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NEWMAN AND CARRANZA'S

Essentials of Clinical Periodontology

AN INTEGRATED STUDY COMPANION

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Newman and Carranza's Essentials of Clinical Periodontology

An Integrated Study Companion

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NEWMAN AND CARRANZA'S ESSENTIALS OF CLINICAL PERIODONTOLOGY:
AN INTEGRATED STUDY COMPANION ISBN: 978-0-323-75456-9

Library of Congress Control Number: 2020949063

Content Strategist: Joslyn Dumas
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Content Development Manager: Andress B. Meghan
Publishing Services Manager: Shereen Jameel
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Printed in China

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

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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

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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

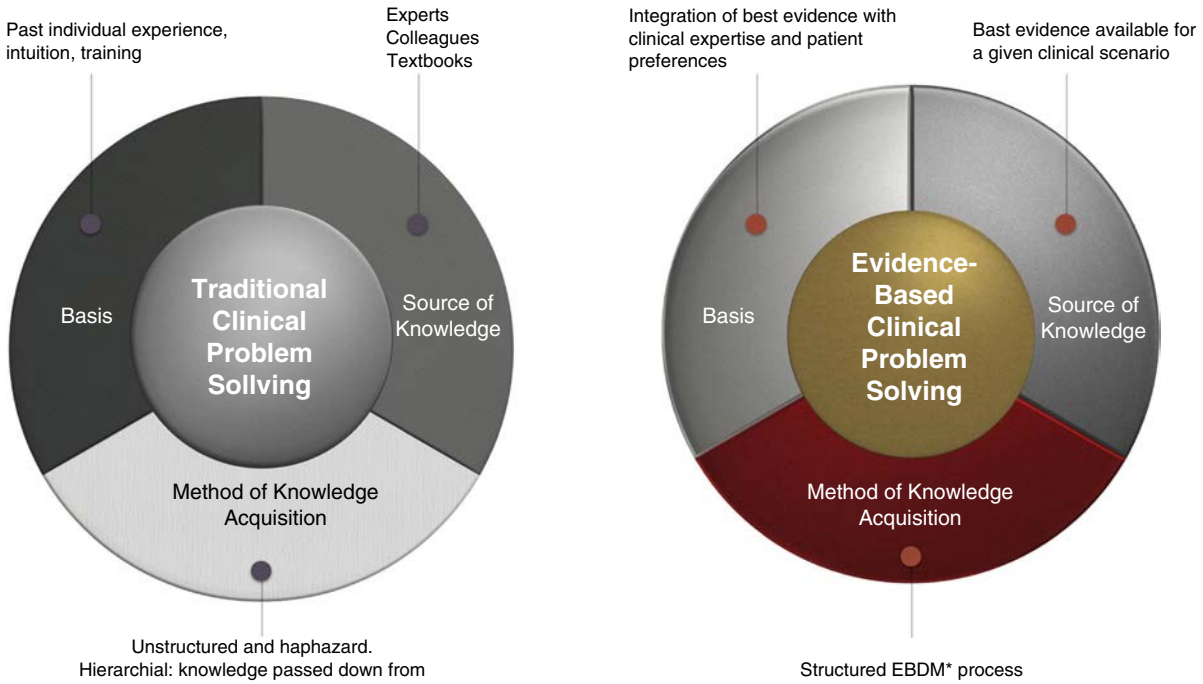
Principles of Evidence-Based Decision-Making

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.

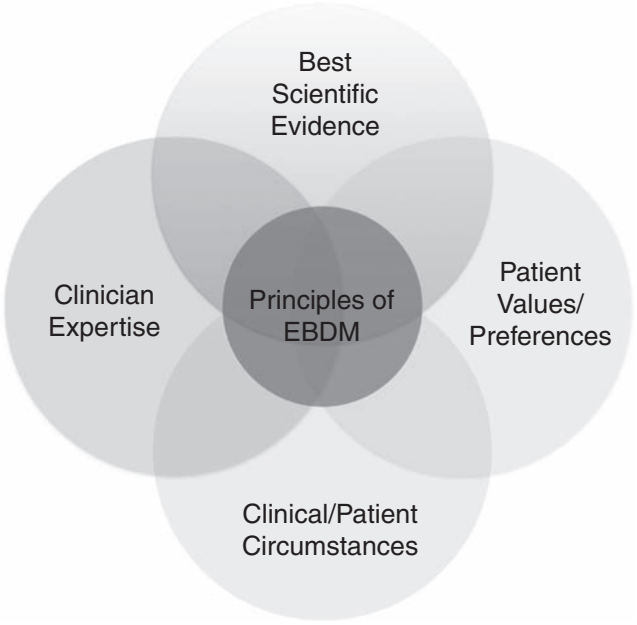
One must know the types of studies that constitute the highest levels of evidence in order to be able to apply filters for efficient searching and retrieval of best evidence (Fig. 1.3).

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):

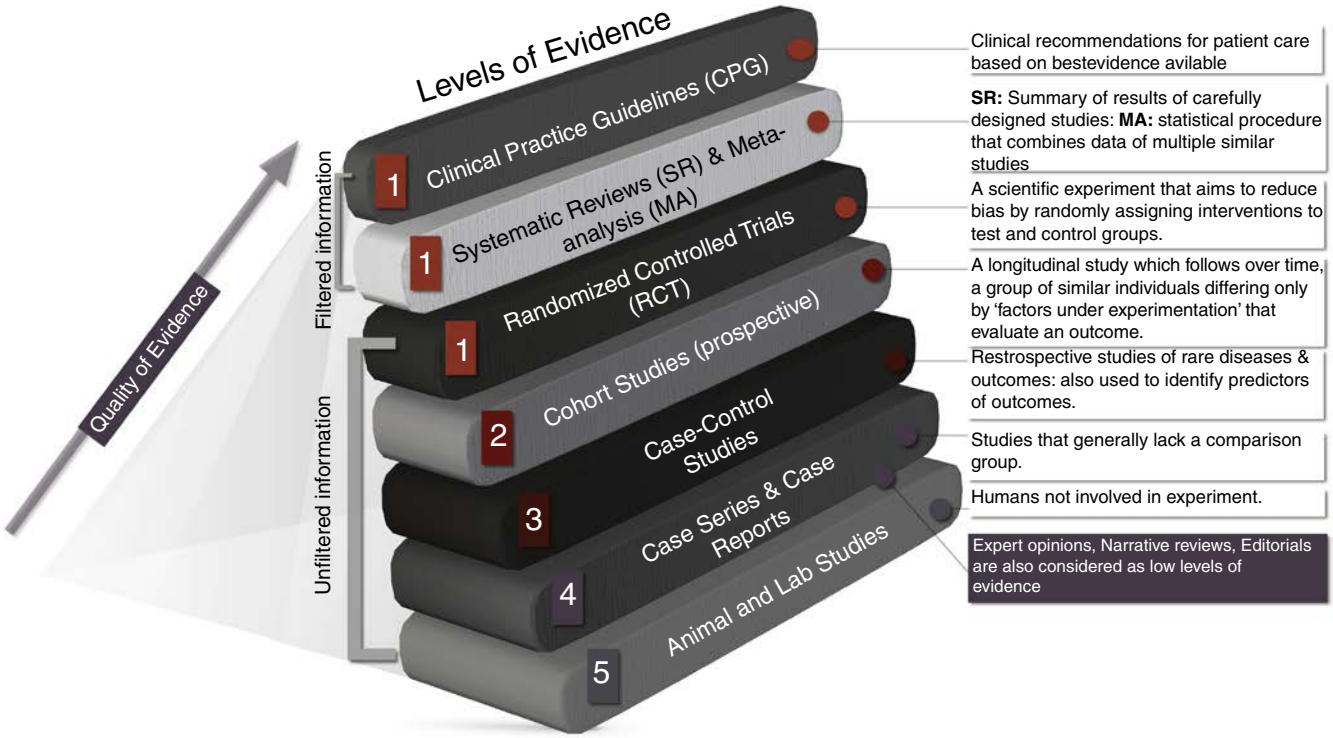
1. **Ask**: Asking the right question follows PICO format, which requires defining four components to a clinical problem (Problem/Population, Intervention, Comparison, and Outcome). This is important for:
   - Forcing the clinician to identify the single most important outcome the search should be focused 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

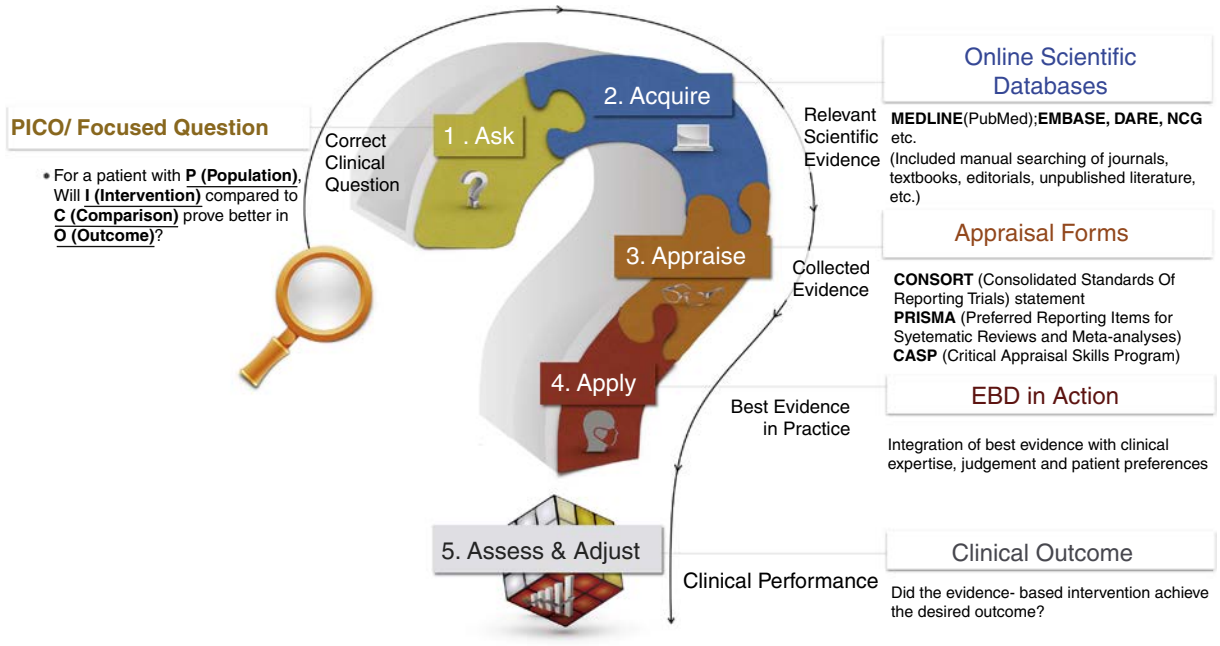
What are the advantages of a formal process of evidence-based decision-making?

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

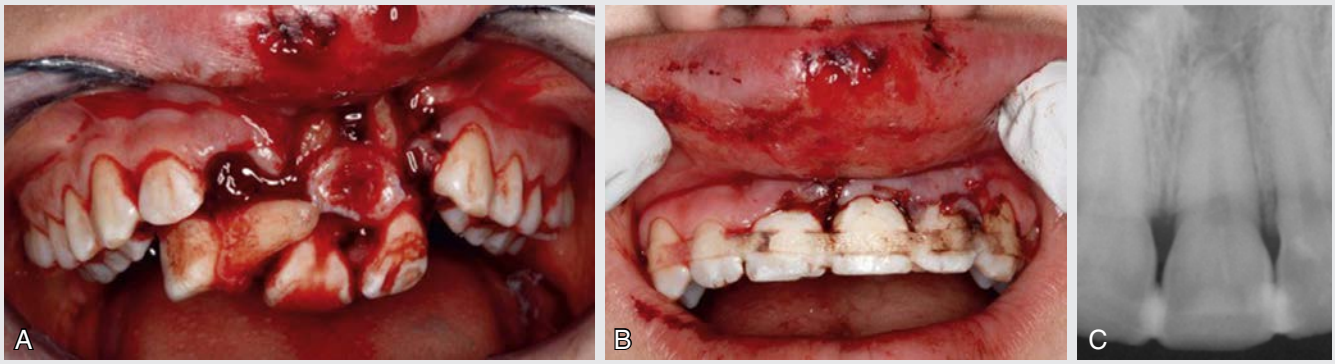
As EBDM integrates into the clinical problem-solving process and becomes standard practice, it becomes vital for clinicians to understand the importance of critical thinking, rigorous methodology in research, and what constitutes credible evidence for clinical use. The EBDM process takes time to learn 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.

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

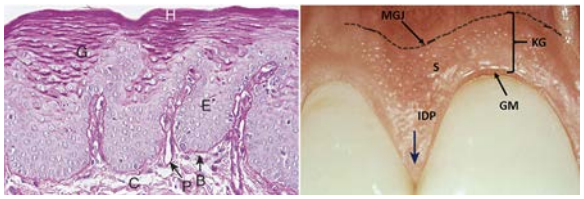
Gingiva

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.

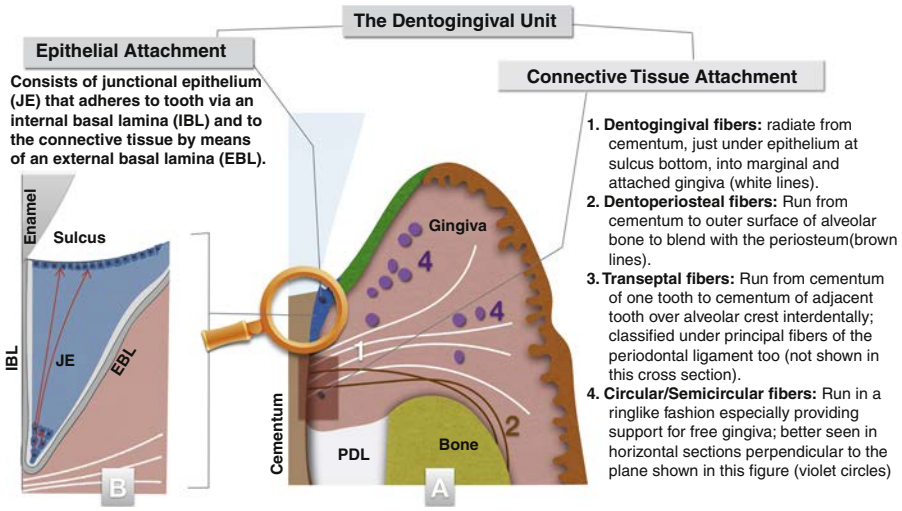
Functions of the Gingiva

- 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

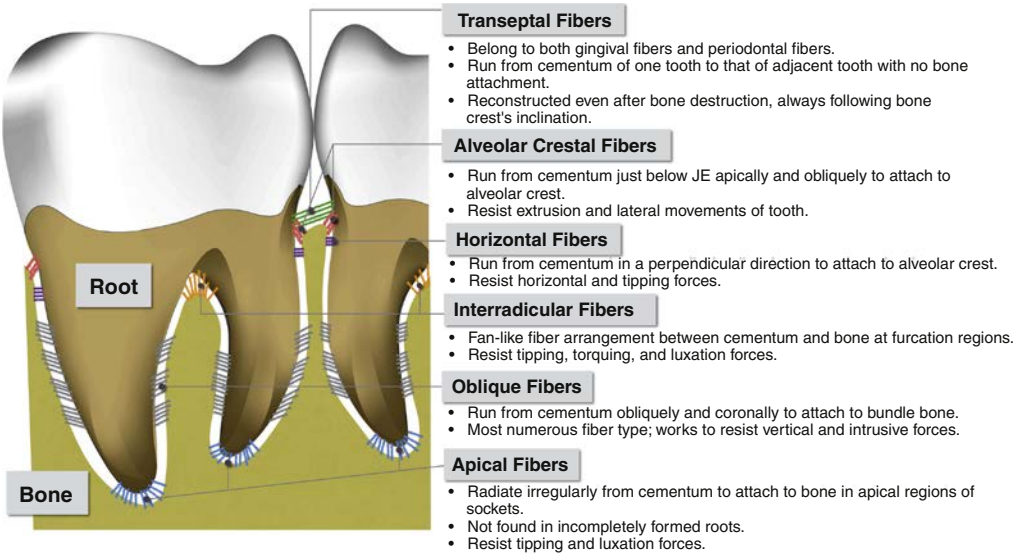
What is the difference between *active* eruption and *passive* eruption?

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

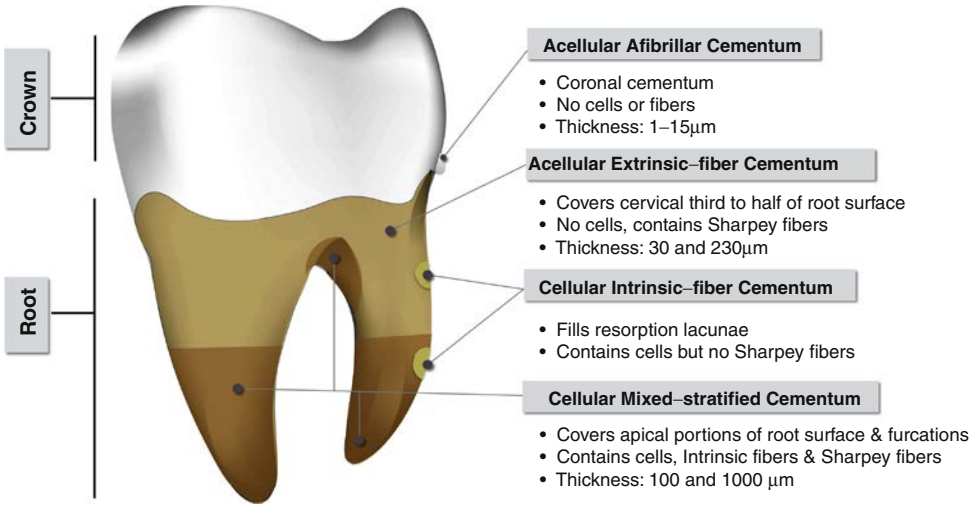
The PDL tissue is composed of:

- **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.

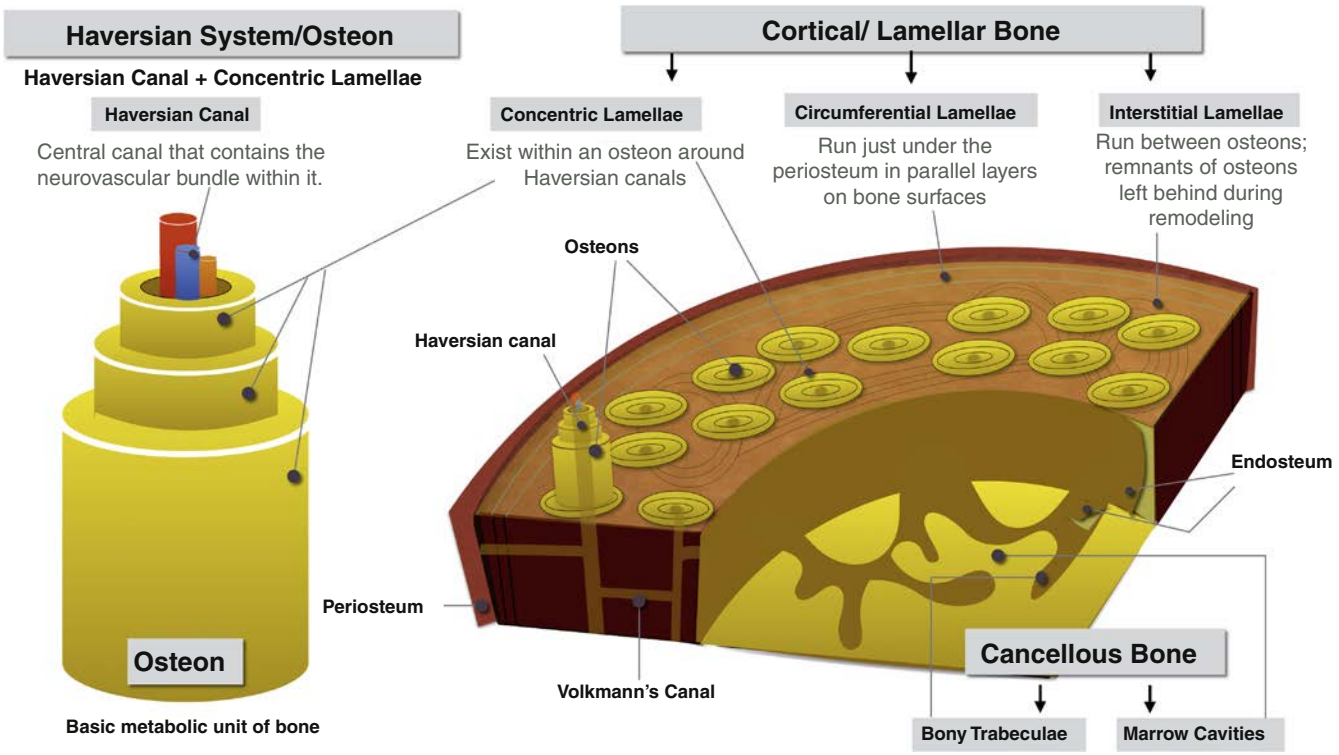
Functions of Cementum

- **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:

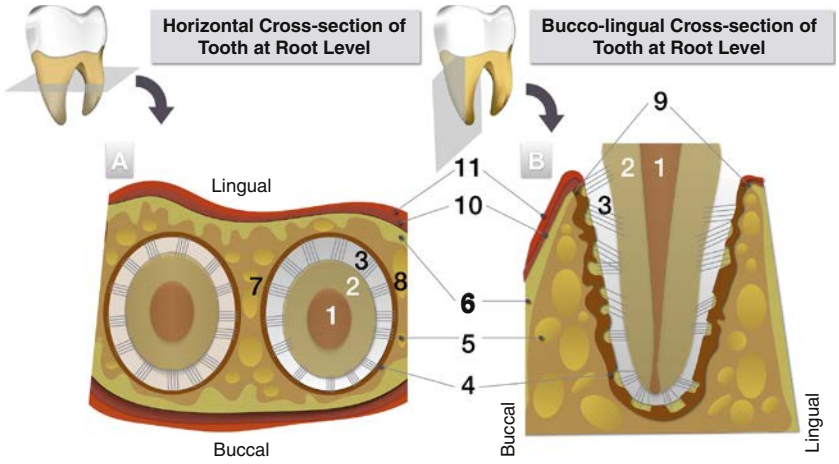
- Houses roots of teeth
- Anchors teeth roots to alveoli via Sharpey fibers
- Helps absorb and distribute occlusal forces generated during tooth contact
- Supplies blood vessels to the PDL
- Organizes eruption of primary and permanent teeth

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:

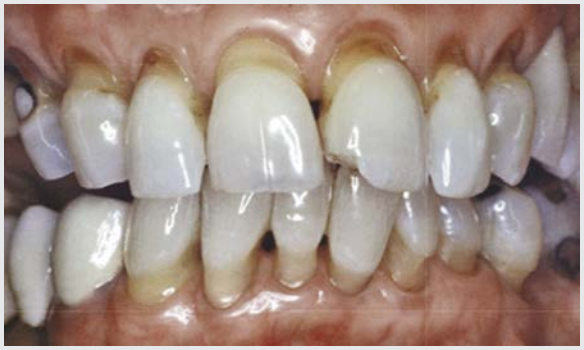
4. Alveolar bone proper—cortical bone plate that immediately lines the periodontal ligament space. Also known as:
   - *Bundle bone*—as it contains bundles of Sharpey fibers inserting into it
   - *Cribriform plate*—a histologic description, due to its porous nature that allows PDL fiber insertion and 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 Isoaded 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

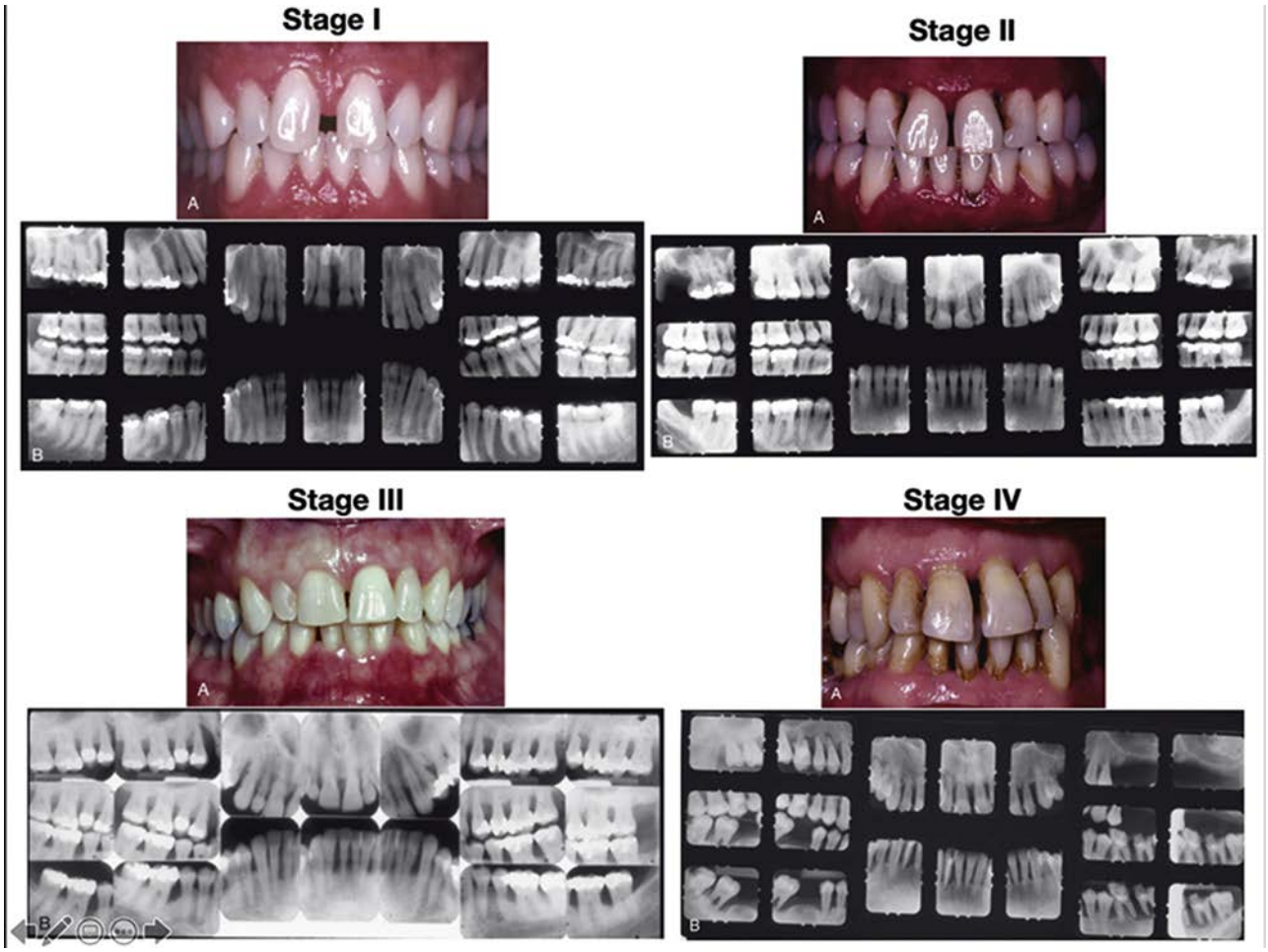
	Periodontitis	Stage I	Stage II	Stage III	Stage IV
Severity	Clinical attachment loss	1–2 mm	3–4 mm	≥5 mm	≥5 mm
	Radiographic bone loss	Coronal third of the root	Coronal third of the root	Middle or apical third of the root	Middle or apical third of the root
	Tooth loss due to periodontitis	No tooth loss	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.

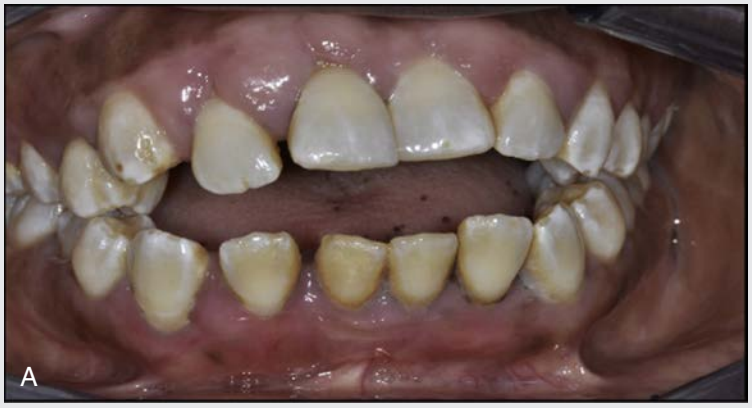
Clinical and radiographic images depicting the various stages of periodontitis are presented in Fig. 3.1.

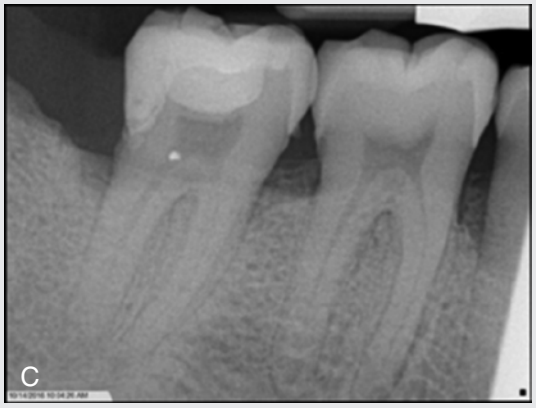
Conclusion

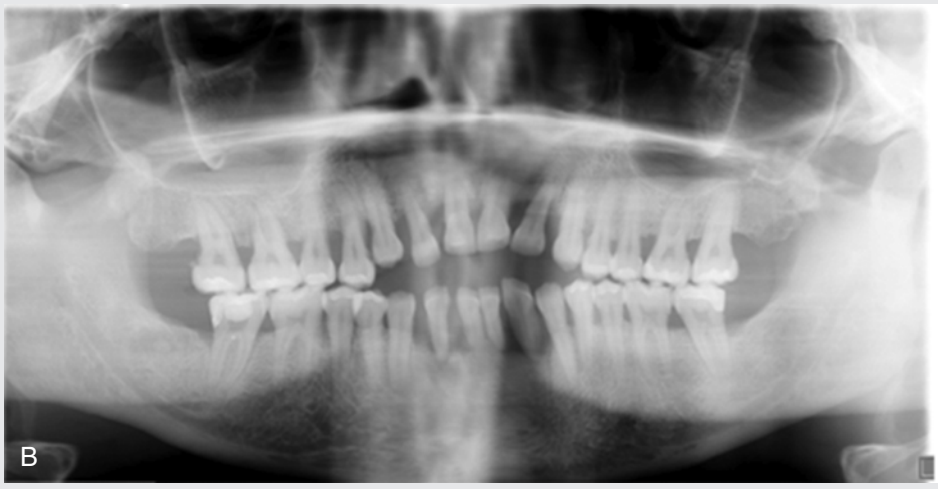
This chapter has provided an overview of the current classification system and the rationale behind the newer categorizations. The reader is referred to the textbook chapter (Chapter 5) as well as the official classification workshop proceedings for detailed reading of the individual entities described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

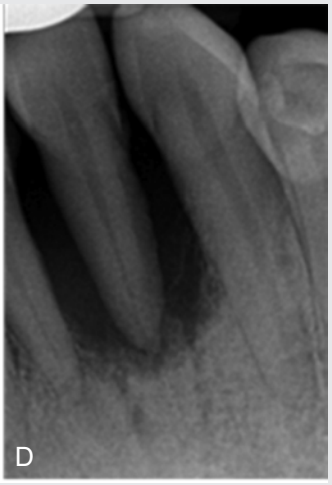
CASE-BASED LEARNING EXERCISE

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.

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