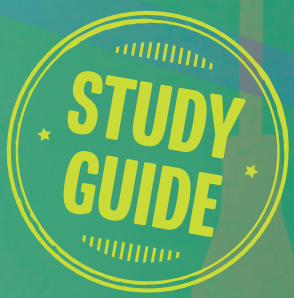
SECOND EDITION

PERIODONTAL REVIEW Q&A

Deborah A. Termeie, DDS



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Periodontal Review Q&A

Second Edition

Deborah A. Termeie, DDS
Clinical Instructor
Department of Periodontics
School of Dentistry
University of California, Los Angeles
Los Angeles, California

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About the Author

Deborah A. Termeie, DDS, is a clinical instructor in the Department of Periodontics at the University of California, Los Angeles. She is a diplomate of the American Board of Periodontology (ABP), and it was her experience preparing for the ABP qualifying exams that inspired her to write this book. Dr Termeie is also an editor and author of *Avoiding and Treating Dental Complications: Best Practices in Dentistry* (John Wiley & Sons, 2016) and has published on the topics of evidence-based dentistry and implantology. She is the recipient of several awards, including the Excellence in Implantology Research award from the California Society of Periodontics, and has been invited to participate in the American Academy of Periodontics Leadership, Engagement, Action, and Development (LEAD) Program. She maintains a private practice in Beverly Hills, California.

Contents

Preface *vi*

1 Evidence-Based Dentistry *1*
2 Periodontal Anatomy *6*
3 Furcations *17*
4 Epidemiology and Etiology *31*
5 Pharmacology *53*
6 Diagnosis *81*
7 Prognosis *113*
8 Occlusion *125*
9 Nonsurgical Therapy *139*
10 Surgical Therapy *163*
11 Mucogingival Therapy *186*
12 Regeneration *221*
13 Implants *252*
14 Inflammation *299*
15 Oral Medicine *311*
16 Oral Pathology *326*
17 Lasers *347*
18 Medical Emergencies *354*
19 Treatment Planning *362*

Appendix *408*
Index *417*

Preface

The first edition of *Periodontal Review Q&A* was very well received, and I am grateful for the opportunity to author this second edition. The new edition contains many new figures, tables, and treatment planning cases as well as a comprehensive review of new classifications. I wrote this book because despite a plethora of study materials and information, there is no comprehensive single source study guide to help students prepare for their examinations. *Periodontal Review Q&A* was specifically written to address this void.

The material in this book is presented in a question and answer format for ease of study. The classic literature is cited as well as more recent and practical literature on topics such as diagnosis, nonsurgical therapy, surgical therapy, regeneration, and implants. Literature evidence for opposing viewpoints is also presented throughout the book. Additionally, each chapter contains clear and relevant tables, illustrations, and pictures. This comprehensive and yet concise approach to periodontics is aimed at preparing the candidate for periodontal examinations and clinical practice.

Periodontal Review Q&A is a useful resource for residents, practicing periodontists preparing for board certification, dental students, and dental hygiene students seeking a broader appreciation and in-depth understanding of periodontics. Topics chosen are those emphasized in periodontal residency graduation examinations as well as the oral examination of the American Board of Periodontology. Readers are urged to study all literature preceding their examination, including literature that may be made available subsequent to this textbook's publication.

Acknowledgments

I would like to acknowledge my mentors, Drs Philip R. Melnick, Thomas N. Sims, Paulo M. Camargo, Thomas Han, Henry H. Takei, and Perry R. Klokkevold, for their advice and guidance. Thank you to all the reviewers, Drs Dennis P. Tarnow, Russell Christensen, Jack G. Caton, Michael P. Rethman, Mary E. Neill, and Sejal R. Thacker.

I would like to thank my loving husband, David; my children, Gabriella and Elliot; and my parents. Without their love and support this book would not have been possible.

I appreciate Quintessence and the editorial staff, especially Zachary Kocanda and Bryn Grisham, whose knowledge and dedicated care to every word and idea made this book what it is.

Evidence-Based Dentistry

1

Background

Q: What is the evidence-based approach?

Evidence-based dentistry is the merging of clinically pertinent scientific evidence to the patient's oral and medical condition and history as well as the dentist's experience (Fig 1-1). The dentist uses the evidence to make sound decisions about diagnosis, prognosis, and treatment. Evidence-based decision making consists of formulating patient-centered questions (Population-Intervention-Comparison-Outcome [PICO]); examining and critically evaluating the evidence; and relating the evidence to practice.[1]

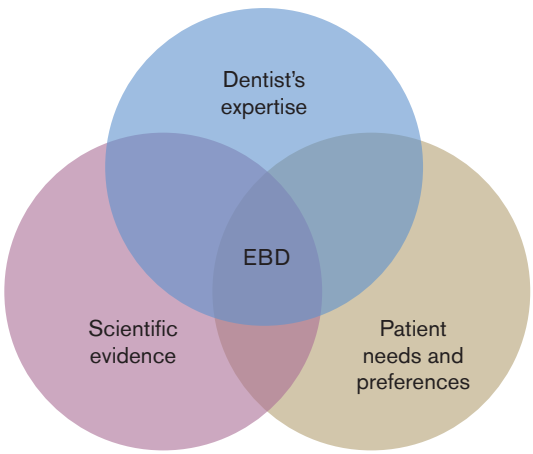

Fig 1-1 Three parts of the decision-making process. (Redrawn from the American Dental Association[1] with permission.)

Q: What is the PICO question?

The *PICO question* is a question that includes a *p*opulation to be examined, the nature of the *i*ntervention to be inspected, a *c*omparison statement, and the type of *o*utcome to be evaluated. It should be problem-focused and concise.

Example: In patients with horizontal alveolar ridge deficiencies (population), what is the effect of horizontal bone augmentation procedures (intervention) compared with controls (comparison) on peri-implant health (outcome)?

Q: What is the step-by-step process for making an evidence-based decision in a dental practice?

The steps involved in evidence-based decision making in a dental practice are shown in Fig 1-2.

Fig 1-2 Evidence-based decision making. (Based on data in Chiappelli et al.[2])

Studies

Q: What are the different study types (ranked from highest level of evidence to lowest)?

The different types of studies are shown, ranked in order of highest to lowest level of evidence, in Fig 1-3.

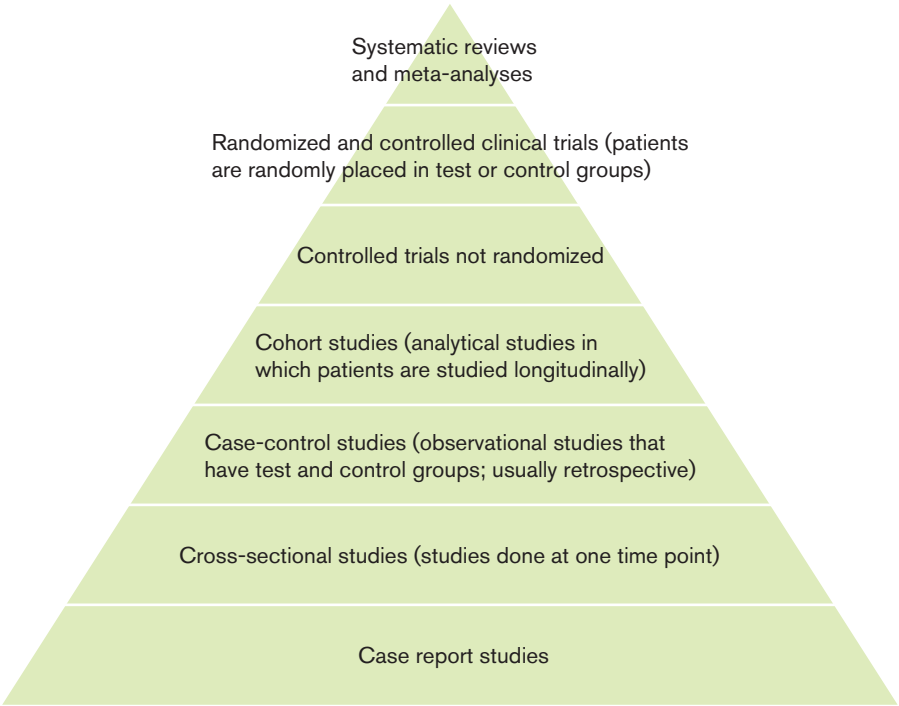

Fig 1-3 Different studies ranked from highest level of evidence to lowest. (Based on Nocini et al.[3])

Q: Describe the difference between a cross-sectional study and a longitudinal study.

A cross-sectional study is done at one time point, whereas a longitudinal study ranges over a period, allowing temporal relationships to be investigated.

Q: What is the *P* value?

The *P* value is the probability of obtaining a test statistic at least as extreme as the one observed, assuming that the null hypothesis is true. The smaller the *P* value, the less likely the effect was due to chance. A *P* value less than or equal to .05 usually indicates statistical significance.

Q: What is the difference between sensitivity and specificity?

Sensitivity is the ability of a test to correctly identify diseased individuals. *Specificity* is the ability of a test to correctly identify a healthy individual.

For instance, the diagnostic sensitivity of a clinical parameter (suppuration, gingival plaque) in predicting disease was expressed as the proportion of sites showing attachment loss that also exhibited the given parameter. Diagnostic specificity was expressed as the proportion of sites not exhibiting the clinical parameter and not showing attachment loss.[4]

Q: What is the difference between internal and external validity?

The difference between internal and external validity is shown in Fig 1-4.

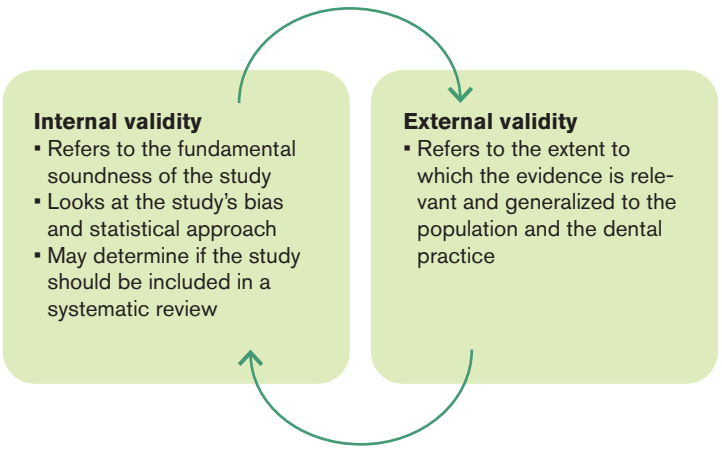

Fig 1-4 Internal and external validity.

Q: Why practice evidence-based dentistry?[5]

1. There are thousands of articles published monthly in dental magazines. It would take hundreds of hours to read the dental literature. Using evidence-based review databases eases the necessary time spent evaluating dental literature.
2. Practicing evidence-based dentistry keeps dentists current on recent evidence and practice standards.
3. A thorough and analytical literature review should be carried out before proceeding in clinical research.

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Periodontal Anatomy

2

Anatomy

Q: Identify the anatomical structures of the periodontium shown below.

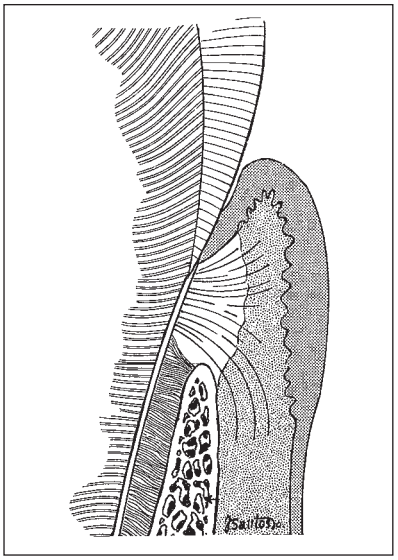

Fig 2-1a Illustration of the periodontium. (Reprinted from Fan and Berry[1] with permission.)

The answers are shown on the next page in Fig 2-1b.

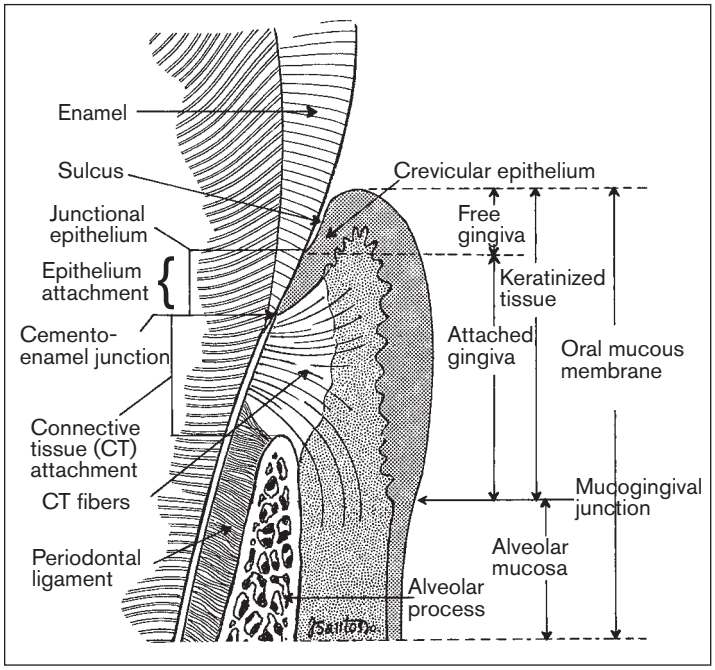

Fig 2-1b Labeled anatomy of the periodontium. (Reprinted from Fan and Berry[1] with permission.)

Q: Where does the vascular supply of the periodontium originate?

The external carotid artery and its main branches, which include the lingual, facial, and maxillary arteries, are the vascular supply for the periodontium. Locally, the blood supply comes from the supraperiosteal vessels and vessels from the periodontal ligament (PDL) and bone.[2]

Q: What is the main innervation for the periodontium?

The trigeminal nerve and its branches provide the main innervation for the periodontium.

Definitions

Q: What is attached gingiva?

The *attached gingiva* is the area from the base of the sulcus to the mucogingival junction. It prevents the free gingiva from being separated from the tooth. Its height is determined by subtracting the sulcus probing depth from the total width of the keratinized tissue. It consists of thick lamina propria and deep rete pegs. Goaslind et al[3] reported that the attached gingival thickness is 1.25 ± 0.42 mm.

Q: What is keratinized attached gingiva?

The *keratinized attached gingiva* is that found from the gingival margin to the mucogingival junction.

Q: What is alveolar mucosa?

Alveolar mucosa is the covering of the alveolar process that is nonkeratinized, unstippled, and movable. It extends from the mucogingival junction to the floor of the mouth and vestibular epithelium.

Q: What is clinical attachment loss (CAL)?

If the marginal gingiva is below the cementoenamel junction (CEJ):

CAL = pocket depth + [CEJ to marginal gingiva]

If the marginal gingiva is above the CEJ:

CAL = [marginal gingiva to CEJ] – [marginal gingiva to bottom of pocket]

Q: What is Ante's law?

Ante's law states that the root surface area of the abutment teeth must be equal to or greater than that of teeth being replaced with pontics. This helps determine the number of abutments needed for a fixed partial denture.

Gingival Epithelium

Q: What are the characteristics of healthy gingiva?

Healthy gingiva is coral pink, firm, follows the CEJ of the teeth, and may be stippled. The color of the gingiva is associated with the pigmentation of the patient. In dark-haired individuals, the gingiva can be darker than that in blond patients.

Q: What are the five types of gingival fibers?

There are five types of gingival fibers:

1. Dentogingival group: There are three types of fibers within this group.
   - Fibers extending coronally toward the gingival crest
   - Fibers extending laterally to the facial gingival surface
   - Fibers extending horizontally beyond the alveolar crest height and then apically along the alveolar bone cortex
2. Alveologingival group: Fibers in this group run coronally into the lamina propria from the periosteum at the alveolar crest.
3. Dentoperiosteal fibers: These fibers insert into the periosteum of the alveolar crest and fan out to the adjacent cementum.
4. Circular group: These are the only fibers that are confined to the gingiva and do not attach to the teeth.
5. Transseptal group: These fibers bridge the interproximal tissue between adjacent teeth and insert into the cementum.

Q: What is the composition of the oral mucosa (the tissue lining the oral cavity)?

The oral mucosa is composed of masticatory, lining, and specialized tissues (Fig 2-2).

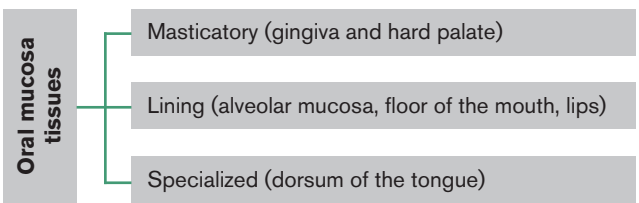

Fig 2-2 Composition of the oral mucosa. (Based on Avery.[4])

Q: What is the composition of the gingival epithelium?

The gingival epithelium consists of oral (masticatory), oral sulcular, and junctional epithelia (average width < 1 mm) (Fig 2-3).

Oral (masticatory) epithelium	Oral sulcular epithelium	Junctional epithelium
▪ Orthokeratinized, stratified squamous ▪ Surface cells lose their nuclei and contain keratin ▪ Made of the following: free gingiva (base of the sulcus to the free gingival margin, ie, the most coronal part of the gingiva), attached gingiva, and palatal tissue	▪ Epithelium that lines the sulcus ▪ No rete pegs in healthy tissue	▪ Attaches to the tooth via a hemidesmosomal layer and a basal lamina ▪ Nonkeratinized and has a fast turnover ▪ Permeable ▪ Most apical part lies at the CEJ in healthy tissue

Fig 2-3 Composition of the gingival epithelium. (Based on Clerehugh et al.[5])

Q: What are the four layers of cells that comprise the masticatory epithelium?

There are four layers of cells that comprise the masticatory epithelium[2]:

1. Stratum basale: Cuboidal cells found at the basement membrane; epithelial cell replication takes place in this location. This layer contains melanocytes and Merkel cells.
2. Stratum spinosum: The "spines" are desmosomes allowing intracellular contacts. It is the thickest layer and contains Langerhans cells, which are derived from bone marrow and take part in immune surveillance.
3. Stratum granulosum: Cells in this layer appear flat. Keratinocytes migrating from the underlying stratum spinosum become known as *granular cells* in this layer. These cells contain keratohyalin granules, protein structures that promote hydration and cross-linking of keratin. →

4. Stratum corneum: Outermost layer containing dead cells and consisting of ortho- and parakeratinization. It is composed of compactly packed tonofilaments.

Q: Where are the widest and narrowest zones of gingiva?

The average thickness of the gingiva is 1.25 mm.[3] The widest zone of gingiva is in the maxillary anterior region; the narrowest zone is at the facial aspect of the mandibular first premolar.[2]

Connective Tissue

Q: What is the composition of connective tissue?

Connective tissue (average width slightly greater than 1 mm) is fibrous, consisting of mostly type I collagen, ground substances, and mucopolysaccharides. It also contains white blood cells, blood vessels, lymphatics, and nerves.[2]

Q: What determines whether epithelium is keratinized or nonkeratinized?

The underlying connective tissue determines whether the epithelium is keratinized.[6]

Q: What is periosteum? What is its function?

The *periosteum* is a highly vascular connective tissue sheath covering the external surface of all bones except areas of articulation and muscle attachment. It consists of an inner cambium layer (contains osteoblasts and osteoprogenitor cells) and an outer fibrous layer.[7]

The periosteum is involved in bone healing and bone regeneration.[8] It also serves as protection as well as a channel for the blood supply and nutrients for bone tissue.

Periodontal Ligament (PDL)

Q: Where is the average width of the PDL greatest and where is it narrowest?

The width of the PDL is greatest at the apex and narrowest in the middle. Older individuals have thicker fiber bundles in the PDL than younger individuals. The average width of the PDL is 0.2 mm.

Q: What provides the blood supply to the PDL?

Superior and inferior alveolar arteries provide the blood supply to the PDL, which is a vascular tissue.

Q: What are the functions of the PDL?

- Protect vessels and nerves
- Transmit occlusal forces
- Attach the tooth to bone
- Perform formative and remodeling functions

Q: What are the fibers of the PDL?

The fibers of the PDL include the alveolar crest, horizontal, oblique (most numerous), interradicular, and apical fibers.

Q: Describe and define ankylosis.

Ankylosis is the fusion of the cementum and alveolar bone with obliteration of the PDL. It develops after chronic periapical inflammation, tooth reimplantation, and occlusal trauma.

Alveolar Bone

Q: What is the composition of alveolar bone?

Alveolar bone consists of[2]:

- Cortical bone
- Cancellous trabeculae (more prevalent in the maxilla)
- Alveolar bone proper (lines the tooth socket)

Q: What are the functions of the alveolar bone?

The alveolar bone has three functions[2]:

1. Protection
2. Support
3. Calcium metabolism

Cementum

Q: Where are acellular cementum and cellular cementum located?

- Acellular cementum is located on the enamel at the CEJ. It does not contain cementocytes and forms slowly.
- Cellular cementum is located at the apical third of the root. It is more irregular and forms rapidly. With age, there is an increase in width of the cellular cementum.

Q: What percentage of the cementum and enamel overlap?

- 60% of the cementum and enamel overlap.
- 30% of the cementum and enamel form a butt joint.
- 10% of the cementum and enamel are separated by a gap.

Q: What is the difference between extrinsic and intrinsic cementum?

Extrinsic fibers are made of Sharpey fibers from the PDL, whereas intrinsic fibers are cementum fibers produced by cementoblasts (Fig 2-4).

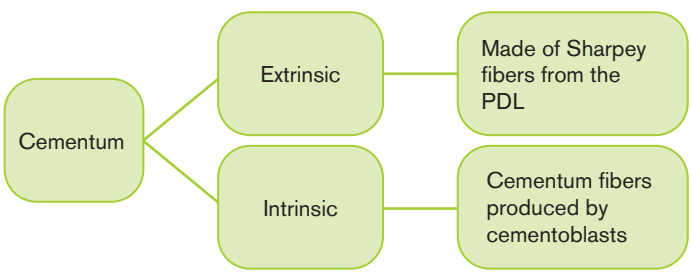

Fig 2-4 Characteristics of extrinsic and intrinsic cementum.

Q: How does the junctional epithelium attach to the cementum?

The junctional epithelium attaches to the cementum via hemidesmosomes and replicates every 5 days.

Temporomandibular Joint (TMJ)

Q: What is the composition of the TMJ disc (meniscus)?

The TMJ disc is composed of dense connective tissue.

Q: Describe the movement of the TMJ.

The meniscus divides the joint into two compartments. The upper compartment has translational movement, and the lower compartment has rotational movement.

Q: What is meniscal derangement with and without reduction?

- With reduction: The disc as well as the posterior band of the meniscus is anteriorly displaced in front of the condyle upon opening. This causes a popping or clicking sound.
- Without reduction: In some patients, the meniscus remains anteriorly displaced at full opening. This is a much more serious condition.

Collagen

Q: Describe the four different types of collagen.

- Type I: Skin, tendon, vascular ligature, organs, bone (main component of the organic part of bone)
- Type II: Cartilage (main component of cartilage)
- Type III: Comprised of reticular fibers, commonly found alongside type I collagen, found mostly in smooth muscle
- Type IV: Forms basis of cell basement membrane

Supracrestal Tissue Attachment (Previously Biologic Width)

Q: What is supracrestal tissue attachment?

The *supracrestal tissue attachment* is defined as the physiologic dimension of the junctional epithelium and connective tissue attachment. It is measured from the most coronal part of the junctional epithelium to the crest of the alveolar bone. In studies on cadavers, Gargiulo[9] found a connective tissue attachment of 1.07 mm and a junctional epithelium attachment of 0.97 mm. Therefore, the supracrestal tissue attachment is about 2 mm. He also found the sulcus, which is not part of the supracrestal tissue attachment, to be 0.69 mm.

Q: What results from violation of the supracrestal tissue attachment?

If subgingival restorations violate the supracrestal tissue attachment, periodontal bone loss and inflammation may occur. The body will try to make room between the margin of the restoration and the alveolar bone to allow for reestablishment of the supracrestal tissue attachment.

Günay[10] did a study comparing crowns with interproximal margins placed at different distances from the alveolar bone. The three groups were $\leq$ 1 mm (I), 1 to 2 mm (II), and $\geq$ 2 mm (III) between crown margin and alveolar crest. It was observed that probing depth and papillary bleeding index was greater for group I (which had an encroachment of its crown margins within the supracrestal tissue attachment).

Miscellaneous

Q: What is the most common area of recurrent pockets?

The mesial aspect of the maxillary first premolars and first molars are the most common areas of recurrent pockets.

Q: What is the relationship between tooth support and root morphology?[11]

1. Root curvatures and concavities increase periodontal support because they increase the surface area and allow for multidirectional fiber orientation, which makes the tooth more stable.
2. Multirooted teeth have increased support and resistance to applied forces (the more coronal the furcation, the more stability).
3. Divergent roots increase stability and allow for more bone support.
4. Conical roots have less attachment area and are not as stable.
5. Enamel pearls can weaken periodontal attachment.
6. Root fractures can lead to periodontal destruction.

Many other factors can influence tooth stability, such as inflammation, occlusion, and the density and structure of bone.

Q: Which muscles elevate and depress the mandible?

- Elevate: Temporalis, medial pterygoid, and masseter
- Depress: Lateral pterygoid, digastric, and mylohyoid

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Furcations 3

Background

Q: Define furcation lesion.

A *furcation lesion* has been defined as "the pathologic resorption of bone in the anatomic area of a multi-rooted tooth where the roots diverge."[1]

Q: What is the prevalence of furcation involvement?

In a study on periodontal patients, Svärdström and Wennström[2] found a greater prevalence of furcation involvement in maxillary molars than in mandibular molars. The narrowest furcation entrance was found on the buccal aspect of maxillary and mandibular molars, and the highest frequency of involvement was the distal aspect of the maxillary first molar. The mesial aspect of the second molar had the least frequency of furcation involvement.

Q: What is a furcation fornix?

The *furcation fornix* is the roof of the furcation.

Q: Is the Nabers probe a valid and efficient tool for detecting furcation invasion?

Eickholz and Kim[3] found that the Nabers probe, marked in 3-mm increments, is a valid method of diagnosing furcation lesions.

Classification

Q: Describe the Hamp classification.

The classification by Hamp et al[4] involves a horizontal measurement:

- F0: No furcation involvement.
- F1: The probe can penetrate the furcation less than 3 mm.
- F2: The furcation can be probed greater than 3 mm, but it is not a through and through furcation involvement.
- F3: Through and through furcation involvement.

Q: Describe the Glickman classification.

The Glickman[5] classification is presented in Fig 3-1.

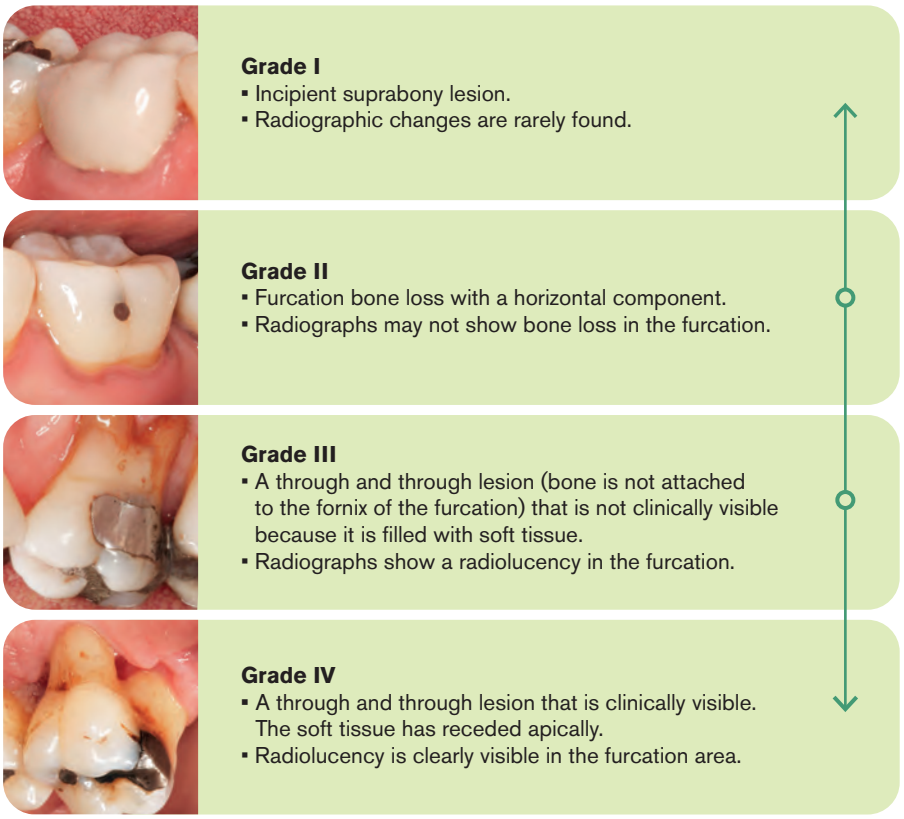

Fig 3-1 Glickman classification.

Q: Describe the Tarnow classification.

The classification by Tarnow and Fletcher[6] is a subclassification of the Glickman furcation classification that measures the vertical probing depth from the roof of the furca:

- A: 0 to 3 mm
- B: 4 to 6 mm
- C: Greater than 7 mm

Treatment Options

Q: What are the treatment options for furcation defects?

- Nonsurgical debridement
- Surgical debridement
- Surgical exposure of the furcation
- Regeneration (guided tissue regeneration [GTR] and enamel matrix derivative [EMD])
- Extraction
- Root resection
- Tunnel preparation

Q: Is open/closed flap scaling and root planing effective in furcation lesions?

A review by Cobb[7] demonstrated a less favorable response to scaling and root planing by molars with furcation involvement compared with those without furcation lesions and single-rooted teeth. He surmised that this was related to the inability to remove all pathogenic microbial flora due to the furcal anatomy restricting access for mechanical therapy.

Bower[8] found that 81% of the time the furcation entrance is 1 mm or less. The study also found that 58% of the time the furcation entrance is 0.7 mm or less. The blade width of commonly used periodontal curettes is 0.75 mm. The ultrasonic (smaller) tip would fit better than the tip of a Gracey curette in a grade II or III furcation.

Wylam et al[9] found no significant difference between open and closed flap root planing. The study further concluded that root planing is inefficient in the debridement of furcation lesions and does not allow for periodontal regeneration.

Q: What are guidelines for root resection?[10]

Situations favorable for root resection:

- The candidate tooth is of critical importance to the overall dental treatment plan (abutments, important to a prosthesis).
- The tooth has enough attachment present to function.
- No other cost-effective therapy is available.
- Patient has good oral hygiene.

How to determine which root to remove:

- Remove the root that eliminates the furcation.
- Remove the root that has the greatest amount of bone and attachment loss.
- Remove the root that eliminates periodontal bone loss on adjacent teeth.
- Remove the root with the most anatomical problems.
- Remove the root that would complicate future treatment the least.
- The most common root resected is the distobuccal root of the maxillary first molar.

See chapter 10 for more information—eg, indications, contraindications—on root resections.

Q: Would you have endodontic therapy done before a root resection and why?

Endodontic therapy should be done prior to root resection for the following reasons:

- Little/no pain.
- It would be prudent to do the root canal therapy so that if a perforation, fracture, or other adverse outcome occurs, the patient does not have to have the resection done.[10]

Q: Describe the technique for root resection.[10]

1. Facial and lingual flaps are elevated.
2. Debridement and exposure of the furcation of the root to be resected.
3. Removal of a small amount of buccal or palatal bone to allow access for elevation and root removal.
4. An oblique cut is made with the high-speed handpiece using a carbide bur (surgical length) through the tooth.
5. The root is elevated from the socket, and if necessary odontoplasty and/or periodontal surgery are done on adjacent teeth. →

6. Sutures should be placed.
7. Occlusion may have to be adjusted because of removal of the root.

Q: Can a grade I, II, III, IV furcation be treated successfully? Which treatment is the most effective?

According to the 2015 consensus report from the American Academy of Periodontology[11] (Table 3-1):

Table 3-1 Most effective treatments

Hamp classification	Most effective treatment
F1	Regenerative therapy may be helpful, although generally F1 furcation defects may be treated successfully with non-regenerative therapies.
F2	Regenerative periodontal therapy should be considered before resective therapy or extraction. A combined therapeutic approach (membrane, bone graft with or without biologics) appears more beneficial over monotherapeutic algorithms.
F3	Proof of histologic periodontal regeneration in mandibular F3 defects is limited to one case report. Favorable outcomes after regenerative therapy for maxillary F3 furcation defects are limited to clinical case reports.

According to a systematic review by Huynh-Ba et al,[12] treatment for the maintenance of teeth with furcation defects (ie, open flap debridement [OFD], tunneling, root amputation, hemisection, and regeneration) are associated with high survival rate ranges, with GTR being the highest one with a survival rate range of 83.3% to 100% after 5 to 12 years. These survival rates are pretty close to implant survival rates in periodontal patients for the same time period (see implant chapter).

Huynh-Ba et al[12] also found the most recurrent complications after resective treatment were not related to periodontal disease but were related to endodontic failures and vertical root fractures. It is important to note that full furcation closure was not predictably attained following GTR or the application of EMD in maxillary and mandibular molars. →

Kinaia et al[13] did a meta-analysis and discovered, "guided tissue regeneration with the use of resorbable membranes was superior to non-resorbable membranes in vertical bone fill. Both types of membranes were more effective than open flap debridement in reducing vertical probing depths and gaining vertical attachment levels and in gaining vertical and horizontal bone."

Chen et al[14] did a meta-analysis and found that the GTR and GTR with osseous grafting (OG) groups obtained greater vertical/horizontal bone fill, furcation closure rate, and vertical/horizontal attachment level gain than the OFD group in mandibular molars. The GTR group obtained greater vertical attachment level gain and vertical/horizontal bone fill than the OFD group in maxillary molars. The GTR with OG group achieved better clinical outcomes than the GTR group did in all the studies comparing outcomes in mandibular molar.

Similarly, Murphy and Gunsolley[15] performed a systematic review and discovered that GTR is more effective than OFD in the treatment of furcation defects. The study also found that in furcation lesions vertical probing depth, vertical probing attachment levels, and horizontal open probing attachment levels—but no intrabony outcomes—were improved by augmentation with a particulate graft in conjunction with the GTR barrier.

Evans et al[16] reviewed 50 papers that studied 1,016 grade II furcations. The teeth had received various treatments such as bone grafts, OFD, and GTR. The study found complete closure of the furcation in 20% of the grade II furcations and an improvement of a grade II to a grade I furcation in 33% of the cases. In general, there was a 50% improvement for grade II furcations. The most effective treatment was a bone graft with GTR, and the least effective treatment was OFD (2% of the grade II furcations had complete closure).

Bowers et al,[17] who treated his patients with a combination therapy using an expanded polytetrafluoroethylene (ePTFE) membrane and demineralized freeze-dried bone allograft (DFDBA), discovered complete closure of grade II mandibular furcations in 50% of patients. There was a 68% improvement of a grade II to a grade I furcation.

Q: What factors influence the success of treatment?

Bowers et al[17] found poorer results in the treatment of furcation lesions in smokers. Smokers had a 62.5% chance of having a grade II residual defect compared with nonsmokers, who had a 14.3% chance. Furcation fill decreases at an increased horizontal and vertical presurgical probing attachment level (greater than 5 mm). The following were found to reduce the frequency of clinical closure: →

- Increased distance between the roof of the furcation and the crest of bone
- Increased distance between the roof of the furcation and the base of the defect
- The depth of the horizontal defect
- Increased divergence of roots at the crest of bone

Q: Are teeth that undergo the tunnel preparation in the furcation area prone to caries?

Hellden et al[18] studied 156 teeth with advanced periodontal furcation defects that were treated by tunnel preparations. The study found that 25% of the teeth developed caries.

A retrospective study by Feres et al[19] demonstrated that a history of root caries was the only factor with a positive association with caries incidence in tunnels.

Q: Are biologic mediators effective in furcation lesions?

Enamel matrix derivative (EMD)

Because grade II or III furcation defects are noncontained defects, the use of biologic materials has the significant limitation that, because of their fluid or gel-like quality, any space-making effect is prevented, and therefore the regenerative possibility of such tools may be inadequate in furcation lesions.[1]

A study by Araújo and Lindhe[20] used EMD and a barrier in the test defects and just a barrier in control defects. The test defect was found to have acellular cementum in the apical portion, while in the coronal portion a thick cellular cementum, similar to the cementum found in the control group, was detected. Both the test and control group furcation defects were found to be clinically closed and to contain bone and periodontal ligament tissue that appeared structurally similar to newly formed root cementum.

A randomized clinical trial by Jepsen et el[21] comparing membrane placement and EMD treatment of buccal grade II furcation involvement in mandibular molars found a significantly greater reduction in horizontal furcation depth and a comparatively lower incidence of postoperative swelling/pain following EMD compared to membrane therapy.

Although enamel matrix protein therapy has exhibited clinical improvements in the treatment of buccal grade II furcation defects in mandibular molars, complete closure of the furcation lesion is attained only in a minority of cases.[1]

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