CORRELATE

What are the objectives of increasing the width of attached keratinized gingiva?

Widening the attached gingiva accomplishes several objectives:

- Enhances plaque removal around the gingival margin and reduces inflammation, especially around restored teeth.
- Resists future gingival recession.
- Improves esthetics.

Classification of Procedures Used in Periodontal Plastic Surgery

A basic classification of periodontal plastic and esthetic surgical procedures is given in Fig. 36.1.

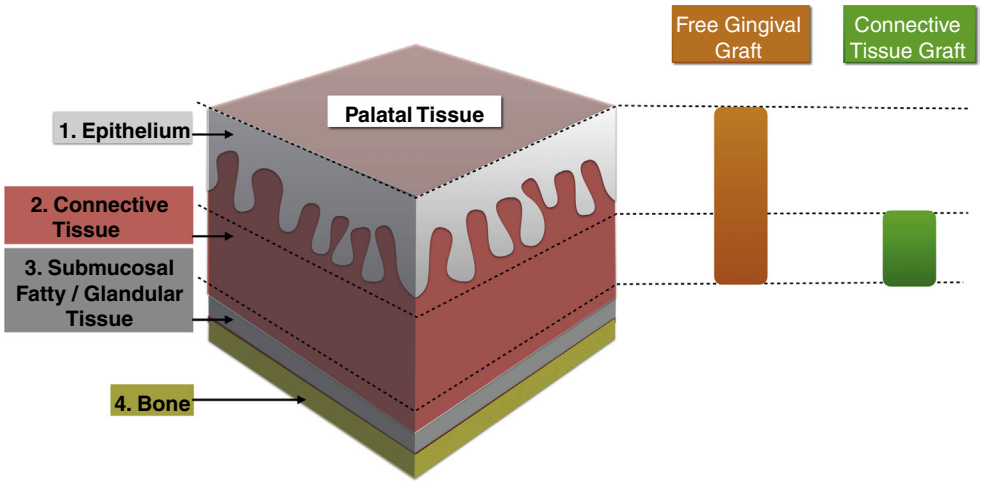
Classification of Donor Graft Tissue

Donor tissue can be obtained from various sites, including the edentulous ridge, the tuberosity area, gingivectomy tissue, and palatal tissue. The usual area of choice is the palatal tissue distal to the anterior rugae in relation to the premolar and first molar areas. This area has the widest gingival zone with the least amount of submucosal tissue, which is fatty anteriorly and glandular posteriorly. Fig. 36.2 illustrates the ideal boundaries of most commonly used free autografts harvested from the palatal region for periodontal plastic surgery.

Gingival Recession and Inadequate Keratinized Gingiva

Among the mucogingival procedures, treatment of lack of keratinized tissue and gingival recession are commonly performed surgeries and are the main focus of this review. Table 36.1 reviews the major differences between procedures performed for augmenting keratinized gingiva and those performed for root coverage.

Fig. 36.3 shows the classification of gingival recession defects.



• **Fig. 36.2 Diagrammatic Representation of Donor Palatal Graft Tissue.** The figure displays the three histologic zones (epithelium, lamina propria/connective tissue, submucosa) in the soft tissue overlying palatal bone. The two grafts most commonly harvested from palatal soft tissue for periodontal plastic surgery are:[4]

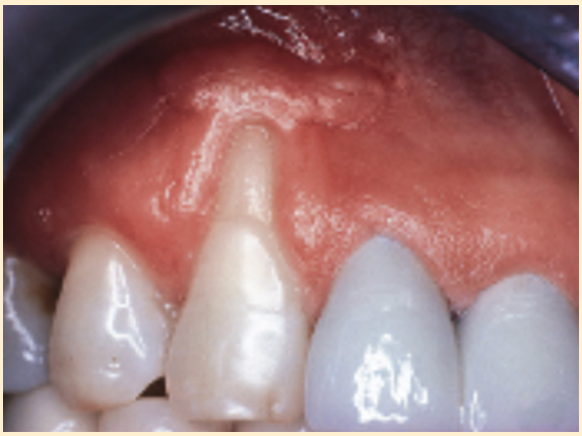
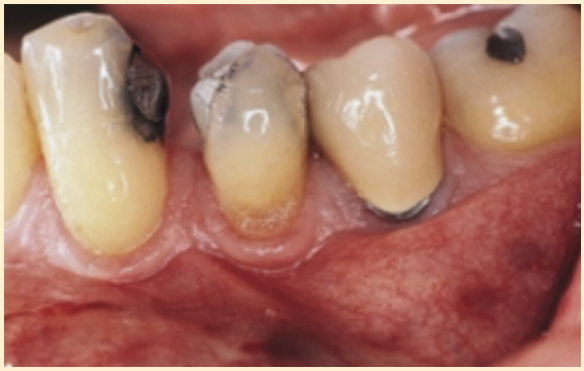
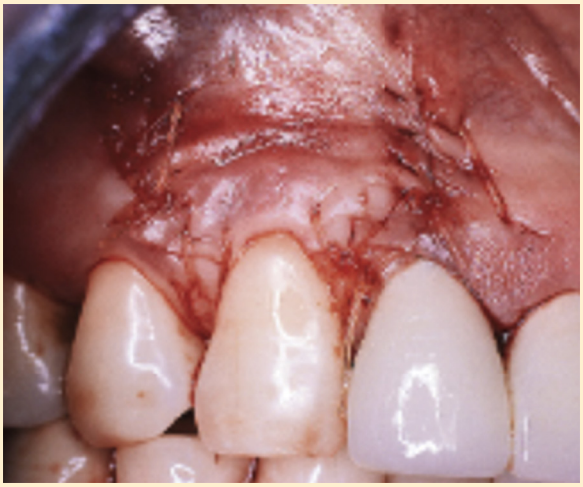
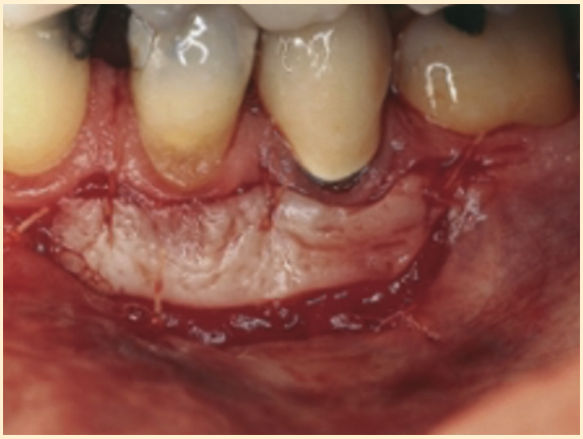
- Free gingival graft—contains both epithelium and connective tissue, and excludes submucosa.
- Subepithelial connective tissue graft—ideally contains only lamina propria, and excludes epithelium and submucosa.

TABLE 36.1 Differences Between Root Coverage and Keratinized Gingiva Augmentation Procedures

		Root Coverage Procedures	Procedures to Augment Keratinized Attached Gingiva
Area of correction		Soft tissue augmentation above existing gingival margin	Soft tissue augmentation below existing gingival margin
Primary objectives		• Recession coverage for esthetics • ↓ dentin hypersensitivity in exposed root surfaces	• ↑Tissue thickness • ↑ Keratinized gingiva width
Goals achieved	**Esthetics**	✓	-
	Hypersensitivity	✓	-
	Treatment of noncarious cervical defects*	✓	-
	Improve thickness/ biotype	✓	✓

Procedures that accomplish primary objectives		• Pedicled grafts
		  • Laterally and coronally displaced flaps
		• Free grafts
		  • FGG
		  • Free CTG
		• Combinations
		  • CAF + SCTG
		  • CAF + GTR
		  • CAF + biologic mediators
			• Pedicled grafts
			  • Apically displaced flaps
			• Free grafts
			  • FGG
			  • Free CTG
			• Combinations
			  • Vestibuloplasty + FGG

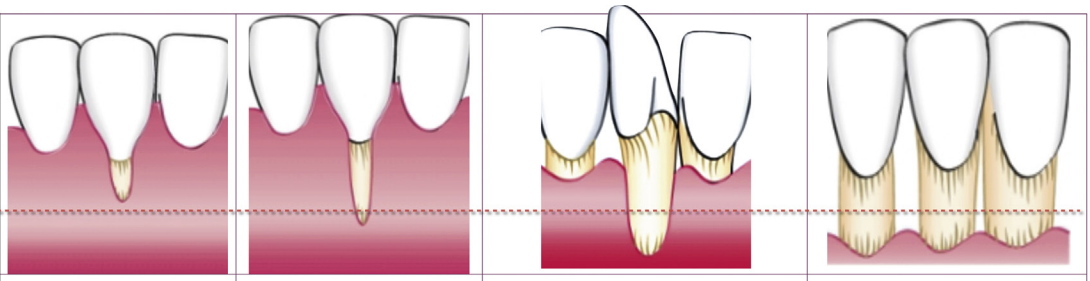
TABLE 36.1 Differences Between Root Coverage and Keratinized Gingiva Augmentation Procedures—cont'd

	Root Coverage Procedures	Procedures to Augment Keratinized Attached Gingiva
	Gingival Recession	Inadequate Keratinized Gingiva (KG)
		
	After CAF for Recession Coverage	After FGG for Augmenting KG
		

↑, increase; ↓, decrease; CAF, coronally advanced flap; CTG, connective tissue graft; FGG, free gingival graft; GTR, guided tissue regeneration; KG, keratinized gingiva; SCTG, subepithelial connective tissue graft.

*Noncarious cervical lesions causes may be: (1) erosion—due to acidic foods, beverages, and medication (mostly associated with saucer-shaped defects); (2) attrition—due to abrasive forces, such as improper tooth brushing techniques (wedge-shaped defects with sharp margins, and scratches on the tooth surface); or (3) abfraction—due to abnormal occlusal loading.[5] They may be treated using restorative procedures, root coverage procedures, or combinations of both.

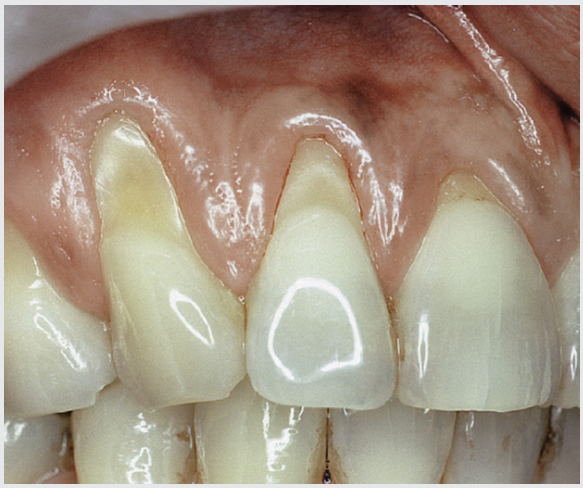
Clinical photos from: Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). Newman and Carranza's Clinical Periodontology (13th ed.). Philadelphia: Elsevier.

MGJ				
Miller, 1985	**Class I.** Marginal tissue recession does not extend to the mucogingival junction. There is no loss of bone or soft tissue in the interdental area.	**Class II.** Marginal tissue recession extends to or apical to the mucogingival junction. There is no loss of bone or soft tissue in the interdental area.	**Class III.** Marginal tissue recession extends to or apical to the mucogingival junction. There is bone and soft tissue loss interdentally or malpositioning of the tooth facially.	**Class IV.** Marginal tissue recession extends to or apical to the mucogingival junction. There is severe bone and soft tissue loss interdentally with severe tooth malposition .
Cairo et al. 2011	**Recession Type 1 (RT1):** Gingival recession with no loss of interproximal attachment*.		**Recession Type 2 (RT2):** Gingival recession associated with interproximal attachment loss ≤ the buccal attachment loss**	**Recession Type 3 (RT3):** Gingival recession associated with loss of interproximal attachment > the buccal attachment loss

• **Fig. 36.3** Classification of Gingival Recession Defects.[6,7] CEJ, cementoenamel junction; *MGJ*, mucogingival junction. * interproximal attachment is measured from the interproximal CEJ to the apical end of the interproximal sulcus/pocket. ** buccal attachment loss is measured from the buccal CEJ to the apical end of the buccal sulcus/pocket. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

CASE-BASED LEARNING EXERCISE

Scenario: A 49-year-old female patient presented to the clinic with the chief complaint: "My gums are receding and I am concealing my smile to avoid showing this area of my mouth." The patient was a past smoker who quit smoking 15 years earlier. She was known to be hypertensive but her blood pressure was well controlled with medications. Periapical radiograph revealed intact bone interdentally in the maxillary right anterior sextant. The patient reported that she brushed her teeth aggressively using horizontal brushing strokes.



Questions

1. Based on the clinical presentation and described radiographic findings, what Miller class of recession defects are noted in the maxillary right canine and lateral incisor?
   a. Class I
   b. Class II
   c. Class III
   d. Class IV
2. Based on the clinical presentation, what recession type (Cairo's classification) is noted in the maxillary right canine and lateral incisor?
   a. Type 1
   b. Type 2
   c. Type 3
3. Which of the following recession defects have better predictability for root coverage?
   a. Miller's Class I and II
   b. Miller's Class III and IV
4. Of the following mucogingival procedures, which is NOT indicated in this clinical scenario?
   a. Coronally advanced flap
   b. Coronally advanced flap with connective tissue graft
   c. Vestibuloplasty
   d. Coronally advanced flap with an allogenic dermal substitute

Clinical photo attribution: Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

This chapter was developed from Chapter 65 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: a

Explanation: They are class I defects, because the gingival recession has not extended to the mucogingival junction and there is no interdental bone loss between the involved teeth.

2. Answer: a

Explanation: Gingival recession was noted on the buccal aspect of involved teeth, but no loss of attachment was observed in the interproximal areas.

3. Answer: a

Explanation: The apicocoronal location of interdental bone plays a major role in root coverage outcomes, with intact interdental bone levels giving the best possible coverage. Miller classes I and II are distinguished from III and IV by the lack of intact inderdental bone loss in the former.

4. Answer: c

Explanation: Options (a), (b), and (d) are performed to achieve root coverage, which will address the primary concern of this patient. Vestibuloplasty is done to increase vestibule, which is not indicated in this patient because adequate vestibule already exists.

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37 Lasers in Periodontics

Relevant Terminology

Terminology	Explanation
ablation	The process by which cells and extracellular matrix in the tissues explode when they adequately absorb the laser energy
biomodulation (or photobiomodulation)	Using low-level lasers on tissues with the goals of reducing inflammation and enhancing wound healing (by increasing epithelialization, fibroblast proliferation, and matrix synthesis)
collimation	The process by which particles or waves are made narrower as they come out of a piece of equipment (e.g., laser equipment), accomplished by a device called a collimator
laser abbreviations	• Neodymium:yttrium-aluminum-garnet (Nd:YAG) • Erbium, chromium:yttrium-scandium-gallium-garnet (Er,Cr:YSGG) • Erbium:yttrium-aluminum-garnet (Er:YAG)
LASER (acronym)	Light Amplification by Stimulated Emission of Radiation
low-level laser therapy	Lasers in the red to near infrared range (600–1070 nm) are considered low-level lasers; they are used primarily for biomodulation
monochromatic	A laser beam that comes out of the laser equipment with only one wavelength (color)
optical penetration depth	The depth of penetration of a laser into a tissue
photodynamic therapy	It has three components: a photosensitizer, light, and oxygen. Light activates the photosensitizer, which then reacts with oxygen to generate singlet oxygen and free radicals, which kill the microbes

Fast Facts

How lasers are produced	The laser source stimulates emission of light energy from a specific medium that comes out as a collimated focused monochromatic beam of light energy
Key components of laser	Energy source, medium, and optical chamber (laser tube)
Potential laser-tissue interactions	Depending on the laser's wavelength, power, and exposure time, there can be reflection, absorption, scatter, or transmission
Commonly used lasers in dentistry	Argon, diode, Nd:YAG, Er:YAG, and carbon dioxide (CO_2)
Nd:YAG versus Er:YAG	• Nd:YAG and Er:YAG lasers have wavelengths of 1064 nm and 2940 nm, respectively • Er:YAG has strong affinity to hydroxyapatite and therefore allows cutting of hard tissues such as dentin and bone • Depth of penetration in tissues is significantly more for Nd:YAG (>3000 μm) than for Er:YAG (<5 μm)
Diode versus CO_2 lasers	Diode and CO_2 lasers have wavelengths of 655–980 nm and 10,600 nm respectively. Diode laser is well absorbed by pigments in soft tissues and therefore is commonly used for soft tissue procedures like frenectomy or soft tissue biopsy

Continued

Fast Facts—cont'd

Commonly advocated uses of lasers in periodontics	• Nonsurgical periodontal therapy • Gingivectomy/gingivoplasty • Crown lengthening • Soft tissue biopsies • Implant decontamination (in peri-mplantitis treatment) • Second stage implant exposure • Biomodulation • Periodontal curettage (not evidence-based). For additional info on lasers in periodontics, refer to Cobb's 2006 review on this topic.[1]
Advantages of lasers in surgical periodontal therapy	• Minimal bleeding and good visibility of the surgical site • Minimal tissue damage • Can be used with precision
Claimed advantages of lasers in nonsurgical periodontal therapy	• Minimally invasive access to root surface for scaling and root planing • Effective removal of calculus, detoxification and killing of periodontal pathogens
Lasers in nonsurgical periodontal therapy	As an adjunct to scaling and root planing, lasers produce only a modest improvement in clinical parameters (especially probing depth reduction) compared with conventional treatment alone. Evidence is limited that laser therapy by itself is either superior or comparable to conventional periodontal therapy.[2]
Lasers in the treatment of peri-implant diseases	There is currently no substantial evidence to support the use of lasers in the treatment of peri-implant mucositis. There is some evidence to suggest short-term reduction in bleeding on probing outcome in the nonsurgical treatment of peri-implantitis when lasers are used adjunctively. Currently, there is a lack of evidence for any long-term benefits of adjunctive laser therapy in peri-implant disease management.[2]
Photodynamic therapy (PDT) in the treatment of periodontitis	In a recent randomized controlled trial, no additional improvement in clinical outcomes was observed when PDT was used as an adjunct to scaling and root planing (SRP), compared with SRP alone[3]

Core Knowledge

Introduction

The constant search to provide the clinician with better equipment and techniques to improve periodontal therapy has identified laser technology as a potential therapeutic option. Recent literature describes numerous positive findings for the use of lasers in the management of periodontal and peri-implant disease, predominantly in the form of case reports and case series. However, further research as to the parameters for clinical efficacy and the biologic basis for laser therapy is still required.

Laser Physics and Biologic Interactions

Laser is an acronym for "Light Amplification by Stimulated Emission of Radiation." Fig. 37.1 reviews possible tissue interactions with laser beams.

BASIC SCIENCE CORRELATE

What are the working principles behind the use of lasers in nonsurgical and surgical periodontal therapy?

Lasers work by the following two principles:[4]

- Biostimulation—When a laser is used in the non-surgical mode for biostimulation, it aims for the following effects: general anti-inflammatory effects, quick wound healing, relief from pain and enhanced collagen production. For example, low-level laser therapy (LLLT) has been shown to reduce inflammation and enhance the outcomes following nonsurgical periodontal treatment in healthy subjects and diabetics. Initial evidence suggests that LLLT may improve the effects of periodontal regenerative procedures.
- Photothermal effect—When a laser is used in the surgical mode, the laser light energy is converted into heat; the 3 main photothermal interactions between the laser beam and target tissue are (1)Excision/incision, (2) Vaporization/ablation an (3) Coagulation/hemostasis. The desired photothermal interaction can be achieved by controlling beam size/spot size, energy and time parameters.

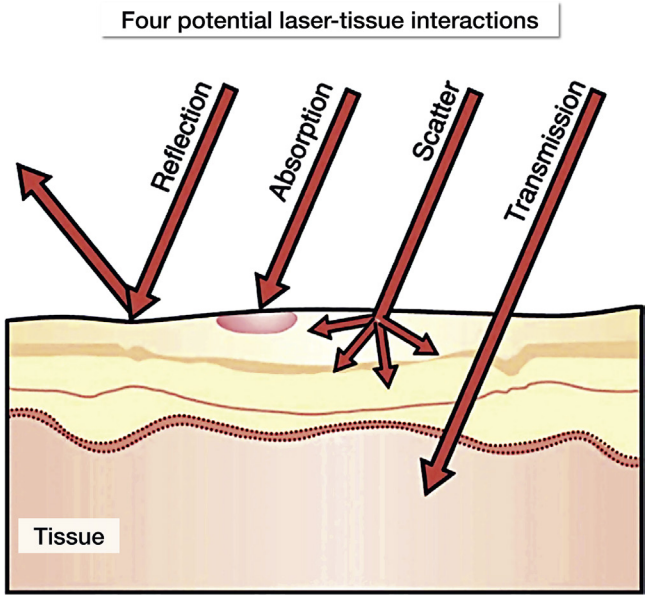
Both the biostimulatory and photothermal effects of dental lasers require the *absorption* of laser energy by intended tissues.

Table 37.1 lists the types of lasers currently used in dentistry.

TABLE 37.1 Types of Lasers Currently Used in Dentistry

	Wavelength (nm)	Current Uses (Advocated but Not Evidence-Based)			
		Restorative Dentistry	Oral Medicine and Oral Pathology	Periodontics	Implant Dentistry
Argon	488–514	Tooth bleaching and advanced curing lights	-	-	-
Diode	655–980	-	• Aphthous ulcer therapy • Biopsies • Dentinal desensitizing	• Gingivectomy/ gingivoplasty • Periodontal curettage	Second-stage implant exposure
Nd:YAG	1064	-	• Aphthous ulcer therapy • Biopsies • Dentinal desensitizing	• Gingivectomy/ gingivoplasty • Periodontal curettage	Second-stage implant exposure
Er,Cr:YSGG	2780	Hard tissue cutting (dentin)	• Aphthous ulcer therapy • Biopsies • Dentinal desensitizing	• Gingivectomy/ gingivoplasty • Periodontal curettage • Hard tissue cutting (osseous surgery)	Second-stage implant exposure
Er:YAG	2940	Hard tissue cutting (dentin)	• Aphthous ulcer therapy • Biopsies • Dentinal desensitizing	• Gingivectomy/ gingivoplasty • Periodontal curettage • Hard tissue cutting (osseous surgery)	Second-stage implant exposure
Carbon dioxide	10,600	-	-	• Gingivectomy/ gingivoplasty • Periodontal curettage	Second-stage implant exposure

Er,Cr:YSGG, erbium, chromium:yttrium-scandium-gallium-garnet; Er:YAG, erbium:yttrium-aluminum-garnet; Nd:YAG, neodymium:yttrium-aluminum-garnet. Adapted from Table 68.1 in Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). Newman and Carranza's Clinical Periodontology (13th ed.). Philadelphia: Elsevier.



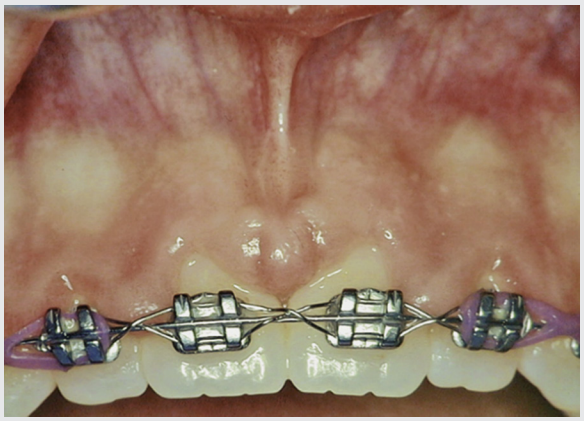
• **Fig. 37.1** Laser-Tissue Interactions

Depending on the optical properties of the tissue, the light energy from a laser may have four different interactions with the target tissue:[4]

- **Reflection**—the beam is redirected off the surface, with no effect on the target tissue.
- **Transmission**—laser energy passes directly through the tissue, with no effect on the target tissue.
- **Scattering**—this weakens the intended energy and causes the photons to change directions, leading to heat transfer to the tissue adjacent to the surgical site. In such cases, unwanted damage could occur.
- **Absorption**—The quantity of laser light energy absorbed by the intended biological tissue depends not only on the tissue characteristics, like pigmentation and water content, but also on the laser wavelength. This interaction (i.e. absorption) is the primary goal of using laser energy. From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

CASE-BASED LEARNING EXERCISE

Scenario: A 14-year-old female patient was referred from the Orthodontics department for evaluation and treatment of high frenal attachment in the maxillary anterior sextant. The patient was healthy and had 6 more months of fixed orthodontic therapy remaining. Periodontal exam revealed plaque-induced gingivitis and gingival (pseudo) pockets in the maxillary anterior sextant.



Questions

1. Which of the following is not a consequence of high frenal attachment?
- **a.** Midline diastema.
- **b.** Root caries.
- **c.** Gingival inflammation.
- **d.** Difficulty in tooth brushing.

2. Frenectomy and frenotomy are different.
- **a.** True.
- **b.** False.

3. Which of the following techniques can be used to perform frenectomy?
- **a.** Electrosurgery.
- **b.** Blade (conventional surgery).
- **c.** Laser.
- **d.** All of the above.

4. All the following are advantages of using laser in soft tissue procedures except:
- **a.** Increases wound contraction and scarring.
- **b.** Better hemostasis.
- **c.** Precise incisions.

Clinical photo attribution: Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

This chapter was developed from Chapter 68 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Solutions

1. **Answer: b**

Explanation: High frenal attachment is associated with all the other options listed.

2. Answer: a

Explanation: Frenectomy is complete removal of the frenum, while incising and repositioning the existing frenum is frenotomy.

3. Answer: d

Explanation: All of the listed approaches can be used to perform frenectomy. Each approach has its advantages and limitations.

4. Answer: a

Explanation: Laser decreases wound contraction and scarring.

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38

Periodontic–Restorative Inter-relationships

Relevant Terminology

Terminology	Explanation
biologic width (supracrestal tissue attachment)	The circumferential area (average 2 mm) in every tooth between the crestal bone and the cementoenamel junction that provides the space for the junctional epithelium and connective tissue fibers to attach.
bone sounding	Clinical procedure done under local anesthesia to determine biologic width. It is carried out by inserting a metal probe in the sulcus all the way to the bone and subtracting the sulcus depth from the resulting measurement.
centric relation	Maxillomandibular relationship in which the mandibular condyles articulate with the thinnest avascular portion of their respective articular disks within the temporo-mandibular joint complex in the anterior-superior position against the shapes of the articular eminences of the temporal bone.[1]
maximal intercuspal position	Maxillomandibular relationship in which there is complete intercuspation of the opposing teeth, independent of condylar position.[1]
occlusal vertical dimension	The distance measured between two points when the occluding members are in contact.[1]

Fast Facts

Periodontal therapy before restorative treatment	• Allows establishment of stable gingival margins (esthetics) • Allows for accurate placement of crown margins • Reduces gingival inflammation—reduces bleeding and improves visibility during restorative treatment
Preprosthetic surgical procedures	• Mucogingival procedures • Crown lengthening procedures • Ridge preservation procedure • Ridge augmentation procedure
Crown lengthening	Surgical procedure aimed at lengthening the crown of the teeth. It can be accomplished by removal of soft tissue only (gingivectomy), hard tissue only (ostectomy), or both.
Esthetic crown lengthening	Surgical procedure performed with the objective of increasing the crown length (mainly in anterior teeth) in order to achieve ideal esthetic proportions.
Functional crown lengthening	Surgical procedure performed with the objective of increasing the crown length so that a new crown or restoration can be placed with better resistance and retention.
Common indications for crown lengthening	• Improving unesthetic appearance (as in delayed passive eruption) • Restoration of a tooth that has fractured at the level of the gingiva or extending subgingivally • Restoration of a tooth that has caries at the level of the gingiva or extending subgingivally • Placing a subgingival restoration

Continued

Fast Facts—cont'd

Orthodontic extrusion	A treatment option that can be used in select cases to increase the crown length by controlled orthodontic extrusion of the tooth out of the socket.
Ridge preservation	Procedure done immediately following tooth extraction to preserve the alveolar ridge dimensions for a future dental implant. It is typically done by packing the socket with bone graft particles and covering it with a resorbable membrane.
Ridge augmentation	Procedure done in patients with ridge deformities to increase ridge dimensions, allowing placement of a dental implant in a prosthetically favorable position.
Mucogingival procedures prior to restorative procedures	• Root coverage procedures (e.g., coronally advanced flap with connective tissue grafting) • Gingival augmentation procedures (e.g., free autogenous gingival graft)
Restorative factors and periodontium	• Depth—deep restorative margins could violate biologic width and should be avoided. Additionally, depth affects the accuracy of impression making and eventually, the marginal fit of restorations • Marginal fit—open margins can harbor bacteria and lead to inflammation • Contour—overcontoured restorations will negatively affect oral hygiene practices, leading to inflammation

Core Knowledge

Critical Periodontal-Restorative Zones

The periodontium interfaces with dental restorations (crowns, fillings, etc) in a very intimate manner. The two entities, one natural and the other man-made, have several critical zones of interactions which must be made harmonious enough for providing long-term, trouble-free restorations that are both functionally and esthetically superior in design. Fig. 38.1 provides a brief review of prosthetic considerations in key transition zones at the periodontal-restorative interfaces.

Clinicians performing restorative procedures must understand the role of biologic width (also called supracrestal attachment apparatus) in preserving healthy periodontal tissues and controlling the gingival form around restorations (Fig. 38.2).

CLINICAL CORRELATE

When do we place restorative margins subgingivally?

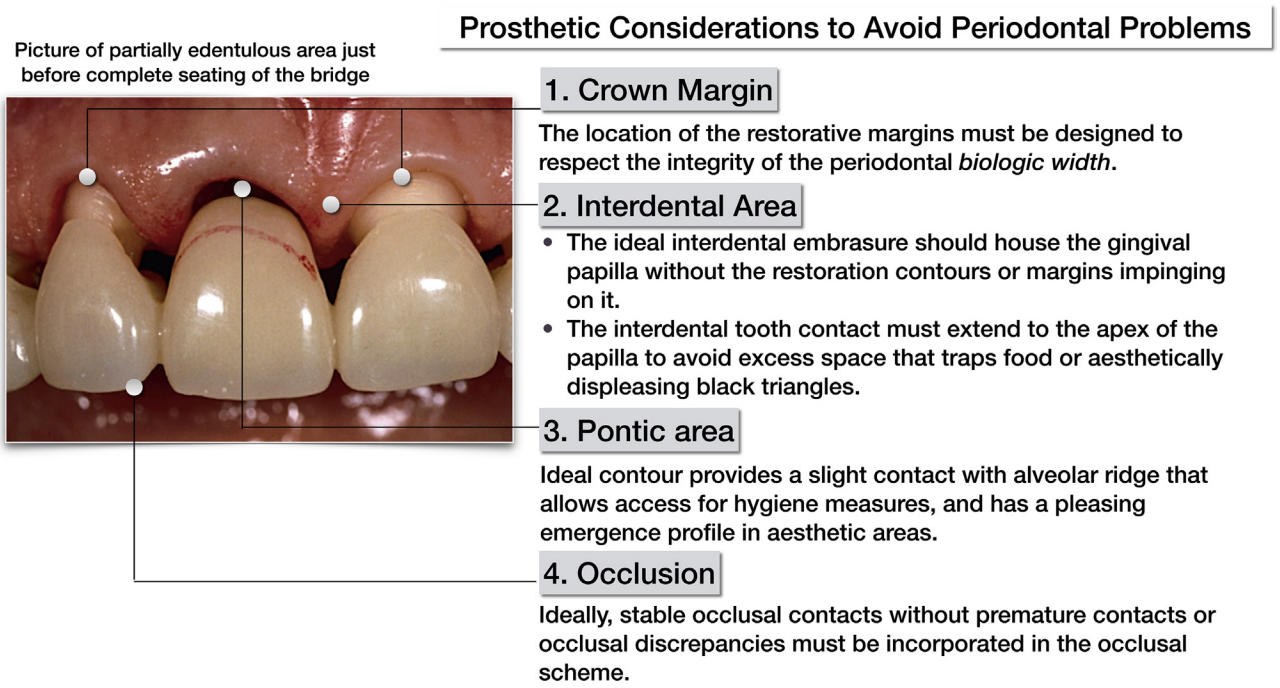
- To create adequate resistance and retention form during crown preparation
- To make significant contour alterations because of caries extent/tooth deficiencies
- To mask tooth-retention interface (mainly in anterior sextants) within the gingiva for esthetic reasons

CLINICAL CORRELATE

What are the guidelines for subgingival margin placement that allow for proper maintenance of biologic width?

It is imperative to follow certain guidelines for subgingival margin placement such that the biologic width is respected at all times. Here the clinician is advised to use the existing sulcus depth as a landmark (note that in a *clinical* situation, the base of the sulcus is taken as the coronal end of the attachment apparatus):

- Rule 1—when probing depth (PD) = 1–1.5 mm, subgingival margin placement = 0.5 mm below gingival free margin.
- Rule 2—when PD = 1.5–2 mm, subgingival margin placement = up to half the sulcus depth.
- Rule 3—when PD > 2 mm, perform gingivectomy to make the PD = 1.5 mm and then follow rule 1. The rationale for this rule is to tackle the difficulties in accurate impressioning of margins and properly finishing margins in deep gingival sulci, and to avoid increased inflammation and risk of recession.



• **Fig. 38.1** Critical Zones at the Periodontal-Restorative Interface. Color Atlas of Periodontology 1985 by H.F. Wolf, K.H. & E.M. Rateitschak T.M. Hassell Ed 3, Thieme, NY. These may be discussed under the following headings:

Crown margins

There are three options for margin placement:

1. Supragingival—finish lines during crown preparation are placed coronal to the gingival margin. This should be preferentially used wherever possible. because it places the crown margins at a level that has the least impact on the periodontium.
2. Equigingival—finish lines during crown preparation are placed at the same level as the gingival margins. This option is also well tolerated and the crown margins in esthetically sensitive areas are better hidden than in supragingival margins.
3. Subgingival—finish lines during crown preparation are placed apical to the gingival margin level. This poses the greatest biologic risk, especially if the margin is incorrectly placed too far apical to the gingival margin as it violates the gingival attachment apparatus. Fig. 38.2 illustrates the concept of biologic width (supracrestal attachment apparatus) and the ramifications of violating the same.

Interdental area

Changes in the shape of the embrasure (e.g., due to more coronally placed contact points between mesially tilted incisors or triangular shaped crowns, or due to interdental bone loss) can impact the height and form of the papilla. If the distance between the interdental bone crest level and interdental contact is 5 mm or less, the maintenance or rebuilding of complete interdental papilla is possible.[2] Some of the clinical possibilities for papilla reconstruction include:

- Restorative approach—in the case of triangular teeth, the apical repositioning of contact points to within 5 mm of bone can be achieved by the addition of proximal material (direct bonding resin, crowns, or veneers) to change the shape of teeth.
- Surgical approach—in the case of interdental bone loss, periodontal regeneration can help with papilla reconstruction.
- Orthodontic approach—in the case of tilted teeth, uprighting them orthodontically will apically reposition the contact point to within 5 mm of the interdental bone crest.

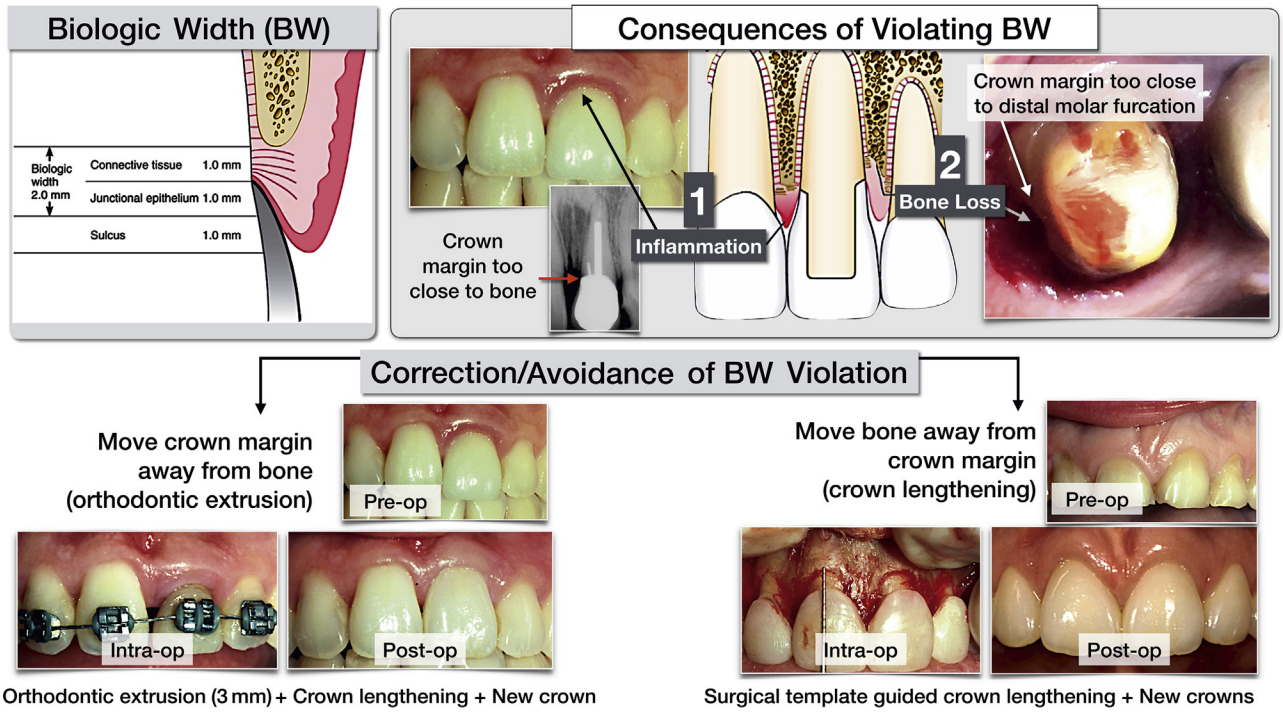
Pontic area

There are four types of pontic design: hygienic, ridge lap, modified ridge lap, and ovate. The shape of the pontic undersurface and its smoothness determines the ease with which plaque and food debris can be removed during hygiene measures. The hygienic and ovate pontics have convex undersurfaces, which makes them easiest to clean.

Occlusion

A mutually protective occlusion (one in which the posterior teeth are in contact during maximal intercuspal position while the anterior teeth are in disclusion or in light contact; the minute the mandible moves, all posterior teeth are in disclusion while only anterior teeth are in contact) is desirable.

From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.



• **Fig. 38.2** The Biologic Width (Supracrestal Attachment Apparatus).

Biologic width

The top left corner box shows a diagrammatic representation of the tissues constituting the biologic width (BW). This term refers to the dimension of space that the healthy gingival tissues occupy between the base of the sulcus and the underlying alveolar bone. It is the combined width of junctional epithelial attachment and the connective tissue attachment.[3] Clinically, a BW violation is diagnosed when the restoration margin is placed <2 mm from the alveolar bone margin and the gingival tissues are inflamed with no other etiologic factors evident.

Consequences of violating BW

Two different clinical ramifications are possible:

1. Inflammation—this is more common with deep margin placement, and is found in normal to thick periodontal biotypes. There is no bone loss but gingival inflammation persists.
2. Bone loss—this is more likely to occur in thin periodontal biotypes/phenotypes. Unpredictable bone loss and gingival recession can occur as the body attempts to regain space between the restoration margin and alveolar bone for tissue reattachment.

Correction of BW violation

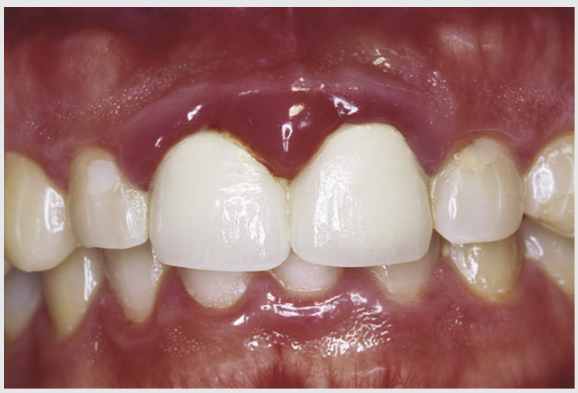
This can be done using the following approaches:

- Surgery (crown lengthening moves bone away from margin);
- Orthodontic extrusion (margin is moved away from bone).

From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

CASE-BASED LEARNING EXERCISE

Scenario: A 49-year-old male patient had been seen earlier by his general dentist and was later referred to a periodontist for evaluation. When he presented to the initial evaluation, his chief complaint was "I have tenderness in the upper front gum area and it bleeds when I brush." The patient was medically healthy, and the crowns in the maxillary central incisors had been delivered 3 months earlier. Periodontal exam revealed dental plaque–induced gingivitis and gingival (pseudo) pockets in the maxillary anterior sextant with 100% bleeding on probing in this sextant. Radiographic exam revealed normal bone levels throughout the dentition. It was clear that the margins of these crowns were more subgingival than the desired location.



Questions

1 Which of the following is a probable cause for the gingival condition observed in relation to the maxillary central incisors?
- **a.** Violation of supracrestal tissue attachment
- **b.** Mouth breathing leading to inflammation
- **c.** Chemical burns
- **d.** High frenum attachment

2 Supracrestal attached tissues (biologic width) encompasses junctional epithelium and _____
- **a.** Keratinized gingiva
- **b.** Alveolar bone
- **c.** Connective tissue attachment
- **d.** Alveolar mucosa

3 A well-made radiograph of this site shows that the distance from the crown margins (on the mesial aspects of the maxillary central incisors) to the interdental bone is less than 1 mm. What is the likely course of treatment needed to address the chief complaint of this patient?
- **a.** Guided tissue regeneration
- **b.** Coronally advanced flap
- **c.** Free gingival graft
- **d.** Crown lengthening

4 All of the following are indications for crown lengthening, except one. Which is the exception?
- **a.** Subgingival caries
- **b.** Lack of crown retention
- **c.** Severe mobility
- **d.** Short clinical crowns

Clinical photo attribution: Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

This chapter was developed from Chapters 69 and 70 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Solutions

1. Answer: a

Explanation: From the available information, the significant gingival inflammation noted around the maxillary central incisors is due to the violation of supracrestal tissue attachment (biologic width) by subgingival placement of crown margins.

2. Answer: c

Explanation: Supracrestal attached tissues consist of junctional epithelium and connective tissue attachment. It is approximately 2 mm in the apicocoronal dimension.

3. Answer: d

Explanation: One of the goals of crown lengthening is to provide adequate space between the crown margins and bone crest for the reestablishment of supracrestal attached tissues (biologic width).

4. Answer: c

Explanation: Doing crown lengthening (which involves removal of tissue attachment) on a tooth with already severe mobility will negatively affect the prognosis/longevity of the tooth. The other conditions listed are general indications for crown lengthening, provided other requirements are met.

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39

Results of Periodontal Treatment and Future Supportive Periodontal Care

Relevant Terminology

Terminology	Explanation
classical longitudinal studies	Clinical studies carried out in different parts of the world in the 1980s and 1990s that evaluated the long-term effectiveness of several periodontal therapies, including scaling and root planing and select surgical pocket reduction procedures.
experimental gingivitis	A clinical research strategy in which subjects were asked to refrain from oral hygiene practices to establish the etiology of gingivitis and the sequence of clinical and microbial changes associated with this condition.
periodontal reevaluation	Carried out 4–8 weeks after nonsurgical periodontal therapy. It comprises complete periodontal evaluation and comparison of the findings with those from the initial examination.
periodontal risk assessment	The process of assessing and assigning risk level to patients, which aids in customizing the supportive periodontal maintenance program based on individual patient needs.
rate of progression of periodontitis (direct evidence)	Slow rate: evidence of no attachment loss over 5 years Moderate rate: less than 2 mm of attachment loss over 5 years Rapid rate: 2 or more mm of attachment loss over 5 years
supportive periodontal maintenance therapy	Patients with periodontal disease are placed in an individualized recall program after therapy, during which periodic examination and scaling/polishing are performed.

Fast Facts

Ultimate goals of periodontal therapy	• To eliminate or control the etiology and risk factors • To create a favorable environment for the patient to maintain a healthy periodontium with regular home care • To create an environment that is conducive for the provider to effectively control local factors on a periodic basis
Prerequisites for gingival health	• Oral hygiene maintenance • Scaling
Experimental gingivitis	It takes on average 21 days of refrainment from oral hygiene to develop gingivitis. Upon recommencement of oral hygiene practices, gingival inflammation disappears within a week.[1]
Progression patterns of periodontal disease	In Sri Lankan tea laborers with no access to oral care, three patterns of periodontal disease progression were observed: rapid (8%), moderate (81%), and no progression (11%).[2]
Periodontal therapy and attachment loss	Absence of periodontal therapy can lead to progressively increasing pocket depth measurements due to attachment and bone loss.[3]

Continued

Fast Facts—cont'd

Periodontal therapy and tooth loss	Tooth loss is the ultimate outcome of periodontal disease. Periodontal therapy reduces the number of teeth lost to periodontal disease compared with no treatment.[3]
Supportive periodontal maintenance program and tooth loss	Supportive periodontal maintenance therapy following periodontal therapy is critical in maintaining the results and reducing tooth loss.[4]
Scaling and root planing effectiveness and pocket depth	The greater the probing depth, the lower the effectiveness of scaling and root planing, due to compromised access to root surface deposits.
Components of supportive periodontal maintenance program	• Clinical examination (hard tissue, soft tissue, periodontal evaluation. etc.) • Treatment (oral hygiene reinforcement, scaling, polishing, etc.) • Write a report, clean up the operatory and scheduling
Recall interval	The recall interval is specific to individual patient and is not fixed. Patients who are just placed in the maintenance program following treatment of periodontal disease are generally seen every 3–4 months. In patients who demonstrate excellent results with results maintained for 1 year or longer, a 6-month recall interval is considered.

Core Knowledge

Introduction

Supportive periodontal therapy (or periodontal maintenance) is distinct from, yet integrated with, active periodontal therapy. This phase of periodontal treatment is started soon after the nonsurgical phase of therapy (see Chapter 21 for correct sequencing of periodontal treatment phases). Transfer of the patient from active treatment status to a maintenance program is a *definitive* step in total patient care that requires time and effort on the part of the dentist and staff.

Rationale for Supportive Periodontal Therapy (SPT)

Insufficient patient compliance and lack of proactive, targeted periodontal maintenance after active therapy can cause:

- Plaque and calculus build-up
- Reinfection of residual pockets
- Persistent pocket activity and gingival bleeding, increasing pocket probing depth
- Increasing attachment loss, tooth mobility, tooth migration and, if unattended, possibly tooth loss
- Cervical caries

Goals of SPT

The goals of SPT include:

- **Maintenance**—of periodontal results (e.g., reduced probing depth), oral health (including cancer screening), chewing function, phonetics, aesthetics
- **Prevention**—of new infection, reinfection of inactive residual pockets, dental caries, tooth loss

CLINICAL CORRELATE

Why Do Patients Fail to Comply With Recommended Maintenance Programs? What are the Possible Methods to Improve Compliance?

Patients fail to comply with recommended maintenance regimens for many reasons, including:[5]

- Negligent attitude toward their dental condition
- Denial that they have any problem at all
- Fear of dentist/dental treatment
- Perceived/actual indifferent behavior on the dentist's part
- Stressful life events
- Economic factors

The possible methods one may employ to improve compliance include:[5]

- Simplifying the required behavior, making it more likely to be performed
- Accommodating patient's needs and values
- Following up with non-compliers closely with gentle reminders for upcoming appointments
- Providing positive reinforcement of good hygiene behaviors
- Emphasizing on the causes of, and importance of preventive measures in periodontitis

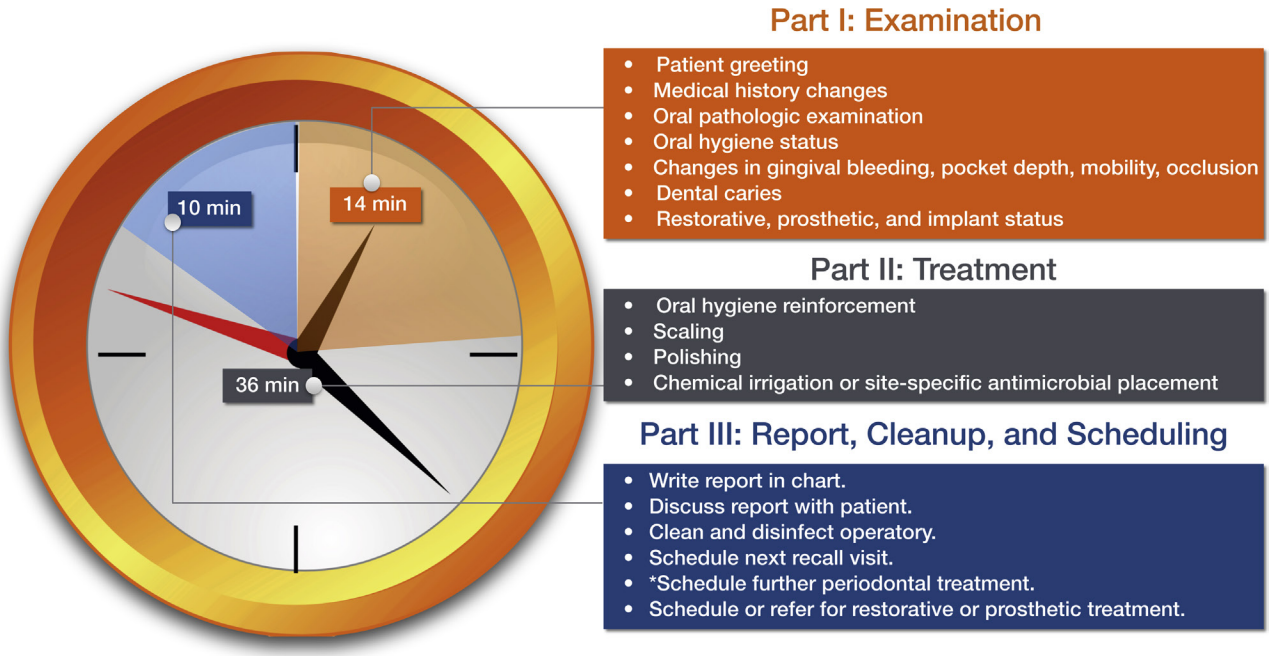
Maintenance Program During SPT

For patients who have been through periodontal therapy, the maintenance interval is typically set at 3 months initially, but may vary according to their individual needs. The time required for a recall visit for patients with multiple teeth in both arches is approximately 1 hour. Fig. 39.1 illustrates the various components of the "recall hour."

Merin's Classification of Post-Treatment Patients

Various categories of periodontal patients will be indoctrinated into a maintenance program after active therapy. Table 39.1 lists these categories, along with a suggested recall interval for each group. Patients can improve, or may relapse to a different classification due to a reduction in or exacerbation of periodontal disease.

This chapter was developed from Chapters 72 and 73 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.



• **Fig. 39.1 The Recall Hour in Supportive Periodontal Therapy**.

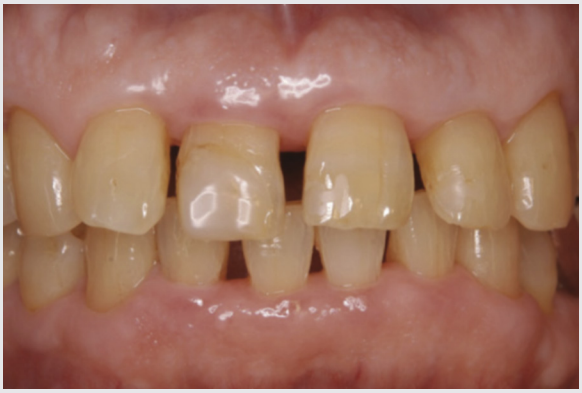
TABLE 39.1 Merin's Classification of Post-Treatment Patients

First Year		After the first year	Class A	Class B	Class C
Routine therapy, uneventful healing	Complicated prosthesis, furcation involvement, poor crown-root ratios, questionable patient compliance		Excellent maintenance of results, good oral hygiene, no complicated protheses, no teeth with <50% remaining bone	Inconsistent oral hygiene, complicated prostheses, some teeth with <50% remaining bone, ongoing orthodontic therapy, recurrent caries, smoking, systemic and/or genetic predisposition, >20% sites with BOP, dental implants	In addition to factors in Class B: Periodontal surgery indicated but not performed due to medical, psychological, or financial reasons; or condition too far advanced to be improved by periodontal surgery
Recall every 3 months	Recall every 1–2 months		Recall every 6 months Can be managed by general practitioner; bite-wing examination every 2–3 years	Recall every 3–4 months Alternatively managed by general practitioner and periodontist; periapical and/or vertical bite-wings every 1–2 years	Recall every 1–3 months Preferably managed by periodontist; periapical and/or vertical bite-wings every 1–2 years

BOP, bleeding on probing.

CASE-BASED LEARNING EXERCISE

Scenario: A 67-year-old female patient presented for periodontal reevaluation, having undergone nonsurgical periodontal therapy (scaling and root planing) 5 weeks earlier, in the two maxillary quadrants to address localized periodontitis Stage II Grade B. Reevaluation revealed significant probing depth reduction and overall improvement in the patient's periodontal health and oral hygiene. Probing depths were in the range of 1–4 mm and BOP was 8% on the day of reevaluation.



Questions

1. Based on the information provided, what will be the next phase of periodontal therapy for this patient?
   - **a.** Scaling and root planing
   - **b.** Regenerative pocket reduction therapy
   - **c.** Supportive periodontal therapy
   - **d.** Resective pocket reduction therapy
2. What supportive periodontal therapy (SPT) interval would be better to start with for this patient?
   - **a.** 3–4 months
   - **b.** 6 months
   - **c.** 8–12 months
   - **d.** 16 months
3. Which of the following is NOT a goal of SPT?
   - **a.** Monitor disease progression or recurrence
   - **b.** Provide professional cleaning
   - **c.** Update medical and dental records and take periodic radiographs (as needed)
   - **d.** Recruit clinical research subjects
4. After 1 year post-treatment, if this patient is placed on a 6 month recall, based on Merin's classification, this patient will be considered:
   - **a.** Class A
   - **b.** Class B
   - **c.** Class C

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Solutions

1. Answer: c

Explanation: Because the patient exhibited expected improvements following scaling and root planing and is in a position to maintain periodontal health by effective oral self-care and periodic periodontal maintenance, she can now be placed on SPT.

2. Answer: a

Explanation: Because she has just been treated for periodontitis, this patient should be seen more frequently for SPT, at least during the first year, to assess her compliance with oral hygiene and to monitor selective sites for disease recurrence, before placing her on a 6-month interval.

3. Answer: d

Explanation: All of the other options listed are goals of supportive periodontal therapy, which usually lasts about an hour to accomplish.

4. Answer: a

Explanation: Please refer to Table 39.1.

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40

Peri-implant Anatomy, Biology, and Function

Relevant Terminology

Terminology	Explanation
keratinized mucosa	The implant counterpart of keratinized gingiva.
primary implant stability	Stability obtained at the time of implant placement; influenced by the macro design (geometry) of implants and the quality and quantity of bone.
Proprioception	The reception of stimulation of sensory nerve terminals within the tissues of the body that give information concerning movements and the position of the body; perception mediated by proprioceptors.[1]
secondary implant stability	Stability obtained over time after implant placement; influenced by the micro design (surface) of implants and the quality and quantity of bone.
Sharpey fibers	Collagen fiber bundles that pass into the cementum and outer circumferential lamellae of alveolar bone.

Fast Facts

Osseointegration	Per-Ingvar Brånemark made an accidental discovery while studying blood supply to bone. He called the integration between titanium implant and bone "osseointegration."
Dental implant types	• Endosseous (blade, pin, disc, or screw-shaped) • Subperiosteal • Transmandibular
Overheating of bone and dental implants	Overheating the bone while preparing the implant osteotomy can lead to bone necrosis. The critical temperature for bone cells to remain viable, that should not be exceeded is 47°C for 1 minute.[2]
Epithelial attachment to dental implants	The long junctional epithelium of mucosa surrounding dental implants attaches to the titanium surface of the implants by means of basal lamina and hemidesmosomes. The epithelial attachment measures about 2 mm in length in the apicocoronal dimension.
Connective tissue surrounding dental implants	• Lacks periodontal ligament and cementum (compared with natural tooth) • No inserting collagen (Sharpey) fibers; fibers run parallel to implants (except for a few studies showing perpendicular orientation when implants with laser-microtextured grooves were used) • Connective tissue attachment measures about 1–2 mm in length in the apicocoronal dimension
Keratinized mucosa and dental implants	The presence of keratinized mucosa around the collar of dental implants offers comfort to patients while performing oral hygiene procedures and has also been shown to reduce inflammation of the periimplant mucosa and other implant complications.
Vascular supply	Compared with the gingiva around natural teeth, there is an overall reduction in vascularity in the periimplant mucosa because of the lack of periodontal ligament and the blood vessels present in it.
Clinical comparisons between natural teeth and implants	• Lack of periodontal ligament with implants means rigid fixation of implant to bone without any possibility for orthodontic movement • Lack of proprioception in implants due to absence of the periodontal ligament that contains the necessary receptors • It is common to notice deeper probing depths around implants compared with natural teeth, due to the anatomic relationship of the periimpant mucosa and the implant shoulder • Progression of inflammation to deeper tissues is faster around implants compared with that in natural teeth

Core Knowledge

Introduction

In the 1950s Per-Ingvar Brånemark, a Swedish professor of anatomy, had a serendipitous finding while studying blood circulation in bone; it marked the beginning of modern implant dentistry and the use of titanium implants. He coined the term *osseointegration* and developed an implant system with a specific protocol to achieve this phenomenon predictably. The discovery of an intimate bone-to-implant apposition with titanium that offered sufficient strength to cope with load transfer allowed the implants to anchor prosthetic replacements of teeth. Today, implant designs, surgical placement techniques, healing times, and restorative protocols continue to evolve to accommodate growing demands.

Osseointegration

Histologically, the term osseointegration refers to the direct structural and functional connection between ordered, living bone and the surface of a load-bearing implant without intervening soft tissues.

CLINICAL CORRELATE

What are some of the clinical factors affecting osseointegration of dental implants?

- **Immobilization:** Micromovements exceeding 150 μm at the bone-implant interface will impair osteoblasts' differentiation, and fibrous encapsulation will occur between the bone and implant surface. Hence implant loading by occlusal forces during the healing period must be planned carefully.
- **Patient-associated factors:** Examples include detrimental habits (e.g., tobacco smoking, alcohol and drug abuse); poor bone quality and quantity; history of radiotherapy to the jaws; and poorly controlled systemic disease (e.g., diabetes).
- **Implant-associated factors:** Examples include material biocompatibility, surface treatment, implant microdesign and macrodesign, loading protocols, and prosthetic considerations.

- *Clinically*, it is the asymptomatic rigid fixation of an alloplastic material (implant) in bone with the ability to withstand occlusal forces.

Fig. 40.1 describes the process of osseointegration.

Comparison of Hard and Soft Tissue Interfaces Around Teeth and Implants

Periimplant soft tissues are mostly similar in appearance and structure to periodontal soft tissues, but there are some differences in their histology:

- The periimplant mucosa is not '*attached*' to the implant; instead it is *sealed* by the barrier epithelium to the implant.
- Although rich in collagen fibers, the periimplant mucosa has fewer fibroblasts and limited blood supply; it is a scar-like tissue with limited potential for repair when compared to the gingiva around natural teeth.

Fig. 40.2 shows a comparison between hard and soft tissue interfaces around teeth and implants.

CLINICAL CORRELATE

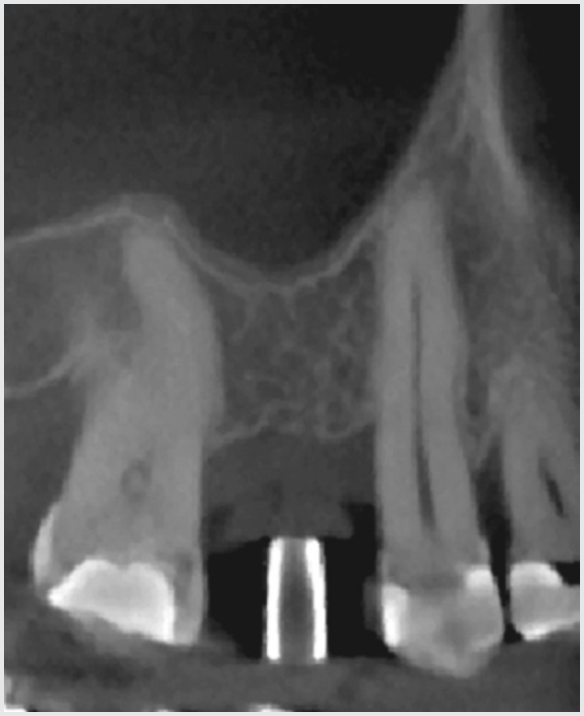
What are the clinical implications of a direct bone-implant interface without an intervening periodontal ligament space?

At the bone level, the absence of the periodontal ligament (PDL) surrounding an implant has important clinical consequences:

- Due to the lack of resilient connection between implants and supporting bone, implants cannot migrate to compensate for the presence of a premature occlusal contact (as natural teeth can). Thus any occlusal disharmony will have repercussions at the restoration-to-implant connection (e.g., screw loosening, abutment fracture), the bone-to-implant interface (loss of osseointegration), or both.
- The absence of a PDL around implants reduces tactile sensation and reflex function (attributed to proprioceptive receptors within the PDL around natural teeth). This is very challenging when osseointegrated, implant-supported, fixed prostheses are present in both jaws.
- Natural teeth continue to erupt and migrate during growth (a function attributed to the presence of PDL), whereas implants do not; hence implants placed in growing individuals (children and adolescents) can lead to occlusal disharmonies.

CASE-BASED LEARNING EXERCISE

Scenario: A 52-year-old male patient presented to the clinic with the chief complaint: "I am missing a tooth in the upper right side and I am interested in a dental implant." The patient had not been to a dentist for 3 years; upon clinical examination, a diagnosis of gingivitis in a patient with a history of periodontitis was made. Attachment loss from past periodontal disease and its treatment was evident. Signs of gingival inflammation and generalized plaque buildup were noted. BOP was 45% and the probing depths were in the range of 1–4 mm (the 4-mm pockets being pseudo pockets). The patient had well-controlled type II diabetes and took metformin for the same. Cone beam computed tomography (CBCT) scan was made with a guide in place. Buccolingual width at the edentulous site measured about 8.5 mm.



Questions

1. Based on the information provided, what should be the immediate next step in the treatment plan?
- **a.** Sinus lift to augment bone height
- **b.** Scaling to address inflammation
- **c.** Sinus lift and implant placement
- **d.** Implant placement.

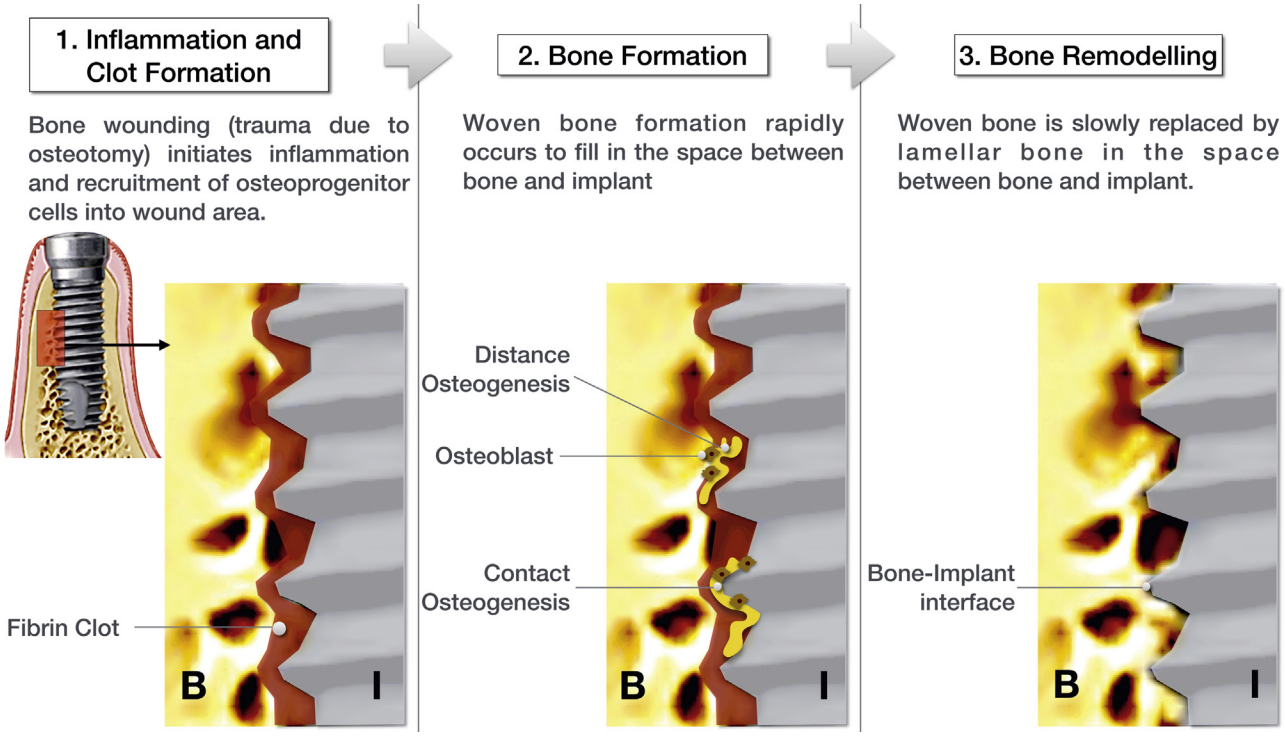
2. The CBCT scan revealed the presence of 7 mm of bone apicocoronally. If we are planning to place an implant of length 9 mm, which of the following approaches is likely needed in this site?
- **a.** Internal (crestal) sinus augmentation
- **b.** External (lateral) approach
- **c.** Combination of internal and external approaches

3. The patient's past history of periodontal disease may have a _____ impact on the outcomes of future dental implants.
- **a.** Negative
- **b.** Positive

4. The patient's diabetic status will have a negative effect on the implant outcomes.
- **a.** True
- **b.** False

Clinical photo attribution: Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.

This chapter was developed from Chapter 74 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.



• **Fig. 40.1** Stages of Osseointegration. The process of osseointegration may be broadly divided into three phases: **Inflammation and clot formation (48 hours):**

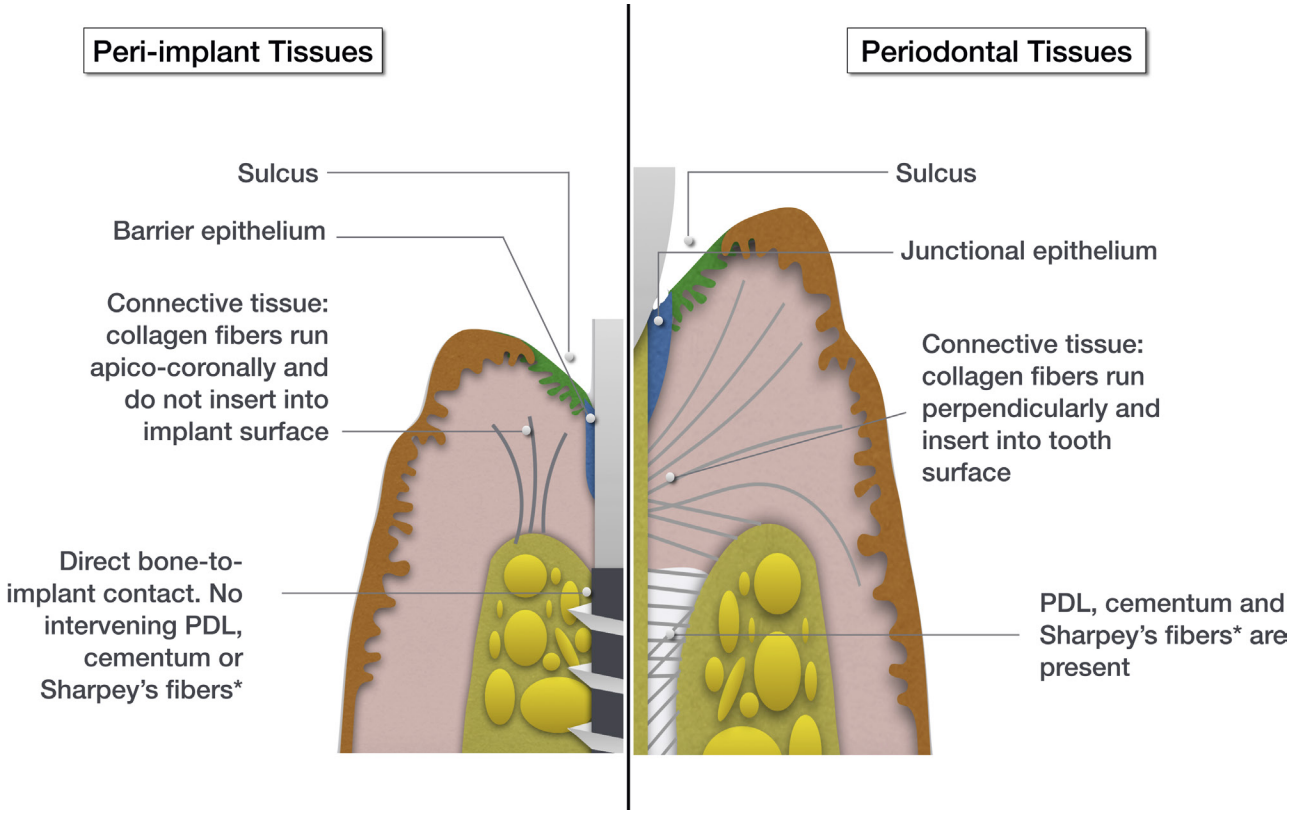
- Following osteotomy (implant site preparation) and placement of the implant (I) in close contact with cortical and trabecular bone (B), a blood clot forms in the space between the two surfaces.
- The inflammatory response to the surgical injury aims to remove the damaged tissues and initiate the healing osteogenic process, which involves the release of regenerative biomolecules (e.g., bone growth factors) from damaged cells.
- The fibrin clot is replaced by granulation tissue which is rich in blood vessels and mesenchymal cells.
- Mesenchymal cells differentiate to form osteoprogenitor cells and bone-forming cells (osteoblasts).

Bone formation (1–4 weeks):

- Osteoblasts from the bone marrow migrate into granulation tissue and lay down an osteoid matrix around blood vessels. When this osteoid matrix is mineralized by hydroxyapatite deposition, woven (immature) bone is formed.
- Woven bone forms either from the side of implant (contact osteogenesis by direct deposition of bone on the implant surface) or from the side of the alveolar bone (distance osteogenesis by bone deposition away from the implant at the bone surface).
- In the case of a textured implant surface, adherence of the fibrin network to implant surface is promoted and this acts as a scaffold over which cells can migrate to establish osteoid formation *directly* on implant surface (contact osteogenesis). This same process cannot be triggered by a smooth machined surface.
- In the case of a machined implant surface or when there exists a significant gap between the implant and bone (also termed 'jumping distance'), osteoid formation is promoted more on the bone surface rather than on implant surface (distance osteogenesis).
- There is gradual coalescence of woven bone across the clot space from both distance and contact osteogenesis, leading to formation of bony bridges between implant surface and bone. This represents the first stages of osseointegration, viz., the direct connection between newly formed bone and the implant.

Bone remodeling (up to 18 months):

- Woven bone is progressively and slowly replaced by lamellar bone with organized, parallel layers of collagen fibrils and dense mineralization.
- Ultimately, a steady state is reached where the lamellar bone is continuously resorbed and replaced. Once osseointegration is achieved, implants can resist and function under the forces of occlusion for many years.
- Osseointegration, the union between bone and implant, is measured as the proportion of the total implant surface that is in contact with bone (also called bone-to-implant contact or BIC). From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.



• **Fig. 40.2** Comparison of Hard and Soft Tissue Interfaces Around Teeth and Implants.

- After implant placement, a delicate periimplant mucosal seal is established with the help of a barrier epithelium via hemidesmosomes. This is considered identical to that of the long junctional epithelial attachment around teeth.
- The connective tissue that is in direct contact with the implant surface has collagen fibers parallel to the implant surface without attachment / adhesion to the implant body (adhesion). As a result, the resistance to probing around implants is less than when probing around teeth, leading to increased probing depths around implants.
- Natural teeth have a periodontal ligament with connective tissue fibers inserting into the cementum and suspending them in the alveolar bone. Osseointegrated implants have no inserting collagen fibers anywhere along the bone-implant interface. Bone is in direct contact with the implant surface without intervening soft tissues; this is considered a "functional ankylosis."
- Due to the lack of an intervening periodontal ligament, the implant blood supply is limited to sources such as the peri-implant mucosa and the supraperiosteal blood vessels.
- **Sharpey fibers* are bundles of collagenous fibers that pass into the outer circumferential lamellae of alveolar bone and the cementum of teeth. *PDL*, periodontal ligament

Case-Based Learning Exercise

Solutions

1. Answer: b

Explanation: The patient's oral hygiene and gingival inflammation should be addressed first by providing thorough oral hygiene instructions and scaling before putting him through any surgery.

2. Answer: a

Explanation: Because only a few millimeters of bone height are needed for the planned implant, it can be predictably achieved with internal sinus augmentation.

3. Answer: a

Explanation: The prevalence of marginal bone loss and biologic implant complications tends to be higher in patients with a past history of periodontal disease.

4. Answer: b

Explanation: This patient's diabetes is well controlled, so his diabetic status should not negatively impact the outcome.

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41 Clinical Evaluation of the Implant Patient

Relevant Terminology

Terminology	Explanation
American Society of Anesthesiologists (ASA) Physical Status Classification[1]	ASA I: Normal healthy patient ASA II: Patient with mild systemic disease ASA III: Patient with severe systemic disease ASA IV: Patient with severe systemic disease that is a constant threat to life ASA V: Moribund patient who is not expected to survive without the operation ASA VI: A declared brain-dead patient whose organs are being removed for donor purposes.
bone density types	Based on bone density, alveolar bone can be classified into:[2] • Type I—highly dense bone; (mainly cortical) • Type II—dense bone but not as dense as type I; (optimal cortical bone covering trabecular bone) • Type III—bone denser than type IV but not as dense as type II bone; (thin cortical plate covering trabecular bone) • Type IV—least dense bone (very thin cortical plate covering sparse trabecular bone) Mandible is denser than maxilla, with the maxillary posterior sextant being the least dense of all
ridge deficiency	Loss of alveolar bone following tooth loss leads to alveolar ridge deficiency. It can be horizontal (loss of ridge width), vertical (loss of ridge height), or combined in nature.
ridge dimensions	The apicocoronal dimension of the alveolar ridge (measuring from the crest at the site of implant placement) refers to the ridge height, while the buccolingual or buccopalatal dimension denotes the ridge width. Available ridge height is dictated by the presence of anatomic structures such as maxillary sinus and inferior alveolar nerve canal in maxillary and mandibular posteriors, respectively.
ridge volume	The total volume of bone at the site of planned implant(s) placement that dictates the position of the implant and the need for additional reconstructive procedures. A cone beam computed tomography (CBCT) scan is required to adequately assess ridge volume.

Fast Facts

Advantages of implant-supported single crown	• Helps to maintain bone volume • Good success rate (high 90s %) in low-risk population • Provides fixed support to the prosthetic crown (compared with removable prosthesis) • Provides opportunity for patients to perform interdental cleaning (compared with fixed partial dentures) • Avoids the need to alter/prepare adjacent teeth (compared with fixed partial dentures)

Continued

Fast Facts—cont'd

Key diagnostics for pretreatment evaluation	• Thorough clinical extraoral and intraoral evaluation (including occlusal and periodontal assessment) • Imaging analysis—appropriate radiographs and CBCT (if needed) • Study models
Local factors dictating implant placement in partially edentulous patients	Adjacent teeth evaluation: • Periodontal and endodontic status of adjacent teeth • Tilt and root anatomy of adjacent teeth.Three-dimensional evaluation of available space for implant and prosthesis: • Mesiodistal edentulous dimension (interdental space) • Apicocoronal edentulous dimension (interocclusal space) • Buccolingual alveolar bone thickness • Alveolar bone height • Anatomic landmarks: ◦ Maxilla—location of the floor of the maxillary sinus (maxillary posterior implants), nasal cavity (maxillary anterior implants), and nasopalatine canal (maxillary anterior implants) ◦ Mandible—vicinity of inferior alveolar canal (mandibular posterior implants) 'mylohyoid ridge' and mental foramen (mandibular premolar site implants) • Other considerations: ◦ Width and thickness of keratinized mucosa • Occlusal scheme
Advantages of CBCT over two-dimensional imaging	• Provides three-dimensional information on bone volume (especially bone thickness) at the site of interest • Provides detailed information on key anatomic structures (e.g., maxillary sinus, location of inferior alveolar nerve canal) • Helps with implant planning and computer-guided implant placement
Accepted distance between implants and teeth	• Between adjacent implants: 3 mm • Between implant and natural tooth: 1.5 mm
Minimal mesiodistal edentulous space required for implants of various diameters	• Narrow diameter implants (e.g., 3.25 mm) = 6 mm • Standard diameter implants (e.g., 4.1 mm) = 7 mm • Wide diameter implants (e.g., 5 mm) = 8 mm
Minimal alveolar bone width	For an implant of 4 mm diameter, a minimal width of 6–7 mm is needed in order to place the implant without any additional bone augmentation procedure. This allows for the presence of at least 1–1.5 mm of bone circumferentially around the implant.

Core Knowledge

Introduction

Dental implants are placed to manage partially and completely edentulous conditions. Patient selection is critical for success in implant dentistry. Most of the evaluation performed for implant cases is similar to routinely performed clinical and radiographic evaluation processes (see Chapter 19); this chapter deals with only the specific considerations that must be included in clinical evaluation of the implant patient. For imaging assessment of the implant patient, see Chapter 42.

Clinical evaluation of the implant patient must include the following components:

- Overall patient evaluation—medical history, attitude, detrimental habits that increase the risk for implant failure
- Oral/dental evaluation—includes extraoral and intraoral evaluations to assess the feasibility of implant placement

Overall Patient Evaluation

Purpose of Medical Evaluation in Implant Dentistry

Implant therapy involves treating an oral (edentulous) condition; this is different from other dental surgeries performed to treat an ongoing oral disease or infection. The medical evaluation of prospective implant patients is done not only to see if they can withstand implant placement (surgical risk) but also to assess their risk for complications associated with implant therapy (implant failure risk). The following must be kept in mind during clinical evaluation:

- Dental implant placement should be deferred in patients with a systemic condition or disease until the systemic condition is managed and stabilized.
- For all medically compromised patients, seeking physician consultation and medical clearance is important.

Medical screening involves:

1. Laboratory tests—complete blood count, prothrombin time, international normalized ratio (INR) and glycated hemoglobin (HbA1c)
2. Vital signs—blood pressure, pulse rate, respiratory rate
3. American Society of Anesthesiologists (ASA) physical status classification system

Following medical evaluation, risk factors for both surgery and implant failure are identified.

Surgical risk factors

Absolute contraindications—implant therapy is inadvisable in:

- Patients with *ongoing* chemotherapy and radiation therapy (head and neck)
- Patients on *intravenous* bisphosphonates
- Patients with psychiatric conditions (e.g., schizophrenia, dementia)
- Patients with end-stage renal disease
- Substance abuse (eg. alcohol, drugs)

Relative contraindications—implant therapy may be performed after risk mitigation only when absolutely necessary in patients with:

- Uncontrolled diabetes
- Uncontrolled hypertension
- Bleeding disorders
- Immunosuppression (steroid therapy, HIV/AIDS)
- Risk of poor wound healing (e.g., oral bisphosphonates, lupus erythematosus, gastroesophageal reflux disease)

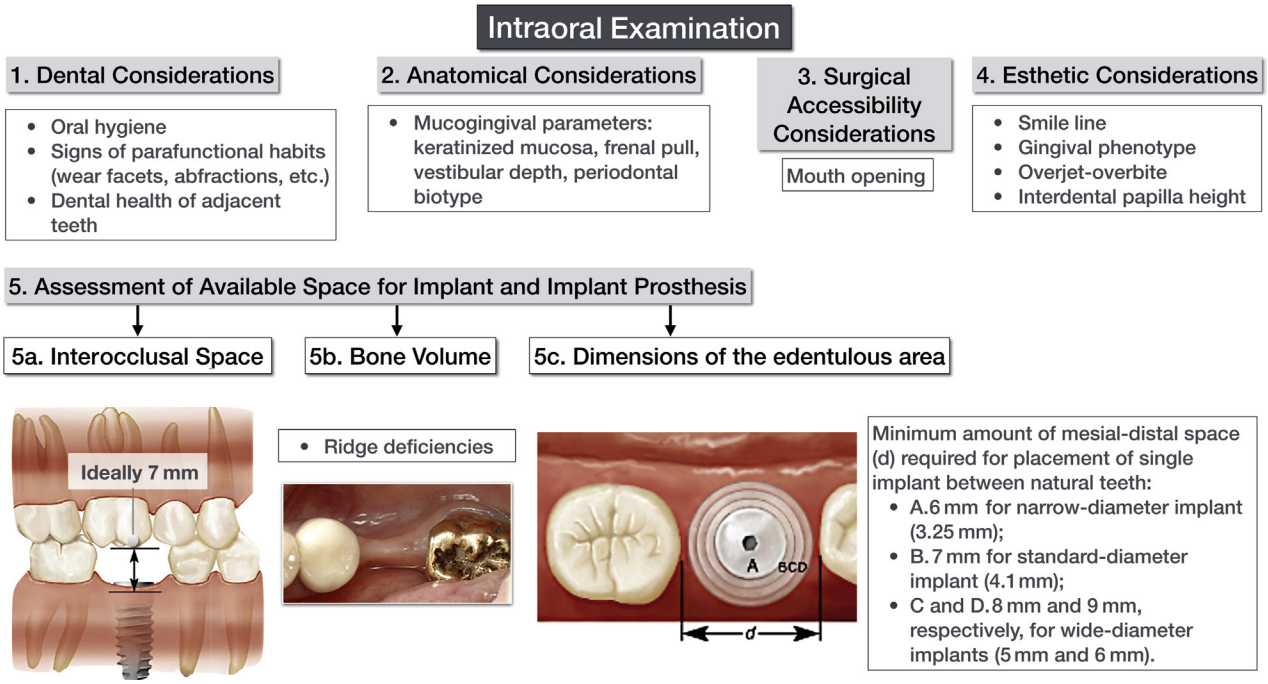
Risk factors for implant failure

- Growing individuals (patients < 18 years of age)
- Smoking
- History of periodontitis (treated or untreated)
- Severe bone disease (e.g., Paget disease, osteoporosis)
- Poor glycemic control

Oral/Dental Evaluation

Extraoral evaluation: Includes assessment of temporomandibular joint, masticatory muscles, head and neck lymph nodes, and facial symmetry.

Intraoral evaluation: Involves detailed consideration of any situation that might pose a risk to ideal placement, successful osseointegration, and restoration of a dental implant at the level of the implant site and surrounding teeth (Fig. 41.1).



• **Fig. 41.1** Clinical Intraoral Evaluation for Implant Therapy. This includes a systematic assessment to identify potential risk factors for implant failure:

1. **Dental considerations**
   - Poor oral hygiene leads to peri-implantitis and, if left untreated, implant failure.
   - Excessive occlusal loading increases the risk of biomechanical complications and must be taken into account while designing the implant prosthesis.
   - Apical lesions, root remnants, and endodontic infection within the jawbone in the vicinity of an integrating implant can interfere with osseointegration. They must be identified and treated before implant placement.
2. **Anatomic considerations**
   - Mucogingival parameters are assessed and augmented, if required, to ensure stability of the peri-implant mucosal complex.
   - Radiographic assessment of vital anatomic structures is discussed in Chapter 42.
3. **Surgical accessibility**
   - Mouth opening: to allow access for drills and for ideal implant placement (angulation, position) especially in posterior areas of the mouth, a minimum of 40-45mm of mouth opening is vital.
   - It is important to test surgical access before confirming the surgery appointment.
4. **Esthetic considerations**
   - A high smile line is very challenging in implant dentistry because the junction between the restorative components and the implant fixture crest may become visible; these patients will show tissues surrounding the implant restoration when they smile.
   - Patients with a thin, scalloped biotype or phenotype are considered as "high esthetic risk" because they experience more pronounced bone resorptions after tooth extraction than patients with a thick, flat biotype. These sites are also more prone to mucosal recession following implant placement.
   - Excessive overjet and overbite affect proper three-dimensional implant positioning, leading to functional and esthetic complications.
   - Proximal bone collapse (such as that seen when two adjacent teeth are extracted) can result in papilla loss. This requires special considerations during treatment planning in the anterior maxilla.
5. **Assessment of implant site**
   - Interocclusal space—this dimension will vary depending on implant design and manufacturer component dimensions. However, an interocclusal dimension of 7 mm is considered ideal for uncomplicated implant restorations. When this dimension is < 5 mm, it is prudent to select screw-retained abutments to, instead of cement-retained ones, avoid prosthetic complications.
   - Bone volume—ridge deficiencies can cause certain problems:
     - Buccal bone concavities (horizontal/buccolingual bone loss)—associated risks include bone fenestration and drilling perforations. Therapeutic options to mitigate these risks include tapered implant and guided bone regeneration (GBR).
     - Vertical resorption (loss of alveolar bone height)—associated risks include injury to anatomic structures (e.g., mandibular nerve) while drilling. This can be mitigated using shorter implants, GBR, and so on.
   - Dimensions of edentulous area—For a standard-size implant, the mesiodistal space between teeth required for ideal placement is as follows:1 implant—7mm; 2 implants-14mm; 3 implants 21mm and so on. Inadequate interdental space requires modifications in position, orientation, implant number and sometimes orthodontic therapy for space creation for ideal implant positioning. Other points to be kept in mind include:
     - A minimum of 3 mm is required between two implants.
     - A minimum of 1.5–2 mm is required between a tooth and an implant. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

CLINICAL CORRELATE

What precautions should be considered to increase implant success in irradiated head and neck cancer patients?[3,4]

- It is best to plan for implant surgery >21 days before radiotherapy (radiotherapy predisposes an individual to bone necrosis due to endarteritis effect).
- To minimize the risk of osteoradionecrosis and osseointegration failure, the total radiation dose should be less than 66 and 50 Gy, respectively.
- If > 50 Gy is used, hyperbaric oxygen should be given.
- Implant surgery should not be carried out during radiotherapy, and implant placement is best postponed for 9 months after radiotherapy.
- Use implant-supported prostheses with no mucosal contact and avoid immediate loading.
- Ensure strict asepsis during the implant surgical procedure, and consider the use of antibiotics.

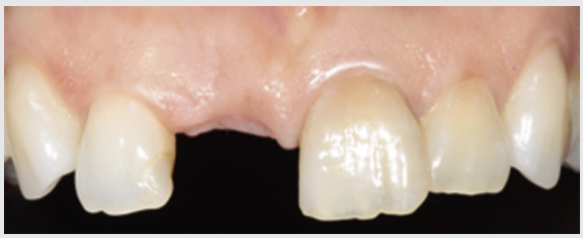
CLINICAL CORRELATE

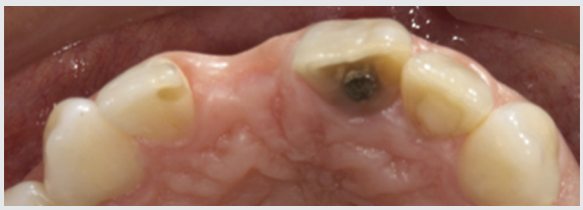
Is implant placement contraindicated in a periodontitis patient?

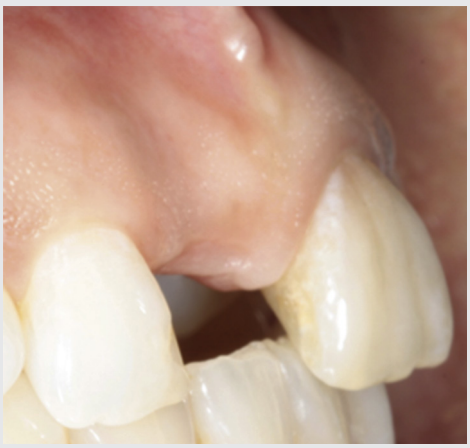
- Implant placement in *treated* periodontitis—dental implant therapy is a viable option and not contraindicated in periodontally compromised patients who have undergone treatment and as long as they are under proper maintenance protocols. Long-term studies over a period of 3–16 years have reported implant survival rates of more than 90% when the patients are well maintained periodontally.[5]
- Implant placement in *untreated* periodontitis—this has questionable prognosis due to the possibility of translocation of microorganisms, disease recurrence/flare-ups, and poor oral hygiene, and the added complications of influencing systemic disorders (e.g., periodontitis influences poor glycemic control) that may contribute to colonization of the periimplant sulcus and lead to periimplantitis. Hence periodontitis must always be treated and disease must be controlled prior to placement of implants.

CASE-BASED LEARNING EXERCISE

Scenario: A 52-year-old female patient presented with the chief complaint "I was eating and my front tooth broke." She had been seen by a general dentist who fabricated a "flipper" type removable partial denture, but she did not like it. The patient was interested in restoring the edentulous area with an implant-supported fixed partial denture. She had no medical conditions and no known drug allergies.







Questions

1. The clinical images reveal all but one of the following findings. Which is the exception?
   - **a.** Discrepancy in the gingival margins
   - **b.** Aberrant frenum attachment
   - **c.** Horizontal ridge deformity
   - **d.** Clinical periodontal health
2. Which of the following image modalities is required to further assess ridge deformity at the site of interest?
   - **a.** Occlusal film
   - **b.** Lateral cephalometrics
   - **c.** Cone beam computed tomography (CBCT)
   - **d.** Panoramic radiograph
3. What is the minimum interocclusal space for a cement-retained restoration?
   - **a.** 4 mm
   - **b.** 5 mm
   - **c.** 6 mm
   - **d.** 7 mm
4. All of the following are precautions that can increase implant success in irradiated head and neck patients *EXCEPT* one. Which is the exception?
   - **a.** Ensure strict asepsis during the surgical procedure
   - **b.** Hyperbaric oxygen should be considered if >50 Gy is used
   - **c.** Implant surgery is best carried out >21 days before radiation therapy
   - **d.** Implant placement is best deferred 3 months after radiation therapy

This chapter was developed from Chapter 75 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Solutions

1. Answer: b

Explanation: Based on the clinical presentation, the tissues appear healthly with an adequate band of keratinized tissue. There is a discrepancy between the gingival margins of canines and central incisor. A horizontal ridge defect around the edentulous area is visible in both lateral and palatal views.

2. Answer: c

Explanation: CBCT will provide more information on the buccolingual bone width, which will allow the clinician to plan the treatment better.

3. Answer: c

Explanation: The interocclusal dimension will vary depending on implant design and manufacturer component dimensions. However, an interocclusal dimension of 7 mm is considered ideal for uncomplicated implant restorations. When this dimension is < 5 mm, it is prudent to select screw-retained abutments to avoid prosthetic complications.

4. Answer: d

Explanation: It is recommended to wait for 9 months following radiation therapy before implant placement.

References

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42

Diagnostic Imaging for the Implant Patient

Relevant Terminology

Terminology / Abbreviation	Explanation
ALADA	Acronym meaning "as low as diagnostically acceptable." This principle emphasizes the importance of using the lowest possible field of view (FOV) to get the clinical benefit.
ALARA	Acronym meaning "as low as reasonably achievable." It underscores the importance of carefully evaluating the risk/benefit ratio before subjecting a patient to radiation.
CT versus CBCT	Computed tomography (CT) or medical CT uses a single x-ray source that produces a fan beam (vs. cone beam computed tomography, CBCT). CT equipment is primarily located in hospitals and the procedure is more expensive than CBCT.
DICOM	Digital Imaging and Communications in Medicine—the universally accepted standard file format for transmitting, storing, retrieving, printing, processing, and displaying information in medical imaging
dynamic navigation	Dental implant placement technique that utilizes special software and sensing devices to allow a clinician to place an implant into a planned position in a more real-time, interactive manner
field of view	Dictates the extent of the imaged volume. A small field of view will have a small extent but with higher resolution, and vice versa.
guided surgery	An implant placement protocol in which the clinician uses a surgical guide fabricated based on the implant position which was planned using a simulation program. The goal is to improve the accuracy of implant placement, especially in cases where the available bone volume is minimal or there is a key anatomic structure in the vicinity.
simulation programs	Specialized third-party software program that utilizes patients' CBCT data to help clinicians plan the implant position in a 3D format

Fast Facts

Objectives of diagnostic imaging of implant patient	• Identify anatomic structures and pathology • Measure quantity, quality, and location of bone
Advantages of periapical radiographs	• Low cost • Can be obtained easily • Low radiation to the patient
Limitations of periapical radiographs	• Provide only two-dimensional data for a three-dimensional structure • Limited field of view • Potential for overlapping • Potential for foreshortening or elongation

Continued

Fast Facts—cont'd

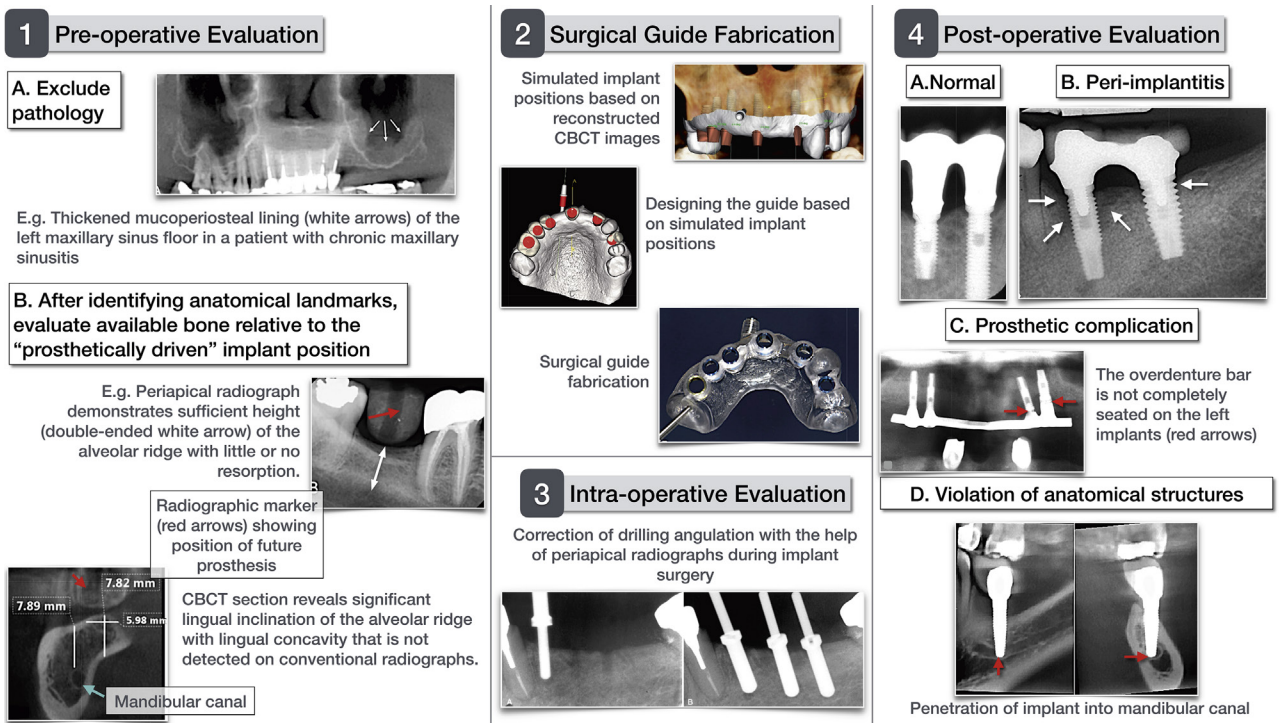
Advantages of CBCT	• Representation (provides cross-sectional view of the ridge) • No magnification • Sufficient detail • Reasonable radiation dose for the information obtained • Digital format • Helps with digital planning and guided surgical protocol • Broad field of view
Limitations of CBCT	• Relatively expensive • Requires special equipment
Key anatomic landmarks in maxilla	• Maxillary sinus • Nasal cavity • Nasopalatine foramen
Key anatomic landmarks in mandible	• Mandibular canal • Anterior loop of mandibular canal • Mental foramen • Submandibular fossa • Midline lingual foramen
Intraoperative uses of imaging	• Allows the clinician to know the depth and angulation of drilling that precedes implant placement and to make the appropriate modifications • Helps to confirm the depth and angulation of the placed implant • avoid key anatomic structures during implant placement
Postoperative uses of imaging	• Allows monitoring of crestal bone levels over time • Helps with early diagnosis of periimplantitis-related bone loss

Core Knowledge

Objectives of Diagnostic Imaging

Objectives of diagnostic imaging in the implant patient include:

- **Preoperative evaluation:**
  1. To assess adjacent teeth and dentition for dental caries and endodontic and periodontic problems
  2. To evaluate extraction sites for bone dimensions (height and width of alveolar bone are assessed to determine the optimum implant size and need for hard tissue grafting procedures)
  3. To evaluate bone around teeth with hopeless prognosis, to check feasibility and plan future implant treatment
  4. To assess three-dimensional orientation of the bone in relation to to the planned orientation of the future implant-supported prosthesis using radiographic guides
  5. To locate important anatomic structures (e.g., mandibular nerve, maxillary sinus, mental nerve, roots of adjacent teeth tilting into proposed implant space) that need to be considered in surgical implant placement
  6. To evaluate bone quality or density. Note: This is difficult to assess accurately using imaging procedures; surgical drilling provides the best way to assess bone density accurately.
- **Surgical template fabrication:** Based on information from the preoperative evaluation, imaging files are used in conjunction with design software packages to fabricate surgical templates that can guide precise surgical implant placement.
- **Intraoperative radiographic assessment of implant placement:** Because of the ease of acquisition and high resolution, periapical radiographs are most commonly taken *during* surgery to evaluate proximity to important anatomic structures. Sequential periapical radiographs guide the clinician to make appropriate angulation changes while drilling. The direction and depth of the drilling procedure, and parallelism to adjacent teeth and other implants, can be maintained correctly with the help of radiographic imaging.
- **Postoperative monitoring of implant:** This is performed to ensure healthy functioning of the implant prosthesis under occlusal loads over time. Imaging features associated with successful implant osseointegration and prosthetic function include:
  1. Absence of characteristic radiolucency around the implant body which must display a sharp thread image (implant threads must be distinguishable and not overlapping) abutting directly with bone (obtained with intraoral radiography using paralleling techniques).
  2. A maximum of 0.1 mm marginal or crestal bone loss per year after the first year of prosthetic loading around the implant-abutment connection/interface.
  3. Absence of gaps between various implant components (fixture, abutment, prosthesis).



• **Fig. 42.1** Diagnostic Imaging in Implant Dentistry: Objectives and Workflow. CBCT, cone beam computed tomography. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

Radiographs are also highly valuable in detecting excess cement, especially if it is radio-opaque. Excess cement retains plaque and is associated with biologic implant complications; it should therefore should be prevented or removed.

Fig. 42.1 shows a diagnostic imaging workflow that fulfills these objectives.

Diagnostic Imaging Techniques Used in Implant Dentistry

All diagnostic images, regardless of technique, should be evaluated to identify or exclude pathology and to identify normal anatomic structures. Imaging modalities for implant diagnosis and evaluation include:

- intraoral periapical radiography
- panoramic radiography
- cone beam computed tomography (CBCT)

A comparison of the most common imaging modalities used in evaluating the implant patient is presented in Table 42.1.

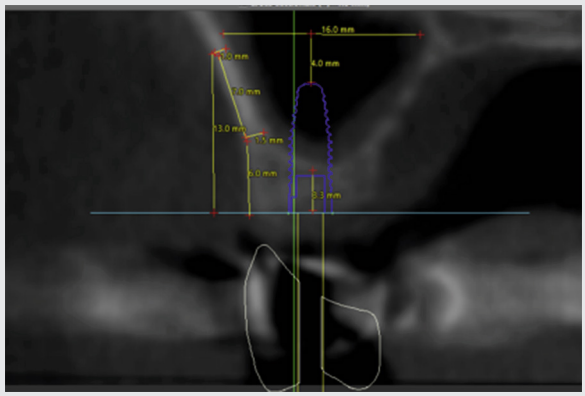
TABLE 42.1 Characteristics of Diagnostic Imaging Techniques Used in Implant Dentistry

	2D Imaging		3D Imaging
	Intraoral (Periapical)	Panoramic	CBCT
Uses	• Preoperative evaluation • Intraoperative monitoring of implant osteotomy and placement • Postoperative monitoring of osseointegrated implant	• Preoperative evaluation • Postoperative monitoring	• Detailed, precise planning of implant placement • Surgical template fabrication
Radiation dose	Low	Low	Low
Cost	Inexpensive	Inexpensive	Relatively expensive
Magnification	Unpredictable	Unpredictable; unequal magnification in vertical and horizontal dimensions	No distortion due to magnification

2D, two-dimensional; 3D, three-dimensional; CBCT, cone beam computed tomography.

CASE-BASED LEARNING EXERCISE

Scenario: A 63-year-old male presented with the chief complaint: "I want to restore my missing tooth in the upper right area." No significant medical conditions were noted. During the initial clinical examination, ridge deformity in the edentulous area #13 was observed (Seibert class III). Upon radiographic examination, a limited bone height was noted, due to possible sinus pneumatization and alveolar bone loss after the extraction of #13. A cone beam computed tomography (CBCT) was ordered.



CLINICAL CORRELATE

What basic tenet must be kept in mind when choosing between various imaging modalities?

ALARA (as low as reasonably achievable) principle—the objective is to select the technique most appropriate for obtaining optimal information with the minimum radiation dose and the lowest financial cost. For example:

- For effectively assessing multiple potential sites for implant placement (e.g. multiple missing teeth in partially edentulous cases or even in completely edentulous cases), opting for a CBCT would be a better choice.
- For evaluating a single potential implant site (e.g., in a case of straightforward implant placement at a site that is free from anatomic constraints and has sufficient bone volume to receive the implant), an intraoral periapical or panoramic radiograph may be sufficient.

Questions

1. The radiographic technique that produces the least distortion is:
   a. Panoramic radiograph.
   b. Periapical radiograph.
   c. Bite-wing radiograph.
   d. CBCT.
2. A three-dimensional analysis of the surgical site using CBCT will provide the following information, except for:
   a. Apicocoronal bone height
   b. Sinus proximity and need for sinus grafting.
   c. Buccolingual bone width.
   d. Type of prosthesis to be used.
3. When considering a lateral window approach for maxillary sinus augmentation, what is the key artery (space) that might be identified by CBCT?
   a. Infraorbital
   b. Ophthalmic
   c. Facial
   d. Posterior superior alveolar
4. What is the acceptable maximum marginal bone loss (mm/year) after implant loading, as measured using serial radiographs?
   a. 0.05
   b. 0.1
   c. 0.15
   d. 0.2

This chapter was developed from Chapter 76 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Solutions

1. Answer: d
Explanation: With a relatively low radiation dose, CBCT provides images with least distortion.

2. Answer: d
Explanation: All the other variables listed are obtainable from CBCT analysis.

3. Answer: d
Explanation: In some cases, during a lateral window approach the posterior superior alveolar artery can traverse within the bone in the vicinity of the window preparation.

4. Answer: b
Explanation: A maximum of 0.1 mm marginal or crestal bone loss per year after the first year of prosthetic loading around the implant-abutment connection is acceptable.

43

Dental Implants: Prosthetic Considerations

Relevant Terminology

Terminology	Explanation
cantilevered bridge	A type of tooth or implant-supported restoration in which the pontic that replaces the missing tooth is supported by an abutment (implant-supported or tooth-supported) on only one side
cement-retained crown	A crown that is retained to the underlying implant abutment by means of cement. There is no access screw hole in this type of crown versus a screw-retained or hybrid crown.
conventional loading	Restoration of the implant after a healing period of 2 months[1]
early loading	Loading of the implant between 1 week and 2 months of healing.[1]
emergence profile	The contour of a tooth or restoration where it emerges from the gingiva. The shape and form of the abutment from the implant platform to the gingival marginal level (the transmucosal component) dictate the emergence profile of the overlying restoration.
hybrid crown	Also called a "screwmentable" crown, this system consists of a crown that is cemented to a stock titanium abutment extraorally and then secured into place via an access screw hole on the crown surface. This has the advantage of preventing retention of excess cement within the sulcus and its easy visual identification also makes the abutment screw accessible for easy retrieval during maintenance visits.
immediate loading	Restoration of the implant within 1 week of implant placement[1]
immediate restoration	Similar to immediate loading, but the restoration is intentionally left out of any functional occlusion[1]
platform-switched implant	Platform switching relies on using narrower abutments on wide-diameter implants. Platform-switched implants maintain more crestal bone due to: • Movement of the implant-abutment connection away from bone margins • Less leakage, less screw loosening, and less stress on peri-implant bone • Growth of connective tissue over the implant shoulder at the level of platform switching, which provides an improved mucosal barrier seal and protection for crestal bone
screw-retained crown	A crown that is connected to the underlying implant and abutment by means of a screw
solid body implant	An implant type that contains both the body of the implant and the abutment as one single unit. The restoration on a solid body implant can only be retained by cement, which is a limitation; also, angulation corrections are not possible. For these reasons, they are not very popular.

Fast Facts

Anticipated papilla height	For papilla to be present, the distance from the contact point between two natural teeth to the crestal bone should not exceed 5 mm[2]
Cement and peri-implant complications	Excess cement during crown cementation has been shown to be associated with signs of peri-implant disease[3]
Major implant prosthetic complications	• Implant failure • Atypical implant bone loss • Persistent inflammation of the soft tissue • Infection of the peri-implant tissues • Failure of porcelain (requiring replacement) • Loss of the prosthesis • Fractured screws, abutments or implants

Continued

Fast Facts—cont'd

Minor implant prosthetic complications	• Screw loosening • Chipped porcelain (not requiring replacement) • Decementation of the prosthesis
Advantages and disadvantages of external hex connection	Advantages: • Greater flexibility and extensive prosthetic options • Greater immunity to implant body fracture. Disadvantages: • Screw loosening is common • Relatively more crestal bone loss is noted

Core Knowledge

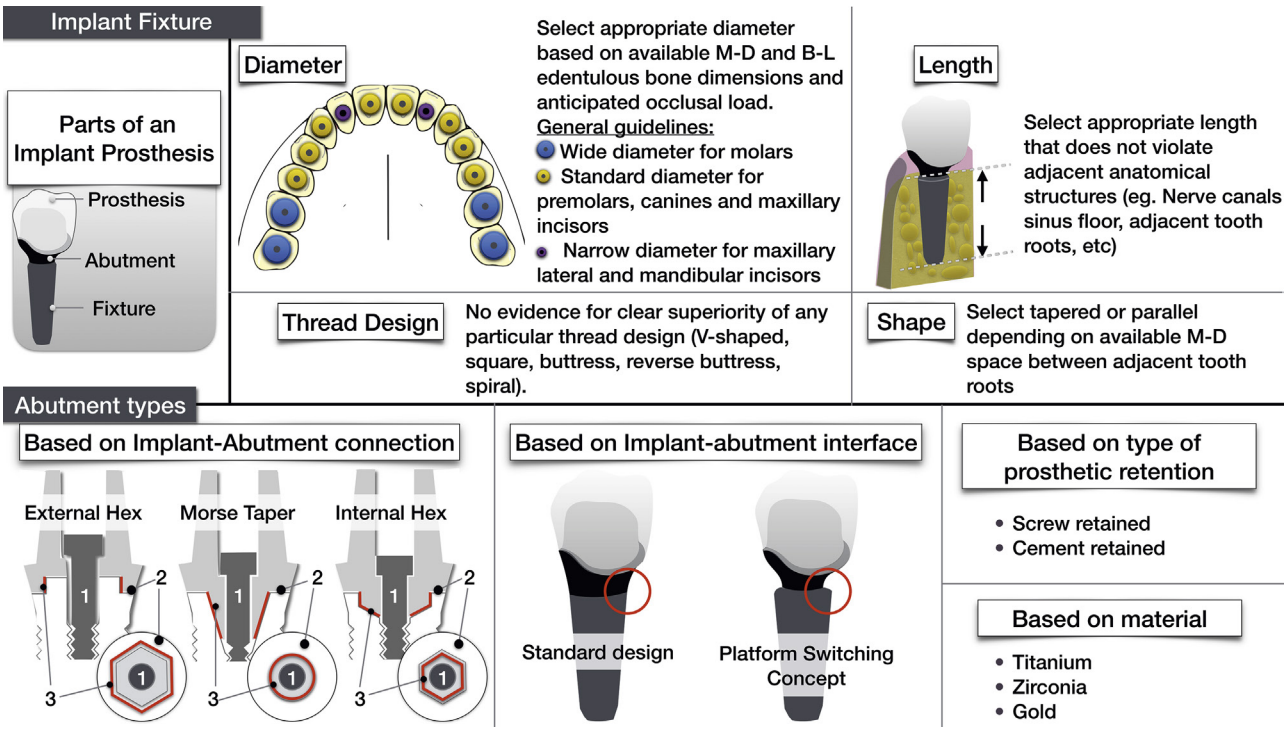
Parts of an Implant and Prosthetic Considerations

Implant dentistry is a prosthetically driven branch of dentistry, and it is important to have a basic understanding of prosthetic considerations in order to successfully practice dental implantology. This includes a working knowledge of the various parts of an implant prosthesis, the materials used, and the types of connections between the various parts. Fig. 43.1 illustrates the parts of an implant-supported prosthesis and the various considerations for prosthetic treatment planning.

CLINICAL CORRELATE

What basic precautions must be taken while fabricating implant-retained provisional restorations in order to maintain periimplant health?

- The implant provisional should be screw retained because (1) the use of cement should be avoided in fresh surgical sites and (2) the ability to remove, modify, and replace the restoration is essential in this phase (which is easier with screw-retained provisionals than cement-retained ones).
- The emergence zone of the provisional must be smooth and well polished.
- The most apical 2 mm of the provisional should be clean titanium (not polyether ether ketone [PEEK], composite, or acrylic) due to its proven effects on the soft tissues and minimal risk of breakage.



• **Fig. 43.1** Choice of Implant Fixture and Abutment: Prosthetic Considerations. This figure explains the major prosthetic considerations in choosing the right implant fixture and abutment types for a clinical scenario.

Continued

The gray box in the left corner shows the three basic parts of any implant-supported prosthesis: the fixture (titanium portion screwed into bone), the abutment (portion that connects fixture within bone to prosthetic portion in oral cavity), and the actual prosthesis (e.g., crown, bridge, overdenture).

Key implant features and selection considerations:

- **Implant diameter**—most implant companies manufacture implants with diameters between 3 mm and 7 mm. The prosthetic advantages of larger implant diameters must be balanced with the surgical considerations of available bone width.
- **Implant length**—longer implants (>10 mm) are indicated where apical anchorage is vital for achieving good primary stability (e.g., in immediate implant placement, or when dealing with poor bone quality). Shorter implants (4–8 mm) may be used in particular conditions (e.g. to avoid bone grafting due to lack of bone height).
- **Implant thread design**—greater thread depth improves bone-implant surface contact and primary stability. It is indicated in cases of poor bone quality where high occlusal loading is anticipated. It is highly recommended to exercise caution while interpreting data on implant thread design, as most of it is based on 'finite element analysis' method, which is considered a 'theoretical' model.
- **Implant shape**—cylindrical (parallel) and tapered implants are the most common designs.

The use of larger implants becomes more important under the following circumstances: enlarged masseter/temporalis muscles, a history of broken teeth and crowns, opposing other implants, and patients with parafunctional habits who are unwilling to wear an occlusal guard.

Abutment designs are based primarily on:

- **Implant-abutment connection**—the figure shows both a cross-sectional view and a top view of the implant crestal platform for each type of connection.

Key: 1. Screw hole with abutment screw (dark gray); 2. Implant platform on which the abutment rests (white); 3. The surface of the abutment which slides over the external connection or slots into the internal connection of the fixture (red lines).

There are three major types of abutment connections with the implant fixture:

(a) External hexagon: earlier implants were designed with this type of connection. It features a butt joint between the abutment and fixture over an external hexagon (i.e., the hexagon projects upward from the implant surface). It is well-suited for fixed implant restorations in fully edentulous patients. The primary drawback of the external hex connection is screw loosening.

(b) Morse taper: an internal connection; a slot is found within the implant fixture at the coronal portion. The slot is conical with a slight tapered design. The rationale for incorporating a taper into the connection is to further stabilize it with a strong friction fit, minimizing leakage, abutment movement, and loosening of screws.

(c) Internal hexagon: the hexagonal connection surface is internally placed within the implant fixture instead of projecting out coronally.

- **Implant-abutment interface**—The existence of a 'microgap' between the implant and the abutment allows for a threat of bacterial colonization in this location and this is believed to influence peri-implant bone morphology. The standard designs of this interface have an abutment base diameter similar to that of the implant platform/head. The "platform switching concept" involves making the abutment narrower than the implant platform. This moves the microgap region horizontally inward and away from the bone margins. This is thought to allow increased blood flow around the implant, providing sufficient room for the soft tissues whilst limiting crestal bone loss.
- **Type of prosthetic retention**—the prosthesis can be cemented over the abutment or screwed into the abutment and implant fixture, depending on the relationship of abutment finish lines to the gingival margins and available interocclusal space. If the finish lines are placed deep into the sulcus, cement is difficult to clean out and screw-retained restorations are preferred over implants. If interocclusal space is very limited (e.g., <4 mm), screw-retained restorations are better than cement-retained restorations.
- **Type of material**—abutments can be made of titanium, zirconia, or gold.

All abutments are connected to the fixture using a titanium abutment screw that can be torque-tightened.

B-L, buccolingual; *M-D*, mesiodistal.

Questions

1 What is the advantage of a screw-retained prosthesis over a cement-retained prosthesis?
- **a** Esthetics
- **b.** Ease of fabrication
- **c.** Ease of retrieval
- **d.** None

2 Considering the high smile line in this patient, what is the most appropriate material for the definitive abutment?
- **a** Gold alloy
- **b.** Titanium
- **c.** Ceramic
- **d.** Titanium alloy

3 To ensure increased accuracy, what type of impression technique and material would you advise the dentist to use to restore the #8 implant?
- **a** Closed tray/polyether
- **b.** Open tray/polyether
- **c.** Closed tray/irreversible hydrocolloid
- **d.** Open tray/irreversible hydrocolloid

4 To verify that impression copings are properly seated over implants, _____.
a. A radiograph is exposed
b. Tactile sense is satisfactory
c. No clinical check is necessary
d. Ask if the patient is comfortable

This chapter was developed from Chapter 77 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Solutions

1. **Answer: c**
Explanation: Compared with the cement-retained restoration, the screw-retained prosthesis is easier to retrieve.
2. **Answer: c**
Explanation: Given that the implant is located in an esthetic area, a definitive abutment fabricated in ceramic is recommended.
3. **Answer: b**
Explanation: The accuracy of impressions with polyether is higher than with irreversible hydrocolloid. For #8, where registering the soft tissue contour in the impression is essential, it is recommended to use an open-tray technique.
4. **Answer: a**
Explanation: In order to assess the proper seating, a periapical radiograph is required. The other options are not acceptable.

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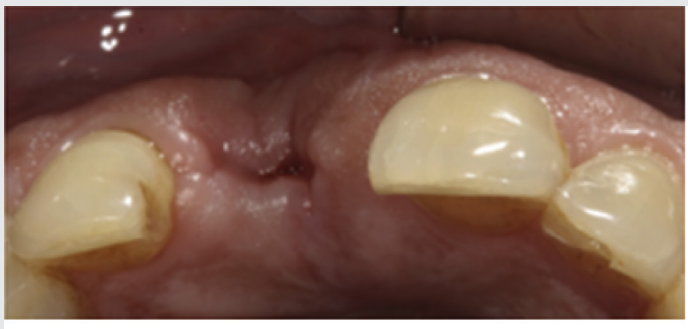
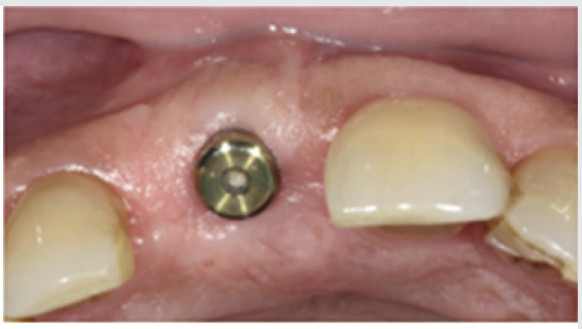
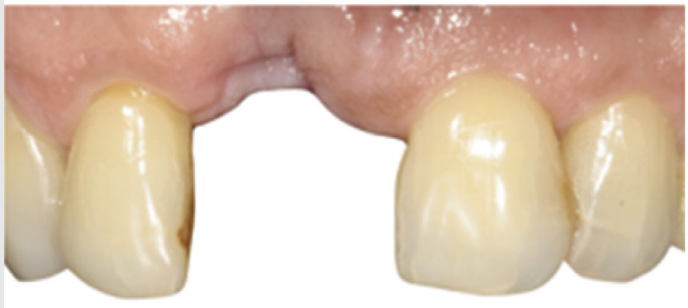
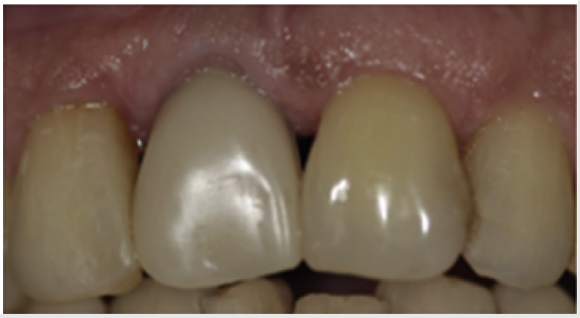
CASE-BASED LEARNING EXERCISE

Scenario: A 63-year-old male patient presented with the chief complaint: "I want to restore my missing tooth." His medical history showed no relevant conditions and he was not taking any medications. The patient's dental history revealed that #8 had been extracted on an emergency basis, and no bone grafting was done at the time of extraction. After site development was achieved, an implant was placed at site #8. In order to assure soft tissue conditioning, a screw-retained provisional was fabricated.

44

Implant Surgical Procedures

Relevant Terminology

Terminology	Explanation
bone tapping	Procedure done in select cases (highly dense bone or when placing a longer implant in moderately dense bone), in which an internal thread pattern is created at the osteotomy site to allow placement of the implant such that it doesn't require excess torque / force
countersinking	Procedure done in select cases using a countersink bur that widens the osteotomy at the bone crestal area to allow subcrestal placement of implant collar and cover screw
guide pins	Metal pins of increasing diameters used during bone drilling (osteotomy widening) to verify the three-dimensional angulation of the drilling process and its relationship to the opposing dentition. Typically, radiographs are taken during the drilling process with a guide pin of appropriate diameter fully seated in the osteotomy site.
timing of implant placement	• Immediate placement—a clinical protocol in which a dental implant is placed into the socket after tooth extraction in the same appointment • Early placement—placement of an implant after tooth extraction but with adequate soft tissue healing (typically 4–8 weeks after extraction); thus primary closure can be achieved at the time of placement (unlike with immediate placement). This protocol may be associated with simultaneous bone grafting at the time of implant placement. • Late placement—a clinical protocol in which a dental implant is placed into the alveolar ridge after adequate healing (typically 6 months after extraction).[2]
timing of final implant restoration	Immediate loading: A clinical protocol in which an implant is provisionalized with a crown and loaded (set in occlusion) on the day of implant placement or within 1 week of implant placement. This is different from an immediate restoration protocol, where the implant is provisionalized on the day of placement but the crown is left out of occlusion. Early loading: implant is prosthetically restored within 1week to 2 months of placement within bone and set in functional occlusion. Conventional loading: implant is prosthetically restored typically after 2 months of placement within bone and set in functional occlusion.[2]
nonsubmerged implant (one-stage protocol)	Procedure that involves attaching a healing abutment (instead of a cover screw) to the implant and leaving it exposed to the oral cavity
implant osteotomy	The process of sequential drilling to create a socket in bone to receive an implant
primary vs. secondary implant stability	Primary stability is mechanical stability offered by physical means (by the bone) at the time of placement, while secondary stability is offered by the biologic interaction of bone with the implant surface (osseointegration) following implant placement
second stage procedure	Procedure done to expose the osseointegrated implant in the two-stage protocol. It can be done by using a tissue punch or by reflecting a full thickness flap.
self-tapping implants	Implants with sharp, cutting thread patterns will not require additional bone tapping. These are called self-tapping implants.
submerged implant (two-stage protocol)	Procedure that involves submerging an implant at the time of placement and necessitating a second stage procedure to uncover the implant. A cover screw is placed into the implant before submerging it.
surgical guide	An appliance used at the time of implant placement that guides the surgeon to place the implant in a prosthetically-driven pre-planned position

Fast Facts

drill sequence	Usually starts with a round bur to mark the osteotomy site, followed by a series of drills of increasing diameter. Based on need, screw tapping and countersinking are performed using special drills, prior to implant placement.
Irrigation during implant placement	Copious irrigation is critical during drillings to reduce heat and flush out debris, because these factors can have an adverse effect on bone healing
drill speeds	Higher speeds (~800–1500 rpm) are usually used for initial drills; tapping and countersinking are done at lower speeds. Implant placement is done at very low speeds (~25 rpm).
postoperative regimen	• Antibiotic therapy (with extensive surgery, or when the patient is immunocompromised) • Pain medication • Antiseptic mouth rinse
radiographs during implant placement	Radiography allows verification of: • Implant mesiodistal angulation and depth of placement • Location of drills or the implant body in relation to anatomic structures (adjacent roots, sinus floor, or inferior alveolar nerve canal)

Core Knowledge

Introduction

Most implant surgical procedures can be done in the dental office under local anesthesia or conscious sedation (oral or intravenous). Regardless of the surgical approach, the implant must be placed in bone with good primary stability to achieve predictable osseointegration, and an atraumatic technique must be followed to avoid damaging the bone. Compact bone offers a much greater surface area for bone-to-implant contact (BIC) than cancellous bone.

Two types of fundamental implant surgeries are performed:

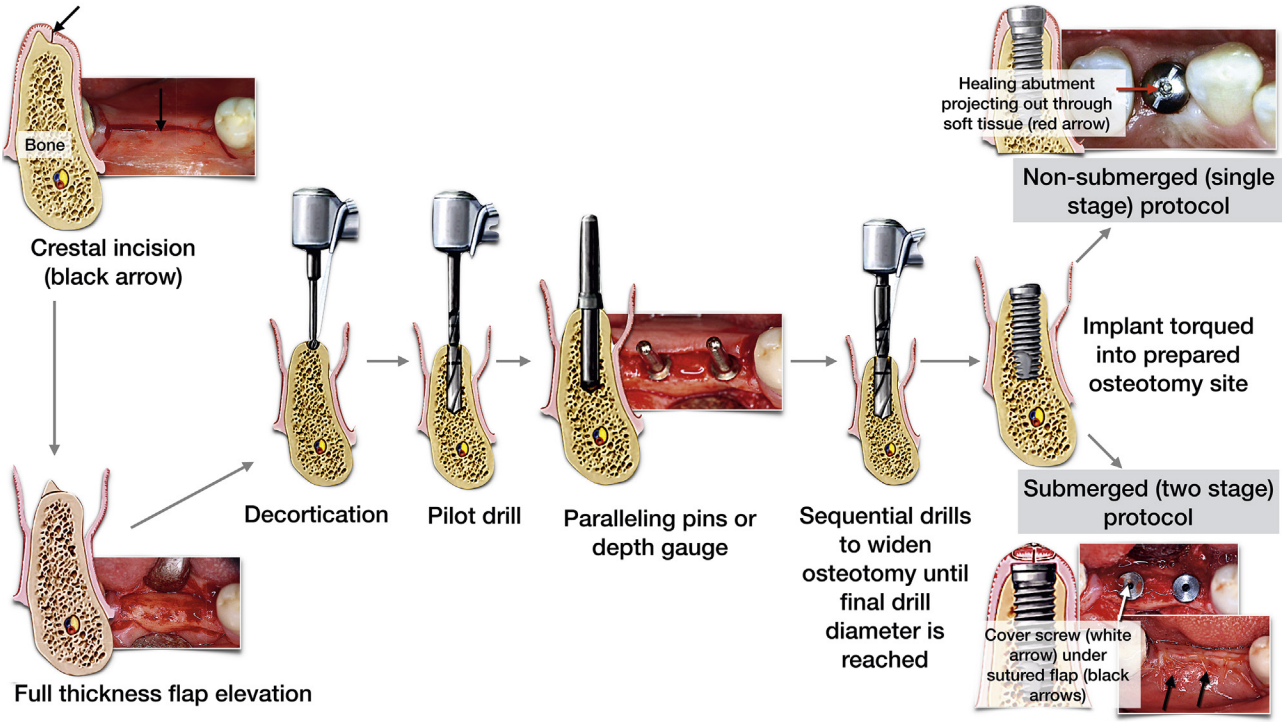
- One-stage placement, in which the implant is placed and left exposed to the oral cavity
- Two-stage placement, in which the implant is placed but submerged within gingival/mucosal tissues, and later exposed by a separate procedure

Fig. 44.1 describes basic implant surgical procedures.

CLINICAL CORRELATE

What are the factors a clinician must consider when faced with the dilemma of whether to submerge an implant or not?

- Primary stability and bone density: When primary stability is >35 Ncm, a nonsubmerged protocol may be safely chosen. However, when implants are placed in soft bone with poor primary stability, submerged two-staged protocol is employed so that micromovements that transmit unwanted loading forces at the bone-implant interface may be minimized during healing. Minimizing micromovements that potentially lead to soft tissue encapsulation of the implant (during the healing phase) increases the chances of successful implant osseointegration.
- Hard/soft tissue augmentation: When barrier membranes and bone grafts are used for guided bone regeneration, it is expected that temporary prostheses could transmit excessive forces on the healing tissue, particularly in fully edentulous patients. It is better to use the submerged two-stage approach to allow for undisturbed healing when augmentation procedures are performed.



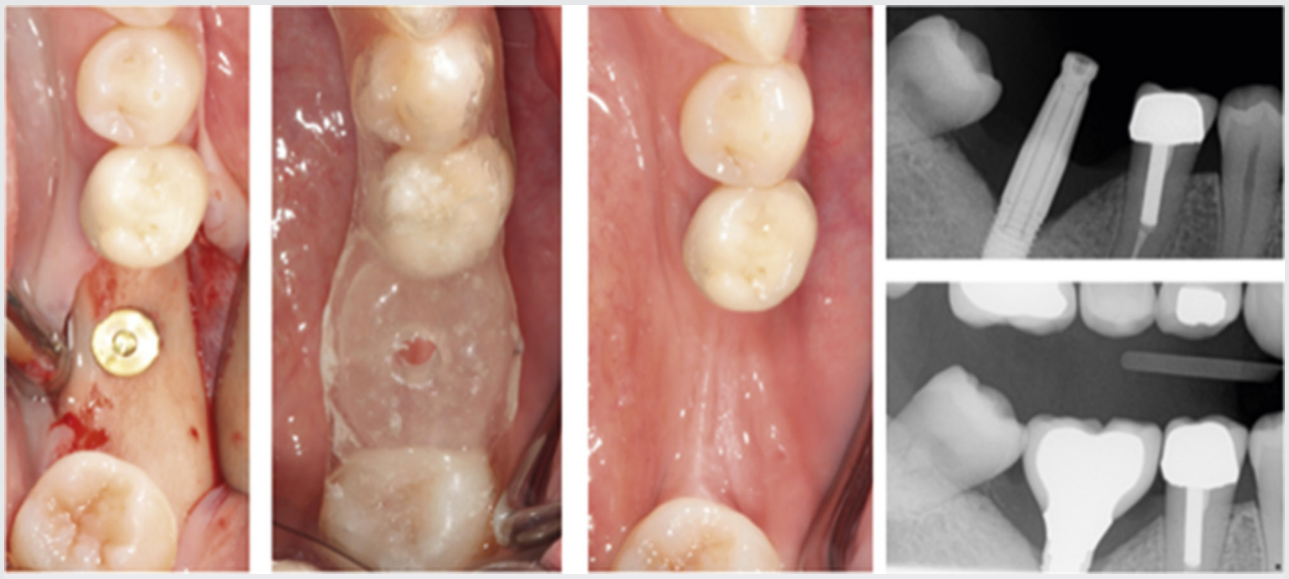
• **Fig. 44.1** Surgical Implant Placement: Standard Protocols. The figure shows the basic step-by-step procedure followed during implant site osteotomy and surgical implant placement within the prepared osteotomy site. The schematic diagrams showing cross-sectional views are accompanied by corresponding intraoral occlusal views wherever possible. Different manufacturers recommend different protocols for implant placement. The given description of the implant osteotomy procedure follows recommended standard protocols for a straightforward uncomplicated clinical scenario that does not require hard or soft tissue augmentations. The steps involved in implant placement are:

1. **Midcrestal incision** is placed in keratinized tissue, with or without vertical releasing incisions.
2. **Full thickness flap elevation** is performed to expose the underlying alveolar ridge.
3. **Implant osteotomy**—all drilling should be performed under copious saline irrigation (internal or external) to prevent overheating bone, at a speed of ~ 800–1500 rpm, using sharp drills, and with due consideration of the bone density. The drills should be intermittently and repeatedly "pumped" or pulled out of the osteotomy site while drilling to expose bone to the liquid coolant (chilled saline) and to facilitate clearing bone debris from the cutting surfaces. Drilling may be performed with or without a surgical guide. The steps involved in osteotomy are:
   i. The alveolar ridge may be optionally modified to present a broad, flat surface for implant placement. A sharp drill or round drill is used to perforate just the cortical bone and mark the precise location of the implant.
   ii. A pilot/twist drill (diameter 2 mm) is used to perform the initial osteotomy up to the appropriate depth in the correct direction within bone.
   iii. The angulation and depth of the osteotomy are checked and validated using paralleling pins/depth gauges and periapical radiographs. At this juncture, any correction in angulations can be made relatively easily using wider drills.
   iv. Sequentially wider drills are used to atraumatically and progressively widen the girth of the preparation until the final-diameter drill is used. The final drill diameter is usually smaller than the diameter of the implant, to aid in primary stability. In case of high bone density, additional drilling steps (e.g., screw tap, dense bone drill) are required to allow implant installation without excessive torquing.
4. **Implant installation**—the implant (of appropriate length and diameter) is placed within bone with the help of an implant driver adapted to a handpiece or torque wrench. Both bone tapping and implant insertion are done at very slow speeds (20–40 rpm).
5. **Wound closure protocols**—once the implant has been placed such that the implant shoulder is positioned at the marginal bone level, there are two approaches for closing the wound:
   - Two-stage (submerged) protocol: A cover screw is inserted into the implant. The flap is placed over the cover screw and sutured to completely cover the implant head. This protocol requires a second-stage surgery to uncover the implant cover screw for prosthetic procedures, because the soft tissues completely cover the entire wound.
   - One-stage (nonsubmerged) protocol: A healing abutment of appropriate diameter and height to transcend the peri-implant soft tissues is inserted into the implant. The flap tissue is adapted *around* the healing abutment and sutured. This protocol does not require another surgery for uncovering the implant. The prosthetic phase can begin directly after the healing period by just unscrewing the healing abutment to gain access to the implant platform for impression procedures.

(From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

CASE-BASED LEARNING EXERCISE

Scenario: A 32-year-old female presented with the chief complaint: "I lost a tooth years ago—I want to close the space". She reported no medical conditions. Dental history: clinical health with no significant probing depths, bleeding on probing, or mobility. The treatment plan for #30 was generated in collaboration with the restoring dentist.



Questions

1. Which technique is commonly employed to test the primary stability of dental implants?
   - **a.** Resonance frequency analysis
   - **b.** Percussion
   - **c.** Reverse torque test
   - **d.** Cutting (insertion) torque test
2. Immediate implant placement is the placement of:
   - **a.** An implant at the time of tooth extraction.
   - **b.** Multiple implants at once.
   - **c.** A restoration at the time of implant placement.
   - **d.** A restoration at the time of surgical uncovering.
3. What distance from the gingival aspect of the interproximal contact to the underlying alveolar bone crest will yield 100% complete papilla fill?
   - **a.** 8 mm
   - **b.** 5 mm
   - **c.** 6 mm
   - **d.** 7 mm
4. A full-thickness flap includes all of the following except one. Which is the exception?
   - **a.** Gingival epithelium
   - **b.** Gingival connective tissue
   - **c.** Cortical plate of bone
   - **d.** Periosteum

This chapter was developed from Chapter 78 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Solutions

1. **Answer: a**

Explanation: Resonance frequency analysis is a commonly utilized method to test the primary stability of the implant. All other techniques are not appropriate.

2. **Answer: a**

Explanation: Immediate implant placement is the placement of an implant at the time of tooth extraction. Introducing a restoration in function at the time of implant placement is called immediate loading.

3. **Answer: b**

Explanation: According to Tarnow,[1] a distance of 5 mm or less between the alveolar crest of the bone and the interproximal contact will yield 100% papilla fill.

4. **Answer: c**

Explanation: A full-thickness flap includes all of the choices listed except the cortical plate of bone.

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2. Gallucci GO, Hamilton A, Zhou W, Buser D, Chen S. Implant placement and loading protocols in partially edentulous patients: A systematic review. Clin Oral Implants Res. 2018;29 Suppl 16:106-134. doi:10.1111/clr.13276

45 Implant Site Development

Relevant Terminology

Terminology	Explanation
block grafts	Bone grafts (autografts or allografts) that are used in the block form (rather than as particles) to augment a ridge. These grafts are stabilized onto the ridge using special screws
crestal (indirect) approach sinus augmentation	In this approach, the maxillary sinus membrane is accessed through a crestal osteotomy that is made at the time of implant placement. This approach is therefore usually carried out simultaneously with implant placement
fenestration and dehiscence	A fenestration is a small window-like opening in the alveolar bone that tends to occur on the buccal aspect of a tooth or an implant. If the bone defect extends all the way to the crest, it is called a dehiscence
guided bone regeneration	A ridge augmentation procedure that employs the concepts of epithelial exclusion and space maintenance to enhance bone regeneration. This is achieved by the use of barrier membranes and particulate bone grafts
lateral window (direct) sinus augmentation	Clinical procedure done to augment bone right below the maxillary sinus by lifting the sinus membrane. In a lateral window or direct sinus augmentation procedure, the sinus membrane is accessed by making a window on the lateral aspect of the maxilla
particulate bone grafts	Bone grafts in the form of particles or granules with a specific size range that can be packed into bone defects or tooth sockets
ridge (bone) augmentation	Clinical procedure done separately from, or simultaneously with, implant placement with the goal of increasing the alveolar bone volume for a future implant, or augmenting bone circumferentially around a placed implant, respectively
ridge preservation	Clinical procedure performed immediately after tooth extraction with the goal of minimizing postextraction ridge resorption. Other terminologies used to describe this procedure include socket preservation and socket grafting
schneiderian membrane	The membrane that lines the internal aspect of the maxillary sinuses; it is pseudostratified, ciliated, and columnar in nature. In a sinus augmentation procedure, this is the membrane that is carefully lifted to provide room for bone grafting
tenting and tacking screws	Tenting screws are used underneath the membrane in a guided bone regeneration procedure to provide space maintenance; tacking screws are used to tack (secure) the membrane edges to the bone

Fast Facts

postextraction resorption	After extraction of a tooth the alveolar ridge undergoes resorption, especially in the buccolingual (width) dimension but also in the apicocoronal (height) dimension. Bone loss is more pronounced in the bucco-lingual dimension and is expected to occur within 6 months after tooth extraction
types of bone replacement grafts	Autografts (from the same patient), allografts (cadaver source), xenografts (animal source) and alloplasts (synthetic)
extraoral sources of autogenous bone grafts	Iliac crest or tibia

Continued

Fast Facts—cont'd

intraoral sources of autogenous bone grafts	Mandibular symphysis or ramus
rationale for ridge preservation	Ridge preservation has been shown to minimize resorption of the alveolar ridge following extraction
rationale for ridge augmentation	Ridge augmentation, when done prior to implant placement, is intended to increase the required bone volume for future implant placement in a prosthetically favorable position. When done at the time of implant placement (simultaneous approach), it is intended to increase circumferential bone around the placed implant
key steps in ridge preservation	Minimally traumatic extraction of the tooth, followed by packing of particulate bone graft into the socket and covering the graft with a barrier membrane
resorbable versus nonresorbable membranes	Resorbable membranes have the advantage of avoiding the need for a second procedure to remove the membrane, while nonresorbable membranes are generally better space maintainers

Core Knowledge

Introduction

Successful implant restorations require the correct placement of dental implants within hard and soft tissue envelopes in a prosthetically driven position. Standard protocols for surgical placement of implants in edentulous areas with adequate bone and soft tissue cover were reviewed in Chapter 44. However, such ideal conditions are not always encountered in clinical situations, and the need for both soft and hard tissue augmentation in deficient sites becomes evident. The topic of implant site development is quite vast, and the reader is referred to Chapter 79 of *Newman and Carranza's Clinical Periodontology* (13th ed.) for detailed reading. This chapter reviews the basic differences between various important surgical augmentation procedures used for implant site development and aims to clarify the rationale behind the different modalities.

Ridge Preservation and Ridge Augmentation

Definitions[2]

- **Ridge preservation**—procedures aimed at maintaining the ridge profile and preserving the ridge volume within the bony envelope that exists at the time of extraction (Fig. 45.1)
- **Ridge augmentation**—enlarging the ridge profile and increasing the ridge volume beyond the skeletal envelope that exists at the time of extraction (Fig. 45.2)

Classification

Ridge defects may be classified as:[3]

- **Class I**—Buccolingual (horizontal) deficiency with normal ridge height. Most predictable management of all defect types.
- **Class II**—Apicocoronal (vertical) deficiency with normal ridge width. More difficult to treat and usually managed by onlay grafts.
- **Class III**—Combination of buccolingual and apicocoronal ridge deficiencies. Most difficult type of defect to manage, requires multiple surgical procedures.

Augmentation procedures for implant site preparation will vary depending on the complexity of the defect. Major defects are better managed prior to implant placement, whereas minor defects can be addressed effectively at the time of implant placement.

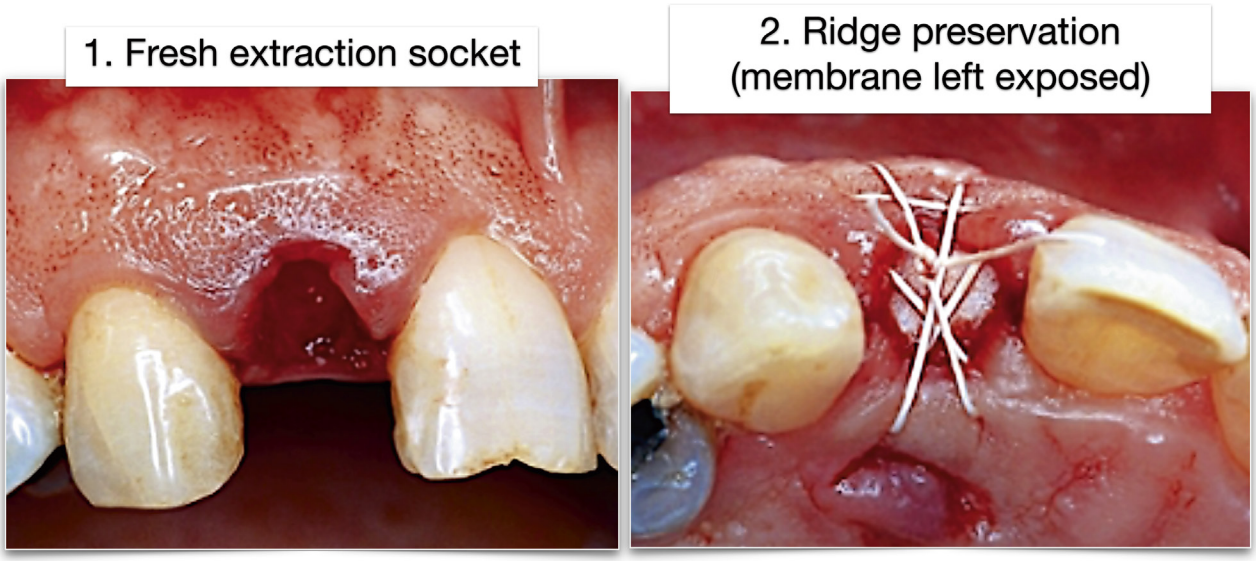
Table 45.1 compares the ridge preservation and ridge augmentation procedures.

Among the ridge augmentation procedures, guided bone regeneration (GBR) and block bone grafting are the most commonly performed. Table 45.2 compares these two procedures.

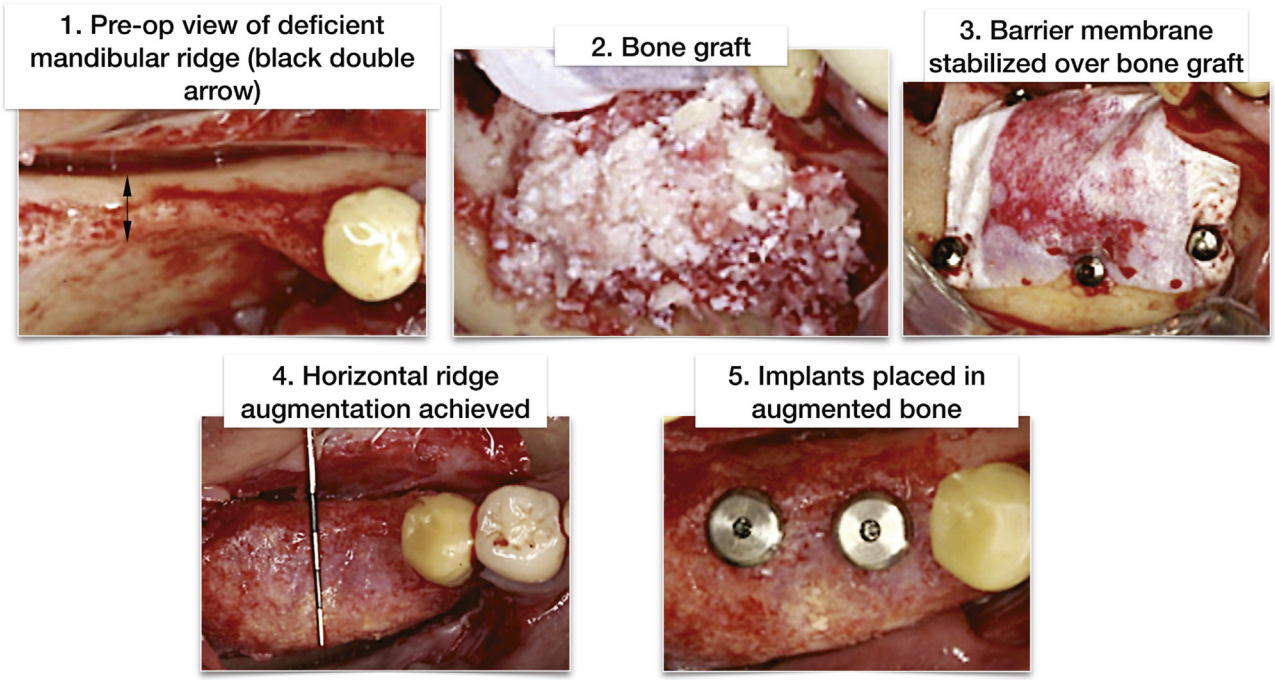
CLINICAL CORRELATE

Is there a benefit to performing ridge preservation procedures in postextraction sockets versus no such measures?

Yes. Multiple studies indicate that there is lesser ridge resorption when ridge preservation is done at the time of extraction using graft materials within sockets than in sockets without ridge preservation (i.e., where the extraction site is left to heal spontaneously). A recent systematic review indicated that ridge preservation prevents, on average, ~2 mm of resorption in the horizontal dimension, and 1.7 mm and 1.16 mm of bone loss in the vertical midbuccal and vertical midlingual dimensions, respectively.[1]



• **Fig. 45.1 Ridge Preservation.** (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)



• **Fig. 45.2 Ridge Augmentation.** (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

TABLE 45.1 Comparison of Ridge Preservation and Ridge Augmentation Procedures

	Ridge Preservation	Ridge Augmentation
Objective	To *prevent* bone resorption in postextraction sockets and hence minimize soft and hard tissue deficiencies May require a second surgery for ridge augmentation	To *correct* existing soft and hard tissue deficiencies in edentulous areas
Timing of procedure and rationale	Performed at the time of tooth extraction to limit the physiological alveolar bone resorption that normally follows tooth extraction	Performed after healing of the extraction socket (prior to or at the time of implant placement, or thereafter) to allow prosthetically driven placement of the implant in the correct position within the bone
Materials used	• **Used within sockets:** Autografts, allografts, bone substitutes, etc. • **Used as barrier membranes over sockets that may or may not be grafted:** Connective tissue autograft, free gingival graft, resorbable and nonresorbable membranes	• **Bone grafts:** Autografts (bone blocks or particulate grafts), allografts, xenografts, bone substitutes, etc. • **Barrier membranes:** Resorbable (e.g., collagen) and nonresorbable (titanium-reinforced ePTFE) membranes • **Distraction osteogenesis:** Vertical and horizontal distraction screws
Methods	• Flapless, with membranes exposed, with or without graft materials within socket • Repositioned flaps, with membranes fully covered, with or without graft materials within socket	Residual ridges can be augmented vertically and horizontally using the following methods: • Guided bone regeneration (GBR) • Block bone grafting: inlay and onlay grafting • Distraction osteogenesis • Ridge splitting/expansion and sandwiched graft materials (horizontal augmentation only)
Complications	• Nonintegrated graft particles compromising primary stability of implant • Infections of grafted materials or membranes • Soft tissue dehiscence and membrane exposure • Early implant failure • Loss of graft • Pain	• Nonintegrated graft particles compromising primary stability of implant • Infections of grafted materials or membranes • Soft tissue dehiscence and membrane exposure • Early implant failure • Loss of graft • Pain • Nerve dysfunction

ePTFE, expanded polytetrafluoroethylene

BASIC SCIENCE CORRELATE

What are the biologic requirements for bone regeneration that the various steps in GBR procedure meet and accomplish?

- **Blood supply:** Cortical perforations performed using round burs during surgery enhance the regional acceleratory phenomenon (RAP), which allows good vascular perfusion of the wound site and provides osteogenic cells from adjacent cancellous bone.
- **Graft stabilization:** Bone fixation screws and membrane tacks ensure that the barrier membrane is held in place, along with any grafted material. Movement of bone graft during the healing process can disrupt the blood flow, which can lead to fibrous healing rather than hard tissue formation and mineralization.
- **Cell exclusion:** Barrier membrane held in place over the graft material ensures that there is a confined space within which bone can regenerate without external interference from epithelial cells.
- **Space maintenance:** Tenting screws and bone graft materials support the barrier membrane and prevent its collapse.
- **Wound coverage:** Atraumatic flap management and proper flap design with tension-free suturing allow for proper covering of the wound site and healing of overlying soft tissues by primary intention.

Advanced Implant Surgical Procedures

These primarily include those procedures that deal with the more challenging presentations of severe vertical ridge deficiencies, such as:

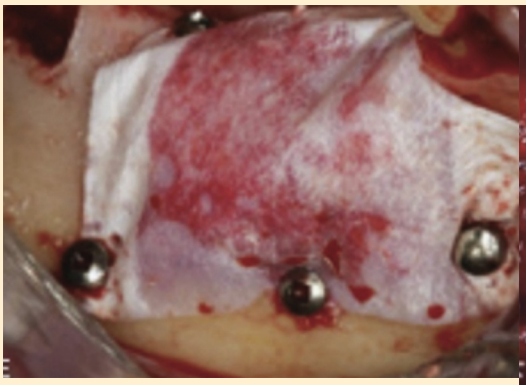
- Maxillary sinus lift procedures (lateral and crestal approaches)
- Vertical bone augmentation using GBR and distraction osteogenesis

TABLE 45.2 Comparison of Guided Bone Regeneration and Block Bone Grafting Procedures

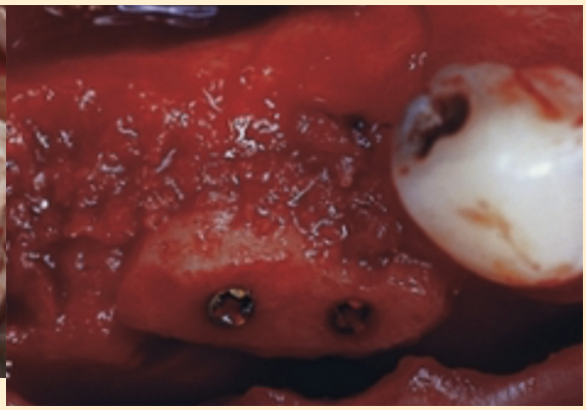
	Guided Bone Regeneration (GBR)	Block Bone Graft

Rationale	• GBR works by the principles of:
	  • Wound and clot stabilization
	  • Selective cellular exclusion
	  • Space maintenance
	• To accomplish these goals, membranes and bone graft particles are used:
	  • Role of membrane—to act as a barrier that prevents the epithelial and connective tissue cells (which have higher cellular kinetics than bone cells) from occupying and populating the space meant for bone ingrowth.
	  • Role of bone grafts—to specifically maintain space and act as a scaffold for the bone precursor cells to attach, proliferate, and differentiate into osteoblasts that produce bone matrix (osteoid), which eventually mineralizes to form mature bone that replaces grafted bone
		• Bone grafts from the same individual are the "gold standard" in bone reconstruction because they possess high osteogenic potential and are effective in managing severe horizontal alveolar bone loss
		• Autogenous bone grafts can be harvested for smaller defects from intraoral sites (ramus, chin, maxillary tuberosity, zygomatic buttress); for larger defects, extraoral sites (iliac crest, calvaria) can act as sources/donor sites.

Advantages	• GBR can be performed with allografts and xenografts with no donor site morbidity • Unlimited bone quantity available for grafting	• Horizontal alveolar deficiencies that might be challenging to reconstruct with particulate grafts can be more amenable to reconstruction with a monocortical block bone graft
Disadvantages	• Premature membrane or screw exposure causes infection or graft loss (incidence of postoperative infection is higher with GBR than with block bone grafts) • Resorbable membranes require graft material under them for space maintenance; they can collapse into the defect and limit bone growth • Nonresorbable membranes require a second surgery for removal	• Donor site morbidity (e.g., altered sensations due to nerve injury while harvesting bone block, pain, swelling, sinus perforation) • Substantial graft resorption • Limited quantity of bone (intraoral source) available for grafting • Biologic limitation of revascularizing large bone blocks • Primary closure is challenging • Grafted bone may be vulnerable to fracture or additional resorption during surgical implant placement



GBR using particulate bone graft and barrier membrane with screws



Block bone graft with fixation screws

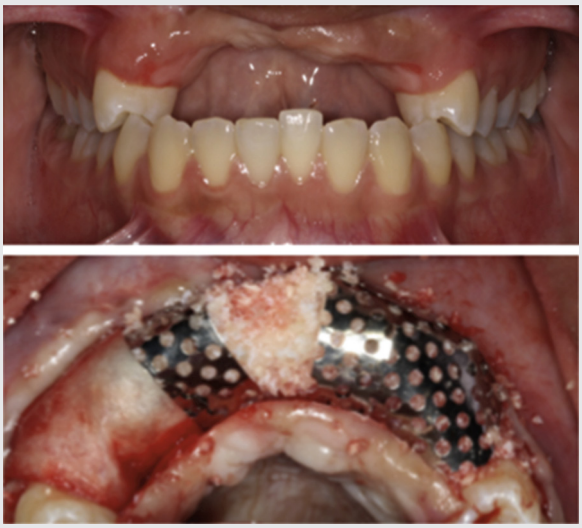
Clinical photo attribution: Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). Newman and Carranza's Clinical Periodontology (13th ed.). Philadelphia: Elsevier.

- Tissue engineering using growth factors for bone regeneration (e.g., autologous platelet-rich plasma, bone morphogenetic protein)

The reader is referred to Chapter 80 of *Newman and Carranza's Clinical Periodontology* (13th ed.) for detailed discussion of advanced implant surgical procedures.

CASE-BASED LEARNING EXERCISE

Scenario: An 18-year-old female presented with the chief complaint: "I want my smile back. I was in an accident and lost my front teeth". She reported no medical conditions. Dental history: clinical health with no significant probing depths, bleeding on probing, or mobility. The treatment plan for #6–11 was generated in collaboration with the restoring dentist. In order to address the malalignment on the mandibular arch, an orthodontist was consulted after the tooth vitality was confirmed by an endodontist.



Questions

1 In the case above, the dentist used a combination of bone graft materials. Bone graft from a bovine source is called:
   a. Autogenous graft.
   b. Allograft.
   c. Alloplast.
   d. Xenograft.

2 A bone graft with osteogenic, osteoconductive, and osteoinductive properties is a(n):
   a. Autogenous graft.
   b. Allograft.
   c. Alloplast.
   d. Xenograft.

3 #6-11 were extracted in the emergency room with no ridge preservation. In general, pronounced bone loss following extraction occurs in which dimension?
   a. Horizontal
   b. Vertical
   c. Neither of the above

4. As shown in the image, what type of membrane was used to repair the defect?
   a. Resorbable
   b. Nonresorbable
   c. Both

This chapter was developed from Chapter 79 and 80 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Case-Based Learning Exercise

Solutions

1. Answer: d

Explanation: Bone graft obtained for clinical use from species other than humans is called xenograft.

2. Answer: a

Explanation: Of the examples listed, the autogenous graft is the only bone graft with osteogenic, osteoconductive, and osteoinductive properties.

3. Answer: a

Explanation: Ridge resorption after extraction tends to be more pronounced in the buccolingual or horizontal dimension. Traumatic extraction can lead to bone loss in both horizontal and vertical dimensions.

4. Answer: c

Explanation: As shown in the clinical images, both resorbable (collagen) and nonresorbable (titanium mesh) membranes were used to regenerate the ridge deformity.

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46

Advances in Dental Implantology: Microsurgery, Piezosurgery, and Digitally Assisted Implant Surgery

Relevant Terminology

Terminology	Explanation
computer-guided implant placement	Implant placement performed using a special set of burs and employing a static guide fabricated based on the virtually planned implant position. The main objective of this approach is to improve the accuracy of implant placement (both the depth and angle of placement).
fiducial marker	Refers usually to a point or line used as a fixed reference for comparison of two entities; relevant in digitally assisted implant surgery, where a fiducial marker is used to relate the image data to the actual patient's anatomy *during* implant surgery.
microprecision	A characteristic of piezosurgery, made possible by mechanical microshock waves (in the range of ~80 μm) produced by a specific ultrasonic frequency
microsurgery	Surgical procedures performed under a high-magnification (≥ 10×) microscope
piezosurgical device	Unit consisting of an ultrasonic transducer powered by an ultrasonic generator, with the capacity to drive a range of specially designed cutting inserts
real-time micropositioning implant placement (RTMIS)	An approach that utilizes simultaneous real-time tracking or guidance to allow the clinician to place the implant in the planned position. In contrast to computer-guided implant placement, in this approach the interaction of instruments with the scanned image is viewed in *real time* by the clinician at the time of surgery.
selective cutting	Ability of a piezoelectric device to cut only mineralized tissues (rather than soft tissues)
virtual implant planning	Presurgical planning that allows the surgeon to virtually place the implant in a three-dimensional fashion, using specific software that utilizes a cone beam computed tomographic (CBCT) image. It allows the surgeon to select implants with appropriate dimensions, to plan the depth and angulation of placement of the implant, and to be well prepared for additional procedures, such as simultaneous hard tissue grafting.

Fast Facts

Proposed advantages of microsurgery	• Aids in minimally invasive surgical approach • Reduced postoperative morbidity and improved healing • Allows better ergonomic position for the surgeon

Continued

Fast Facts—cont'd

Types of piezo inserts	Based on function, inserts can be classified into: • Sharp—used for osteotomy and osteoplasty • Smoothing—used for smoothing action • Blunt—used to refine a cut Clinical classification (insert identification) codes include: • OT—used to perform osteotomy • OP—used to perform osteoplasty • EX—used to perform extraction • IM—used to perform implant site preparation
Advantages of piezosurgery	• Precise cuts in hard tissues (microprecision) • Selective cutting (cuts only the hard tissues) • Hemostasis (via cavitation), leading to improved visibility • Improved soft and hard tissue healing
Piezosurgery and bone healing	The vibration mimics bone loading and stimulates the release of growth factors such as bone morphogenetic protein (BMP) and transforming growth factor beta 2 (TGF-β2) that aid in bone healing
Applications of piezosurgery in periodontics	Nonsurgical procedures: • Scaling, planing, and debridement Surgical procedures: • Crown lengthening • Tooth extraction • Implant site preparation (osteotomy) • Lateral sinus window preparation
Steps in real-time micropositioning implant surgery	• Data acquisition • Identification • Registration • Navigation • Accuracy

Core Knowledge

Microsurgery

Microsurgery is a treatment approach comprising surgical procedures (and other nuanced dental procedures) performed under magnification, which allows for improved visualization. This refined surgical modality also employs basic optical magnification to aid in ergonomic techniques and technology, allowing the operator to maintain the correct posture while not compromising on a closer view of the field of operation. Microsurgery can contribute to improved esthetics, rapid healing, reduced morbidity, and enhanced patient acceptance. Fig. 46.1 reviews relevant concepts relating to the use of microsurgery in dental implantology.

Piezosurgery

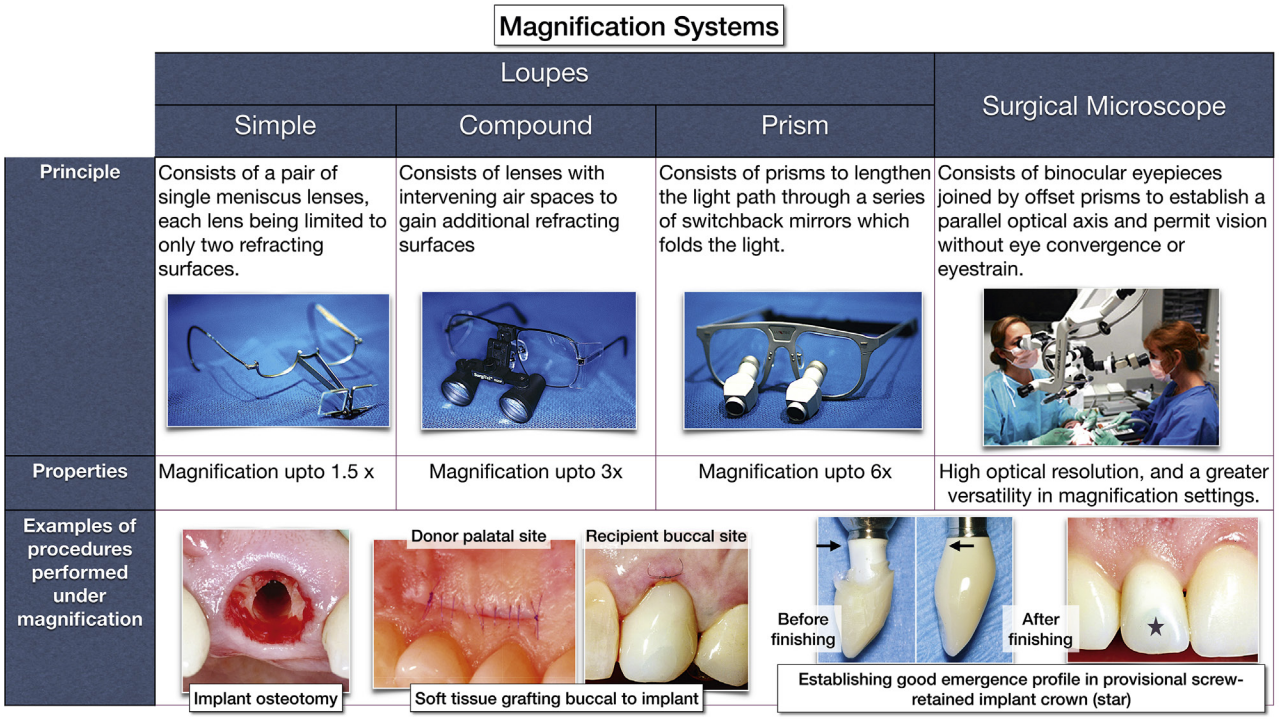
The important goal of minimal surgical trauma for optimal bone healing and regeneration is achieved by piezoelectric bone surgery, which includes extraordinary cutting properties and has many dental applications. Piezosurgery has the capacity to redefine the idea of minimally invasive surgery in bone-related procedures.

Working Principle

Piezosurgery devices employ ultrasonic vibrations to cut mineralized tissues. To this end, a primary frequency of 30 kHz is superimposed with a sound wave of 30-60Hz (called *frequency overmodulation*) to generate a hammering action that effectively cuts bone without harming soft tissues and with minimal heat production. This is termed *frequency overmodulation*.

Advantages of Ultrasonic Cutting

- **Microprecision**—the extraordinary surgical control that characterizes piezosurgery is the result of its linear microvibrations requiring only light pressure (a stroke similar to the smooth precision used to draw a picture) to be applied to the handpiece.
- **Selective cutting**—microvibrations of the piezosurgical tips are physically unable to cut soft tissue, where kinetic energy is easily dissipated. This helps to preserve the integrity of soft tissues (alveolar nerve, mental nerve, maxillary sinus membrane, blood vessels, etc.) while effectively cutting the mineralized tissue (bone) in close proximity to these tissues.
- **Maximum visibility**—this is achieved by creating a surgical field that is blood free during cutting because of the "cavitation" effect (nebulization of the cooling saline solution when it contacts the ultrasonically vibrating insert), which creates a microspray that cleans debris from the surgical field.
- **Excellent healing**—the improved hard tissue healing is due to stimulation of release of growth factors (e.g., bone morphogenetic protein) and inhibition of



• **Fig. 46.1** Microsurgery in Implant Dentistry. Microsurgery fundamentally refines surgical procedures for implant dentistry through improved motor coordination and is capable of providing a seamless esthetic solution for failing anterior teeth. The philosophy of microsurgery embraces three core values:

- **Enhanced motor skill** for better surgical performance—accomplished through improved visual acuity by magnification and the use of a precise hand grip to increase accuracy.
- **Minimal tissue trauma**—accomplished through smaller incisions and reduced surgical fields.
- **Primary passive wound closure**—accomplished by microsuturing (microsutures in the range of 6-0 to 9-0 are needed to approximate the wound edges accurately to eliminate gaps and dead spaces at the wound edge).

Magnifying systems:

- **Loupes**—fundamentally, loupes are telescopes with side-by-side lenses that converge to focus on the operative field (a convergent lens optical system is called a *Keplerian* optical system). The need for the clinician's eyes to converge on the operative field can result in eyestrain, fatigue, and pathologic vision changes, especially after prolonged use. The three types of loupes commonly used in periodontics are simple or single-element loupes, compound loupes, and prism telescopic loupes.
- **Surgical microscope**—Surgical microscopes designed for dentistry employ *Galilean* optics, having binocular eyepieces that permit vision without eye convergence or eyestrain. Fiber-optic coaxial illumination is a major advantage because it focuses light such that it eliminates shadows. High-definition video cameras capture still and video images simultaneously to permit **documentation** of procedures. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

expression of inflammatory factors following piezosurgery. Improved soft tissue healing has also been demonstrated in histologic studies, which showed less damage to periosteum elevated using exclusive piezosurgery tips than with manual use of periosteal elevator.

Fig. 46.2 shows some clinical applications of piezosurgery in implant dentistry.

Digitally Assisted Implant Surgery

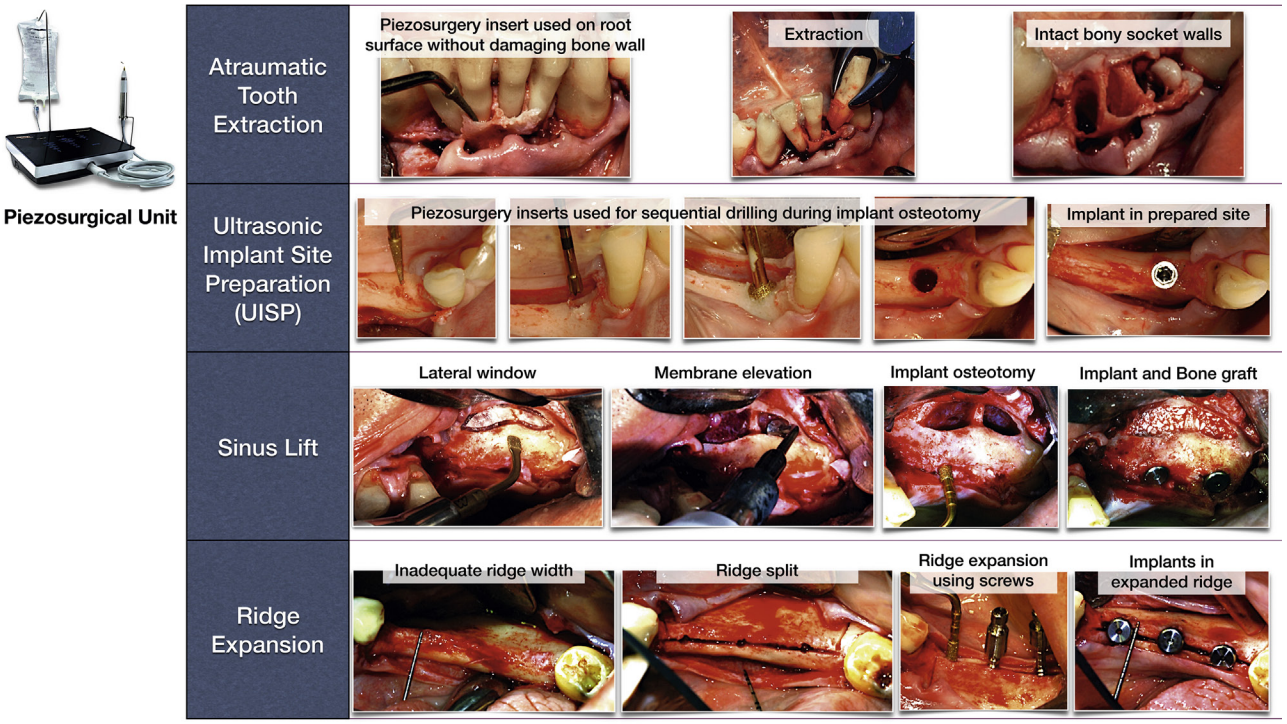
Digitally assisted implant surgery, or real-time micropositioning implant surgery (RTMIS) (Fig. 46.3), uses simultaneous tracking and "guidance" of the implant instrumentation to follow the planned treatment accurately *during* surgery. Variations from the ideal position when using RTMIS can be restricted *at the time of drilling* by the software through inactivation of the drill (stop-and-go action), or by an audible or visual cue.

Clinical Advantages of RTMIS

- Improved precision
- Reduced postoperative complications
- Perspectives for improved prosthetic treatment.

Challenges With RTMIS

- Learning curve and cost
- Time spent for installation (but overall, surgery is shorter).

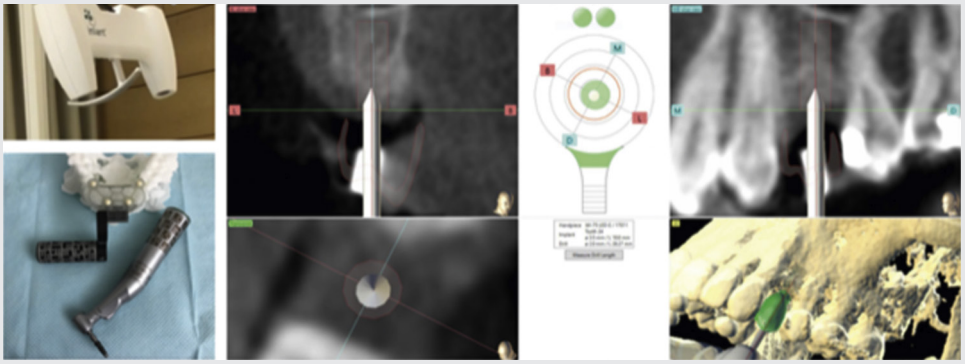


• **Fig. 46.2** Piezosurgery: Applications in Implant Dentistry. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

• **Fig. 46.3** Dental Implant Real-time Navigation System. This figure (Open Pilot System, Stereovision Haptitude) shows a device with infrared stereovision cameras and a monitor display showing the three-dimensional ultrasonic reconstruction of a mandible with the planned implant position in a coronal and a panoramic view. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

CASE-BASED LEARNING EXERCISE

Scenario: A senior dental student is preparing for his first implant placement procedure, at the edentulous site #12. In order to facilitate his learning for the surgical procedure, he is planning to use the dynamic navigation system (Figure) and a 3D-printed model, to "rehearse" the implant placement prior to the actual procedure.



Questions

1. Which component is unique to dynamic navigation systems?
   - **a.** Handpiece
   - **b.** Surgical stent
   - **c.** Fiducial marker
   - **d.** Dental light
2. Identify an advantage of using dynamic navigation versus the classical surgical stent/guide for placing an implant.
   - **a.** Reduced cost
   - **b.** Decreased 3D accuracy
   - **c.** Decreased presurgical planning
   - **d.** Increased irrigation to the surgical site
3. Ultrasonic cutting (piezosurgery) is indicated in all of the following surgical procedures, except one. Which is the exception?
   - **a.** bony window preparation for maxillary sinus elevation
   - **b.** tooth extraction
   - **c.** gingivectomy
   - **d.** ridge expansion
4. All of the following are characteristics of ultrasonic cutting (piezosurgery), EXCEPT one. Which is the exception?
   - **a.** Microprecision
   - **b.** Delayed healing
   - **c.** Increased visibility
   - **d.** Selective cutting

This chapter was developed from Chapters 67, 82, 83, and 84 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

Solutions

1. **Answer: c**

   Explanation: The dynamic navigation system is a technology designed to guide the placement of dental implants in real time by a computer, based on information generated from the patient's computed tomography scan. A unique component in dynamic surgical guidance system is the fiducial marker, attached to the patient during surgery.
2. **Answer: d**

   Explanation: Although the static surgical guide is a great tool that can enhance the accuracy of implant positioning during surgery, there are some limitations that need to be considered. Access for proper irrigation at the osteotomy site is sometimes a limitation with static guides. Dynamic navigation allows for increased irrigation at the surgical site.
3. **Answer: c**

   Explanation: Gingivectomy is a soft tissue resective procedure for which piezosurgery is not used. Rest are clinical indications of piezosurgery.
4. **Answer: b**

   Explanation: All of the listed features are characteristic of ultrasonic cutting, except for delayed healing. In fact, improved tissue healing is seen due to stimulation of growth factors (e.g., bone morphogenetic protein) following the application of piezosurgery.

47

Complications in Dental Implantology

Relevant Terminology

Terminology	Explanation
early implant failure	Implant loss or failure that occurs prior to restoration of the implant
failed versus failing implant	Failing implants present with bone loss (as in peri-implantitis) but are not mobile; a failed implant is either lost or mobile
hyperesthesia	A neuropathy (injury to nerve) leading to excessive sensory response.
hypoesthesia	A neuropathy (injury to nerve) leading to impaired/diminished sensory response (e.g. partial loss of sensation)
implant success	Parameter that is defined by specific criteria evaluating the condition and function of the implant. In general, implant success may be associated with any implant-retained restoration in which (1) the original treatment plan is performed as intended without complications, (2) all implants that were placed remain stable and functioning without problems, (3) the peri-implant hard and soft tissues are healthy, and (4) the patient and treating clinicians are pleased with the results.
implant survival	Parameter that assesses the presence of implants (restored or nonrestored) that have osseointegrated. It does not consider the condition or function of the implant.
late implant failure	Implant loss or failure that occurs during or after restoration of the implant
peri-implant health	Characterized by lack of erythema, swelling, or bleeding on probing (inflammation) of the periimplant soft tissue. Probing depths are usually ≤5 mm with no bone loss beyond initial healing (up to 2 mm is acceptable).
peri-implant mucositis	A reversible inflammatory process confined to the mucosa surrounding the neck of the implant(s), with no loss of supporting bone beyond initial bone remodeling
peri-implantitis	An inflammatory process affecting the bone surrounding the dental implant(s) and leading to progressive loss of supporting bone beyond initial healing. Probing depths are usually ≥6 mm.
Re-osseointegration	Refers mainly to new bone formation and new bony attachment to implant surfaces previously denuded of bone due to peri-implantitis.

Fast Facts

Examples of biologic complications	• Peri-implant mucositis • Peri-implantitis
Examples of prosthetic complications	• Abutment and prosthetic screw loosening • Fracture of implant or prosthesis
Examples of surgical complications	• Malposed implants • Soft tissue and bone dehiscence • Impingement or damage to anatomic structures
Esthetic complications	Complications associated with not meeting the patient's esthetic expectations. Usually seen in patients with high treatment expectations and demands.

Continued

Fast Facts—cont'd

Management of malposed implants	They can be corrected to an extent using custom angled abutments, but extremely malposed implants are nonrestorable; they must be removed and replaced with an implant in a restorably favorable position.
Prevalence of peri-implant mucositis and peri-implantitis	Based on a recent systematic review of 47 studies, the implant level prevalence of peri-implant mucositis and peri-implantitis were close to 30% and 10%, respectively.[1]
Implant fracture	A rare complication that can be prevented by managing heavy occlusal forces with the use of occlusal guards, and by avoiding cantilevered implant restorations. Repeated screw loosening and bone loss often precede implant fracture.
Complications following immediate implant placement	Gingival recession, poor implant positioning, inadequate postoperative band of keratinized mucosa, and unesthetic outcomes
Complications following ridge augmentation	Bleeding, infection, nerve dysfunction, loss of bone grafts, sinusitis (after sinus augmentation procedures), and wound dehiscence
Complications during lateral window sinus augmentation	Schneiderian membrane perforation, bleeding, injury to posterior superior alveolar vessel, and buccal flap perforation
Complications during crestal approach sinus augmentation	Schneiderian membrane tears, lack of adequate primary stability, malleting-related vertigo, and headache

Core Knowledge

Introduction

Implants offer a highly predictable treatment option for the replacement of single and multiple missing teeth; however, surgical, biologic, prosthetic, and esthetic complications can occur (Fig. 47.1 gives some clinical examples). The reader is referred to Chapter 85 of *Newman and Carranza's Clinical Periodontology* (13th ed.) for a detailed discussion of the management of various implant complications.

CLINICAL CORRELATE

When is an implant considered clinically successful?

An implant is considered clinically successful when there is:[2]

- absence of persistent pain, foreign body sensation, and/or altered sensation
- absence of mobility
- absence of recurrent peri-implant infection with suppuration
- absence of a sustained radiolucency around the implant

In the recently concluded American Academy of Periodontology/European Federation of Periodontology Joint Workshop on Periodontal Disease Classification, the following parameters are considered to indicate peri-implant health:[3]

1. Absence of visual signs of peri-implant mucosal inflammation (erythema, edema)
2. Lack of profuse bleeding on probing
3. Probing depths remaining stable over time
4. Absence of additional bone loss beyond initial healing (should not exceed 2 mm after implant loading)

CLINICAL CORRELATE

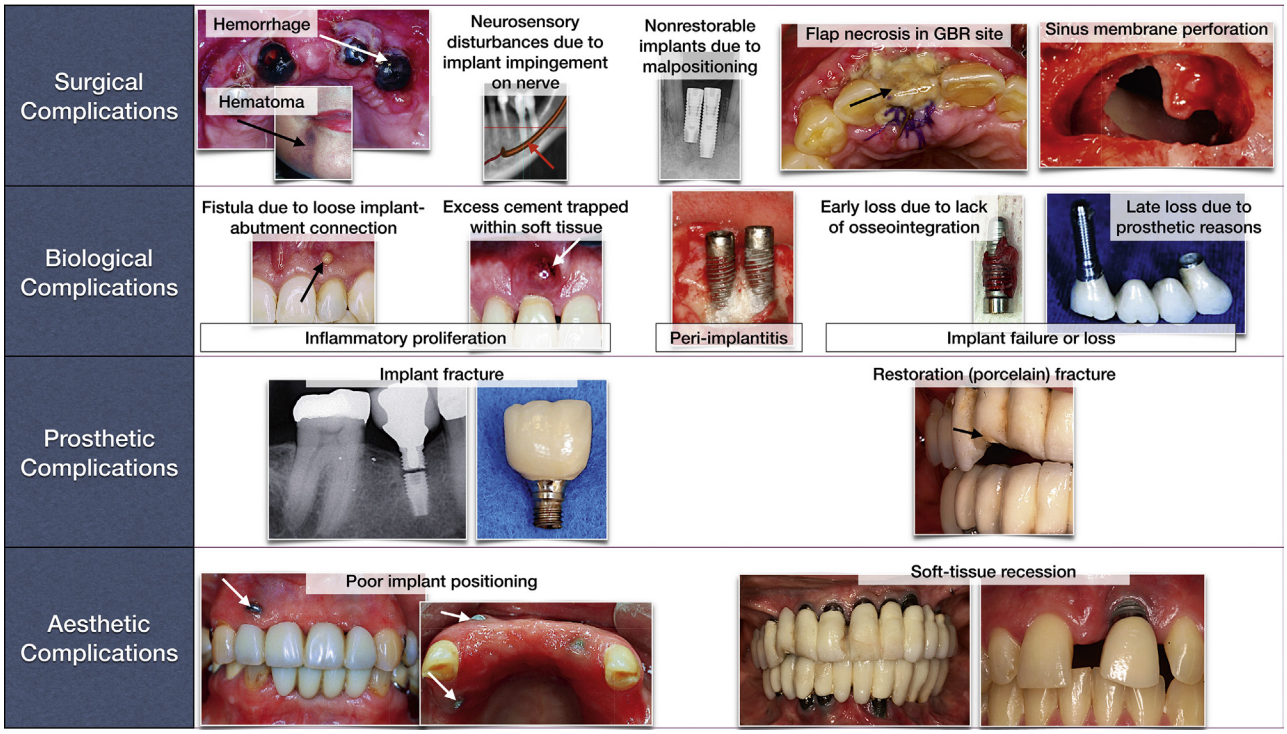
With regenerative therapy, is re-osseointegration of an ailing implant possible?

Implant surfaces that have lost supporting bone due to bacterial contamination may be treated by open flap debridement where implant surface decontamination and regenerative procedures may be combined to potentially result in *re-osseointegration.*[4]

However, just as in the case of periodontal regeneration, in human clinical cases the postoperative radiographs only indicate *bone fill* after regenerative procedures, and not necessarily re-osseointegration (which can only be confirmed by histology). Hence, while re-osseointegration is possible, its confirmation (especially in humans) is extremely difficult.

Peri-implantitis

Peri-implant diseases are inflammatory lesions strongly associated with poor oral hygiene, and their prevention is similar to the practices for preventing periodontal disease. Fig. 47.2 shows the differences between healthy peri-implant tissues, peri-implant mucositis, and peri-implantitis.

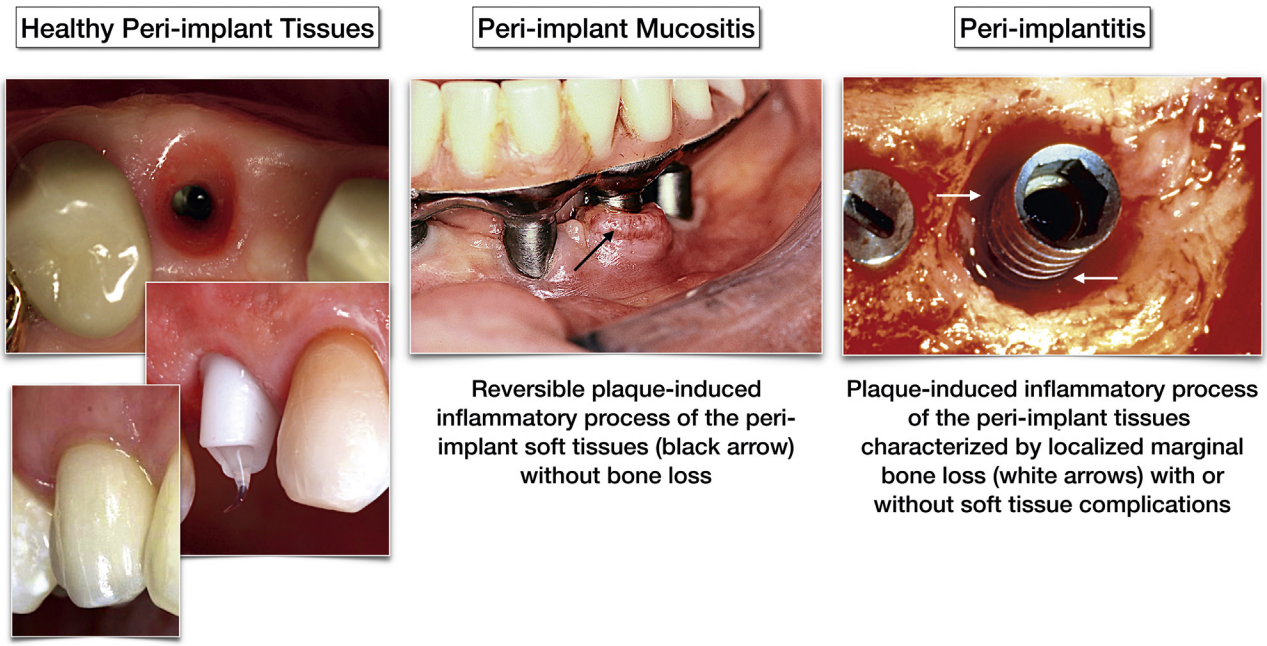


• **Fig. 47.1** Complications in Implant Dentistry. Several factors can influence implant treatment outcome. Apart from patient-related factors (poor oral hygiene, systemic conditions, medications, poor bone quality, detrimental habits like smoking, etc), the risk of implant failure or complications can also be increased by procedure-related factors, including:

- Clinician inexperience
- Bone overheating and excessive surgical trauma
- Inability to achieve optimal primary implant stability
- Insufficient sterilization
- Immediate loading protocol performed without adequate knowledge or training
- Insufficient number or inadequate size of supporting implants
- Non-optimal surface properties and implant design
- Poor prosthetic design
- Occlusal overload

GBR, guided bone regeneration

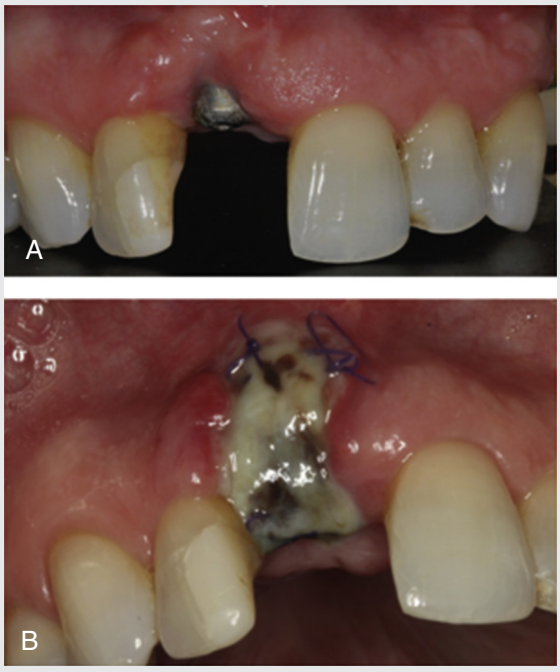
From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.



• **Fig. 47.2** Peri-implant Health, Peri-implant Mucositis, and Peri-implantitis. Peri-implant diseases present as inflammatory processes involving the soft tissues and/or the bone surrounding a functioning dental implant. While the lesion of peri-implant mucositis is restricted to the soft tissues, peri-implantitis also affects the supporting bone. Radiographic evaluation, bleeding on probing (BOP), and probing depth (PD) are used together for early detection of peri-implant pathologies. Peri-implant mucositis is usually treated by nonsurgical therapy involving mechanical debridement, polishing, and antiseptics (mouth wash, subgingival irrigation, or antibiotics). Peri-implantitis may be treated using nonsurgical therapy and locally delivered antibiotics, or combinations of surgical therapy, systemic antibiotics, and regenerative procedures.

CASE-BASED LEARNING EXERCISE

Scenario: A 41-year-old male presented to the clinic for restoration of the edentulous site #8. The implant was placed 6 months earlier; since then the patient had moved to a different city. He presented to the clinic for the final implant-supported prosthesis (Fig. A). Given the soft tissue deficiency noted at site #8 in the esthetic zone with a high smile line, subepithelial connective tissue graft (SCTG) was performed to correct the mucogingival deformity. At 2 weeks following SCTG, the patient presented with necrosis of the graft (Fig. B).



Questions

1. All of the following statements about implant treatment–related complications are true, except one. Which one is the exception?
- **a.** A complication is a secondary condition that develops during or after implant surgery
- **b.** Implant complications can be life-threatening
- **c.** Occurrence of a complication indicates that substandard dental care was provided
- **d.** Occurrence of a complication does not necessarily mean clinical failure

2. In the current case, the dentist recommended the SCTG in order to improve the _____ profile:
- **a.** Convergence
- **b.** Angular
- **c.** Emergence
- **d.** Divergence

3. Which of the following are identified risk factors that may contribute to peri-implant disease?
- **a.** Poor oral hygiene
- **b.** Cigarette smoking
- **c.** History of periodontitis
- **d.** All of the above

4. The most common complication with implant overdentures is _____.
- **a.** Prosthesis fracture
- **b.** Clip/attachment loosening
- **c.** Implant fracture
- **d.** Abutment screw loosening

This chapter was developed from Chapter 85 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapter. The reader is encouraged to read the reference chapter for a complete understanding of this important topic.

Solutions

1. **Answer: c**

Explanation: All of the statements are true, except (c). The occurrence of a complication does not indicate that substandard dental care was provided. Multiple etiologic factors that cannot be controlled can lead to complications. It is critical for the dentist to be aware of various intraoperative and postoperative complications, so that they can be appropriately diagnosed and managed.

2. Answer: c

Explanation: The cross-sectional shape and dimensions of an implant are not the same as those of a natural tooth. In order to achieve an esthetic outcome, soft tissue contouring is very important. This is where the emergence profile of the implant-supported restoration becomes critical.

3. Answer: d

Explanation: There is accumulating evidence that poor oral hygiene, cigarette smoking, and history of periodontitis are all risk factors for the development of periimplant disease.

4. Answer: b

Explanation: Clip/attachment loosening is the most common complication with implant overdentures.

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3. Renvert, S., Persson, G. R., Pirih, F. Q., & Camargo, P. M. (2018). Peri-implant health, peri-implant mucositis, and periimplantitis: Case definitions and diagnostic considerations. *Journal of Periodontology*, *89*(Suppl. 1), S304–S312.
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48

Results of Implant Treatment and Future Supportive Implant Care

Relevant Terminology

Terminology	Explanation
implant outcomes:	Refers to various ways in which individual clinical investigators / researchers measure, interpret, and report implant treatment results. e.g. implant success, implant survival, patient reported quality of life post-treatment, etc.
implantoplasty:	removal of exposed implant threads using rotary instruments to produce a smooth non-contaminated surface that will discourage plaque accumulation in future.

Fast Facts

Factors affecting peri-implant probing	• Size of the probe • Force and direction of insertion of the probe • Health and resistance of peri-implant tissues • Level of bone support • Implant features and prosthetic design
Implant probing recommendations	• Studies indicate that traditional metal periodontal probes can be used to probe around implants • Implant probing at the time of prosthesis delivery will provide baseline information, which should be followed by documentation of probing measures on an annual basis • It is usual for probing depths to be deeper than those around natural teeth, depending on the depth of implant placement • The main objective of this process is to monitor changes in probing pocket depth over time
Implant percussion	A solid, resonating sound typically indicates osseointegration; a dull sound indicates fibrous encapsulation
Treatment of peri-implant mucositis	• Treated nonsurgically with the goal of removing supramucosal and submucosal biofilm and calculus using hand and ultrasonic instruments • Antimicrobials can be used adjunctively
Treatment of peri-implantitis	• Treated by nonsurgical and surgical means • Nonsurgical treatment is the same as for treating peri-implant mucositis • Surgical procedures involve flap elevation followed by decontamination of the implant surface and grafting of the defect (when applicable) • Currently, there is lack of evidence to recommend the most effective approach to treat peri-implantitis
Instruments and implant surface	Metal instruments are known to cause scratches on the implant surface, and therefore specific instrument tips are recommended to debride a titanium surface (e.g., plastic, Teflon-coated, carbon, and gold-coated curettes).

Fast Facts—cont'd

Factors affecting implant outcomes	• Anatomic location—there are differences in bone density between different areas of the alveolar bone; implant outcomes tend to be better in areas with high-density bone. For this reason, implants placed in maxillary posterior sextants tend to have more complications than those placed in the mandibular anterior sextant. • Implant design characteristics—dimensions, geometries, and surface characteristics • Placement and loading protocols • Presence of risk factors (smoking, diabetes, and periodontitis)

Core Knowledge

Introduction

The desired outcome of implant therapy includes both the prosthetic and surgical success of implant-supported restorations (see Chapter 47 to learn the criteria for peri-implant health). Variations from the desired outcome of implant therapy include:[1]

- Implant loss or fracture
- Mobile implant or prosthesis
- Damaged prosthesis
- Persistent pain
- Inability to chew using the implant prosthesis (functional loss)
- Progressive bone loss and persistent peri-implant radiolucency
- Persistent inflammation/infection in peri-implant tissues
- Inability to restore the osseointegrated implant

CLINICAL CORRELATE

What is the rationale behind performing supportive implant treatment?

- Although dental implants are not vulnerable to caries, they are susceptible to mechanical complications and biofilm-induced inflammatory peri-implant tissue changes (peri-implant mucositis and peri-implantitis) over time.
- The relationship between peri-implant mucositis and peri-implantitis is similar to that between gingivitis and periodontitis. Although peri-implant mucositis does not necessarily progress to peri-implantitis, it is likely to be the precursor.
- While peri-implant mucositis can be treated effectively with nonsurgical mechanical therapies, the results of surgical treatment for peri-implantitis are not predictable. Prevention, early detection, and early treatment of peri-implant diseases, with the help of periodic supportive implant treatment, are therefore crucial to the long-term success of dental implant therapy.

Success in implant therapy is judged by the maintenance of a stable, functional, and esthetically acceptable replacement for tooth and supporting structures that have been lost. Table 48.1 lists components of supportive implant care that must be performed at regular intervals to monitor peri-implant health and ensure a successful outcome.

CLINICAL CORRELATE

How should an implant prosthesis be evaluated during supportive implant treatment?

At the time of prosthesis delivery:

- Radiographs perpendicular to the implant should be obtained for baseline documentation and to verify complete seating of the restorations, especially at the implant-abutment interface.
- Immediately after delivery, cement-retained implant restorations should be thoroughly checked for residual excess cement within and adjacent to peri-implant sulcus; excess cement must be removed.

During follow-up visits:

- Implant restorations should be carefully examined for fractures and loose screws.
- Loose abutment screws must be evaluated, possibly replaced, and properly torqued down.
- Worn-out retentive components in removable prostheses (i.e., Hader clips and locator attachment inserts) must be replaced if necessary.
- Occlusal wear of teeth and fit of tissue-borne surfaces of an implant prosthesis should be assessed, and corrected if required.
- Occlusal guards must be recommended to protect implants and restorations in cases of oral parafunctions.

Implant Maintenance

Patient Plaque Control Around Implants

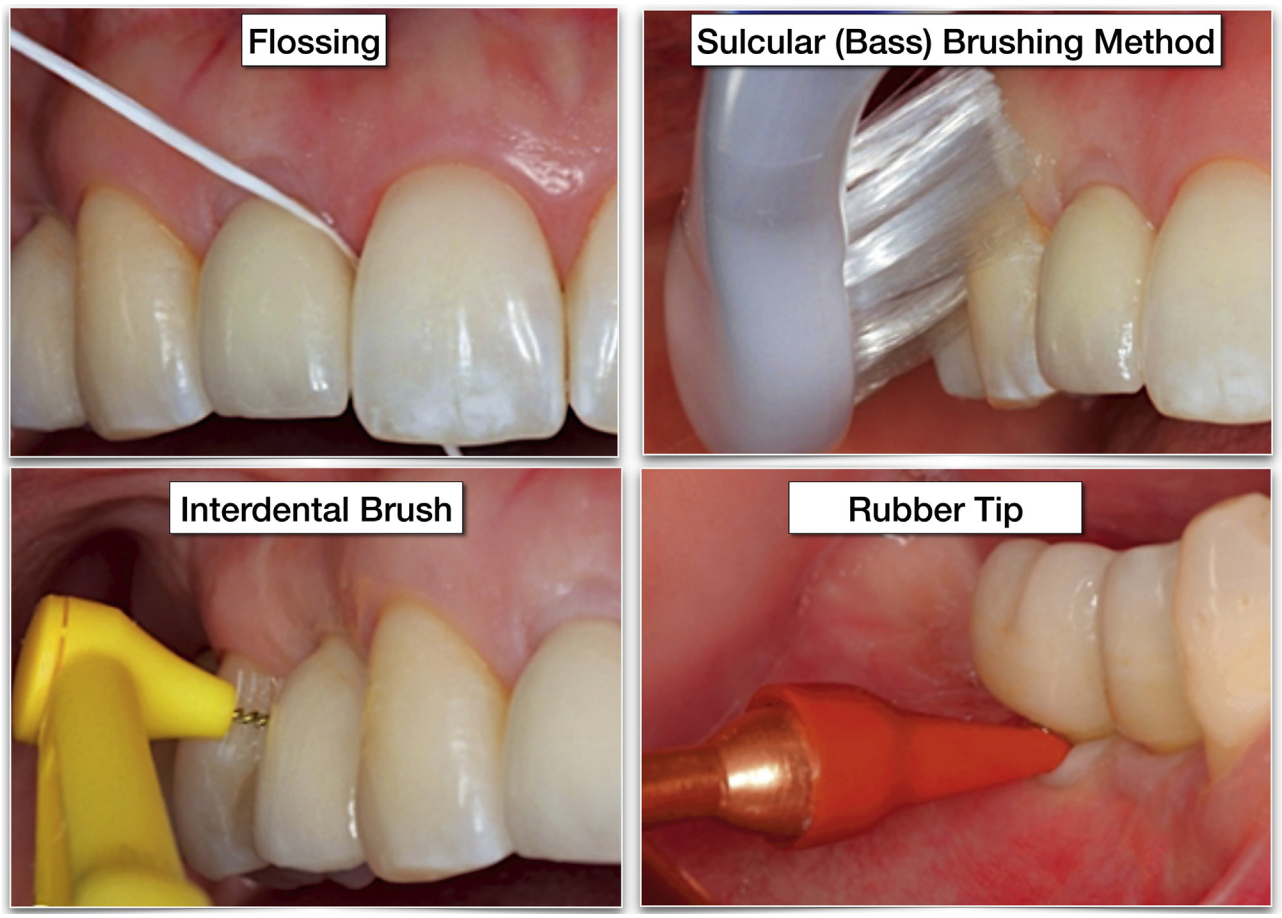
Although plaque control techniques for dental implant-supported restorations mirror traditional oral hygiene procedures on natural teeth, certain modifications are required in accordance with the prosthetic design. These are instructed specifically to the patient by a knowledgeable clinician (Fig. 48.1).

- Because implants have deeper crevices than natural teeth, a sulcular technique of brushing (e.g., the Bass method) may be more effective in cleaning an implant below the mucosa than other techniques (see Chapter 27). An intrasulcular method of brushing should be recommended

TABLE 48.1 Examination of Implants and Prostheses: Possible Findings During Supportive Implant Care[2,3]

		Peri-implant Health	Peri-implant Mucositis	Peri-implantitis
Presence of plaque and calculus		+ / -	+	+
Implant prosthesis	Stability	Stable	May be mobile	May be mobile
	Integrity	Intact	May be damaged	May be damaged
	Cement in sulcus	No	May be present	May be present
Peri-implant soft tissues	Clinical appearance	Pink, firm, well-adapted soft tissue	• Erythema • Edema • Inadequate keratinized soft tissue may be present • Inflammatory proliferation may be present • Fistula or sinus tract may be present	• Erythema • Edema • Inadequate keratinized soft tissue may be present • Inflammatory proliferation may be present • Fistula or sinus tract may be present
	BOP and/or suppurative exudate	-	+	+
	Probing depth (PD)	≤5 mm	Increasing probing pocket depths as compared with measurements obtained at placement of the suprastructure	• Increasing probing pocket depths as compared with measurements obtained at placement of the suprastructure • In the absence of initial PD values, probing depths ≥6 mm in conjunction with profuse bleeding represents peri-implantitis
	Pain on probing	-	+/-	+/-
Peri-implant hard tissues	On percussion of implant	Solid, resonating sound elicited	Solid, resonating sound elicited	Possible dull sound and pain upon percussion
	Implant stability	Stable	Stable	May be mobile
Radiographic evaluation	Implant-abutment junction	No gap	Gap may be present	Gap may be present
	Bone-implant interface	No peri-implant radiolucency	No peri-implant radiolucency	Peri-implant radiolucency of varying degrees
	Apicocoronal distance from implant platform to peri-implant crestal bone after the first year of functional loading	Should not be ≥2 mm (this value accounts for the remodeling process of alveolar bone crest during the first year after installation)	Should not be ≥2 mm (this value accounts for the remodeling process of alveolar bone crest during the first year after installation)	• Presence of bone loss beyond the expected crestal bone level changes from initial remodeling, in conjunction with BOP after the implant has been placed in function • (In the absence of initial measurements) Radiographic evidence of bone level ≥3 mm apical distance from implant platform

+, present; -, absent; +/-, may or may not be present; BOP, bleeding on probing; PD, probing depth.



• **Fig. 48.1** Plaque Control Aids in Implant Maintenance. (From Newman, M.G., Takei, H.H., Klokkevold, P.R., et al. (2019). *Newman and Carranza's Clinical Periodontology* (13th ed.). Philadelphia: Elsevier.)

with caution in individuals with a narrow width of peri-implant keratinized mucosa.

- A cotton tip or soft toothbrush can be used to gently remove biofilm from healing abutments or provisional restorations during the early postoperative phase of healing. (Caution: before implant osseointegration, the use of powered toothbrushes and forceful subgingival irrigation during home care should be limited.)
- After implant osseointegration has been achieved and verified, brushing with dentifrice and use of dental hygiene aids (e.g., dental floss, rubber tips, and interdental brushes) can be employed.

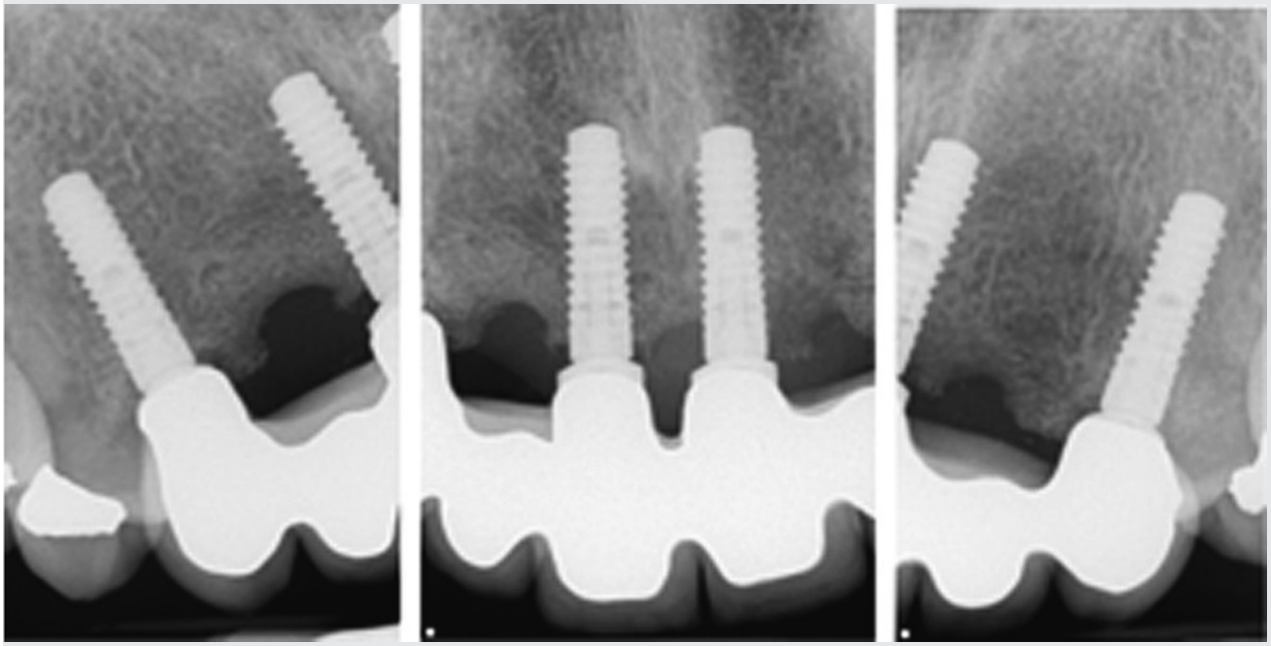
Professional Plaque Control Around Implants

Periodontal maintenance is better performed every 3–4 months if the reason for the implant placement is to replace teeth lost due to past caries or periodontitis.

- Probing with a conventional metallic probe is not a cause for much concern, because the surface alteration caused by the tip of a metallic probe against an abutment is minimal; however, plastic probes are available and can be equally effective in measuring probing depth.
- Care should be taken to minimize damage to transmucosal implant surfaces (e.g., polished titanium implant collars) during instrumentation for biofilm and calculus removal. Rubber cups and polishing paste can be used to remove biofilm from machined and polished surfaces. Materials that are friendly to the abutment surface include Teflon, titanium, gold, or plastic tips.
- Gold alloy or ceramic surfaces can be debrided with most scalers and curettes (e.g., plastic, gold-coated, stainless steel) without damaging the surface.
- Use magnetostrictive and piezoelectric ultrasonic instruments with metal tips (e.g., Cavitron) with caution because of irregularities that can easily be created in the surface. Special ultrasonic tips are now available to clean implants efficiently without damaging the implant surface.

CASE-BASED LEARNING EXERCISE

Scenario: An 18-year-old female presented with the chief complaint: "I want my smile back. I was in an accident and lost my front teeth." An interdisciplinary team collaborated to restore her maxillary anterior dentition with implant-supported prosthesis. The patient had been visiting her dental office for periodic recalls every 6 months, and her remaining teeth were periodontally healthy. After 2 years, she came back to the surgeon's office for a 2-year postoperative check. Periapical radiographs were taken.



Questions

1. Which of the following is the least likely to be encountered 2 years after the treatment completion?
   - **a.** Speech problems
   - **b.** Difficulty cleaning
   - **c.** Fractured screw
   - **d.** Paresthesia
2. The final prosthesis was screw-retained. What is the main advantage of this method over cement-retained prosthesis?
   - **a.** Cost
   - **b.** Esthetics
   - **c.** Retrievability
3. The patient was maintaining excellent plaque control. What are the most likely tools she used?
   - **a.** Flossing under bridges
   - **b.** Water jet irrigation device
   - **c.** Toothbrush
   - **d.** All of the above
4. Considering the etiology of the initial tooth loss, how often should the patient come in for periodontal recall visits?
   - **a.** Monthly
   - **b.** Quarterly
   - **c.** Biannually
   - **d.** Annually

Solutions

1. Answer: d

Explanation: Paresthesia is the least likely complication to occur 2 years after the treatment was completed.

2. Answer: c

Explanation: It is easier to retrieve a prosthesis when it is screw-retained than when it is cement-retained. Esthetics and cost are comparable for both types of restorations.

3. Answer: d

Explanation: The patient was very compliant with the oral hygiene instructions. She used all the recommended tools to maintain peri-implant health.

4. Answer: c

Explanation: Considering the overall periodontal health and the young age of the patient, periodontal recall visits every 6 months are recommended. Periodontal maintenance with a 3- to 4-month interval would be considered if the tooth loss was the result of rampant caries or periodontitis.

This chapter was developed from Chapters 86 and 87 in *Newman and Carranza's Clinical Periodontology* (13th Edition), and is a summary of many of the important sections of the chapters. The reader is encouraged to read the reference chapters for a complete understanding of this important topic.

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